SA-PO0531

Hyperkalemia in finerenone-treated patients with chronic kidney disease without diabetes: A FIND-CKD trial analysis

Rajiv Agarwal,[1] David Z. I. Cherney,[2] Carolyn S. P. Lam,[3,4] Brendon L. Neuen,[5,6] Vlado Perkovic,[7] Katherine R. Tuttle,[8] Pantelis Sarafidis,[9] Meike Brinker,[10] Hiddo J. L. Heerspink,[5,11] on behalf of the FIND-CKD investigators

[1]Division of Nephrology, Richard L. Roudebush VA Medical Center, Indianapolis, Indiana University School of Medicine, Indianapolis, IN, USA; [2]Division of Nephrology, University Health Network, Toronto General Hospital, University of Toronto, Toronto, ON, Canada; [3]National Heart Centre Singapore, Singapore; [4]Duke-National University of Singapore Medical School, Singapore; [5]Renal and Metabolic Division, George Institute for Global Health, University of New South Wales, Sydney, NSW, Australia; [6]Department of Renal Medicine, Royal North Shore Hospital, Sydney, NSW, Australia; [7]University of New South Wales, Sydney, NSW, Australia; [8]Providence Inland Northwest Health, University of Washington School of Medicine, Spokane, WA, USA; [9]1st Department of Nephrology, Aristotle University of Thessaloniki, Hippokration Hospital, Thessaloniki, Greece; [10]Cardiology and Nephrology Clinical Development, Bayer AG, Wuppertal, Germany; [11]Department of Clinical Pharmacy and Pharmacology, University of Groningen, University Medical Center Groningen, Groningen, Netherlands

Corresponding author: Rajiv Agarwal, Division of Nephrology, Richard L. Roudebush VA Medical Center, Indianapolis, Indiana University School of Medicine, Indianapolis, IN, USA, ragarwal@iu.edu

Background

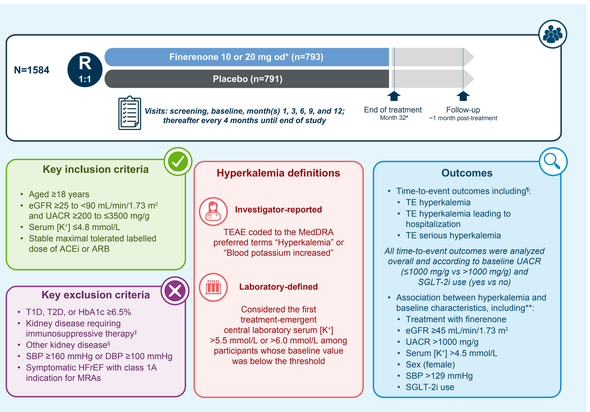
- The selective nonsteroidal mineralocorticoid receptor antagonist finerenone was shown to reduce the risk of adverse kidney and cardiovascular events in patients with nondiabetic chronic kidney disease (CKD) in the phase III randomized trial FIND-CKD (NCT05047263)[1]
- In FIND-CKD, finerenone treatment was associated with a predictable and clinically manageable increase in serum potassium concentration ([K+]) compared with placebo[1]
- In the phase III FIDELIO-DKD trial, higher baseline serum [K+], lower estimated glomerular filtration rate (eGFR), and increased urinary albumin-to-creatinine ratio (UACR) were identified as independent risk factors for hyperkalemia in patients with CKD and type 2 diabetes.[2] It remains to be seen whether these findings can be extended to patients with nondiabetic CKD
- This subgroup analysis of the FIND-CKD trial aimed to investigate the effect of finerenone on hyperkalemia-related outcomes in key subgroups of interest and identify baseline characteristics that may be risk factors for hyperkalemia in patients with nondiabetic CKD

Methods

Study design and outcomes

- FIND-CKD randomized adults with nondiabetic CKD (eGFR 25 to <90 mL/min/1.73 m², UACR ≥200 to ≤3500 mg/g, and serum [K+] ≤4.8 mmol/L) treated with a stable, maximum tolerated dose of renin–angiotensin system inhibitor were randomized 1:1 to oral finerenone or matching placebo[1]
- Participants with eGFR ≥25 to <60 mL/min/1.73 m² received finerenone 10 mg and participants with eGFR ≥60 mL/min/1.73 m² received finerenone 20 mg
  - Participants were up-titrated from finerenone 10 mg to 20 mg from month 1 provided serum [K+] was ≤4.8 mmol/L and eGFR decrease was <30% of the last measured value
  - The study intervention was interrupted if serum [K+] was >5.5 mmol/L
- The FIND-CKD study design, inclusion and exclusion criteria, and outcomes assessed in this subanalysis are shown in **Figure 1**

Figure 1. FIND-CKD study design



*For 10 mg od: eGFR ≥25 to <60 mL/min/1.73 m²; for 20 mg od: eGFR ≥60 mL/min/1.73 m²; #participants will stay in the study until the last randomized participant has reached 32 months of treatment; ‡lupus nephritis, anti-neutrophil cytoplasmic antibody–associated vasculitis, or any other kidney disease within 6 months prior to screening; §known autosomal recessive or dominant polycystic kidney disease; ¶prespecified safety outcomes; **post hoc analyses

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; HFrEF, heart failure with reduced ejection fraction; [K+], potassium concentration; MedDRA, Medical Dictionary for Regulatory Activities; MRA, mineralocorticoid receptor antagonist; od, once daily; R, randomized; SBP, systolic blood pressure; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; T1D, type 1 diabetes; T2D, type 2 diabetes; TE, treatment-emergent; TEAE, treatment-emergent adverse event; UACR, urinary albumin-to-creatinine ratio

Statistical analysis

- All analyses used the safety analysis set, which comprised randomized participants who took at least one dose of study drug
  - Time to first event was compared between groups using Cox proportional hazards models stratified by the randomization strata
  - Change in serum [K+] was estimated using a mixed model for repeated measures
  - To identify predictors, logistic regression models were fitted separately for investigator-reported hyperkalemia and for laboratory-reported hyperkalemia
    - Candidate variables were retained by backward elimination at a likelihood-ratio $p<0.10$, with treatment assignment forced into the model. Deaths were counted as nonevents

Results

Baseline characteristics

- Among the 1584 participants, 953 had baseline serum [K+] ≤4.5 mmol/L and 631 had serum [K+] >4.5 mmol/L
- Baseline characteristics were generally balanced between serum [K+] subgroups (**Table 1**), with a few exceptions:
  - The proportion of Asian patients with serum [K+] ≤4.5 mmol/L was numerically higher than those with serum [K+] >4.5 mmol/L (57.9% and 50.1%, respectively)
    - The inverse was seen for White patients, who had numerically higher rates of serum [K+] >4.5 mmol/L compared with serum [K+] ≤4.5 mmol/L (46.0% and 37.6%, respectively)
  - Baseline mean eGFR was numerically higher and median UACR was numerically lower for participants with serum [K+] ≤4.5 mmol/L versus those with serum [K+] >4.5 mmol/L

Table 1. Baseline characteristics according to serum [K+]

Characteristic	Serum [K+] ≤4.5 mmol/L (n=953)	Serum [K+] >4.5 mmol/L (n=631)	Overall (N=1584)
Treatment group			
Finerenone	477 (50.1)	316 (50.1)	793 (50.1)
Placebo	476 (49.9)	315 (49.9)	791 (49.9)
Age, years, mean ± SD	55.2 ± 14.4	54.0 ± 14.1	54.7 ± 14.3
Sex, male, n (%)	636 (66.7)	413 (65.5)	1049 (66.2)
Race, n (%)			
Asian	552 (57.9)	316 (50.1)	868 (54.8)
White	358 (37.6)	290 (46.0)	648 (40.9)
Black	26 (2.7)	11 (1.7)	37 (2.3)
Other*	17 (1.8)	14 (2.2)	31 (2.0)
CKD etiology confirmed by biopsy, n (%)	465 (48.8)	323 (51.2)	788 (49.7)
BMI, kg/m², mean ± SD	27.9 ± 5.7	27.2 ± 5.4	27.6 ± 5.6
Serum [K+], mmol/L, mean ± SD	4.2 ± 0.3	4.9 ± 0.3	4.5 ± 0.4
HbA1c, %, mean ± SD	5.5 ± 0.4	5.5 ± 0.4	5.5 ± 0.4
eGFR, mL/min/1.73 m², mean ± SD	49.3 ± 16.9	42.8 ± 13.9	46.7 ± 16.1
UACR, mg/g, median (Q1–Q3)	807.1 (580.8–1231.9)	831.6 (571.7–1257.6)	818.9 (577.4–1244.0)
SBP, mmHg, mean ± SD	130.6 ± 14.8	127.8 ± 12.7	129.5 ± 14.1
Medical history, n (%)			
ASCVD	121 (12.7)	74 (11.7)	195 (12.3)
Hypertension	850 (89.2)	557 (88.3)	1407 (88.8)
Heart failure	21 (2.2)	14 (2.2)	35 (2.2)
Medication use, n (%)			
ACEi	NR	NR	NR
ARB	NR	NR	NR
Beta blockers	NR	NR	NR
Diuretics	194 (20.4)	96 (15.2)	290 (18.3)
Glucocorticoids	58 (6.1)	34 (5.4)	92 (5.8)
SGLT-2i	154 (16.2)	116 (18.4)	270 (17.0)

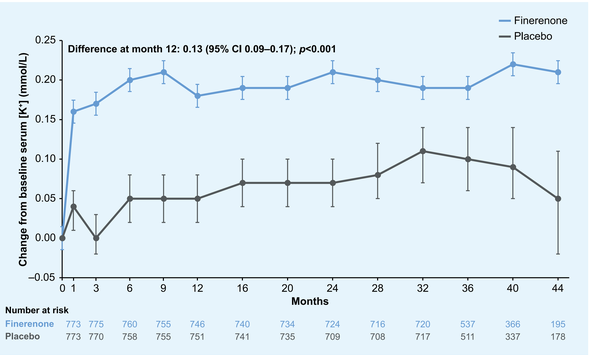
*Includes American Indian, Alaska native, native Hawaiian, Pacific Islander, participants who reported that they belong to more than one race, and participants whose race was not reported

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ASCVD, atherosclerotic cardiovascular disease; BMI, body mass index; CKD, chronic kidney disease; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; [K+], potassium concentration; NR, not reported; Q, quartile; SBP, systolic blood pressure; SD, standard deviation; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UACR, urinary albumin-to-creatinine ratio

Change in serum [K+] from baseline over time

- In the finerenone arm, serum [K+] increased within the first month and was consistently higher compared with placebo for the remainder of the trial (**Figure 2**)
  - The placebo group also demonstrated an upward trend in the change in serum [K+] from baseline, increasing from 0.05 mmol/L at month 6 to 0.11 mmol/L at month 32
  - The placebo-corrected increase for the finerenone group reached its maximum of 0.16 mmol/L at month 3
  - The placebo-corrected increase for the finerenone group was 0.13 mmol/L (95% confidence interval [CI] 0.09–0.17) at month 12
- Similar trends were observed across eGFR (≤45 mL/min/1.73 m² vs >45 mL/min/1.73 m²), UACR (≤1000 mg/g vs >1000 mg/g) and sodium-glucose co-transporter-2 inhibitor (SGLT-2i) use (yes vs no) subgroups, with no significant heterogeneity in the placebo-corrected increase with finerenone at month 12 detected between subgroups (p-interaction>0.1)

Figure 2. Overall change in serum [K+] from baseline. Least squares means with 95% CIs; safety analysis set



Error bars denote 95% CI

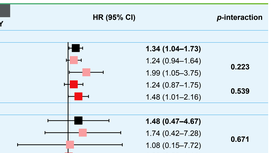
CI, confidence interval; [K+], potassium concentration

Time-to-event outcomes

- The rates of all investigator-reported outcomes were higher with finerenone compared with placebo, including treatment-emergent hyperkalemia (hazard ratio [HR]=1.34; 95% CI 1.04–1.73), treatment-emergent hyperkalemia leading to hospitalization (HR=1.48; 95% CI 0.47–4.67), and treatment-emergent serious hyperkalemia (HR=1.68; 95% CI 0.55–5.14), and did not differ according to baseline SGLT-2i use (with vs without; p-interaction=0.223, 0.671, and 0.573, respectively) or baseline UACR levels (≤1000 mg/g vs >1000 mg/g; p-interaction=0.539, 0.377, and 0.254, respectively) (**Figure 3**)
- The incidence of hyperkalemia among participants with serum [K+] >5.5 mmol/L was 18.8% and 12.2% for finerenone and placebo, respectively. For participants with serum [K+] >6.0 mmol/L, the incidence of hyperkalemia was 4.3% and 2.3% for finerenone and placebo, respectively
- The rates of laboratory-reported outcomes were higher with finerenone compared with placebo, including time to first serum [K+] >5.5 mmol/L (HR=1.59; 95% CI 1.23–2.06) and time to first serum [K+] >6.0 mmol/L (HR=1.87; 95% CI 1.05–3.31), and did not differ according to baseline SGLT-2i use (with vs without; p-interaction=0.184 and 0.279, respectively) or baseline UACR levels (≤1000 mg/g vs >1000 mg/g; p-interaction=0.101 and 0.605, respectively)

Figure 3. Investigator-reported and laboratory-reported hyperkalemia outcomes

Outcome	Finerenone Events, n/N	Finerenone Rate/1000 PY	Placebo Events, n/N	Placebo Rate/1000 PY	HR (95% CI)	p-interaction
Investigator-reported: TE* hyperkalemia						
Overall	**135/793**	**66.7**	**105/791**	**49.8**	**1.34 (1.04–1.73)**	
Without SGLT-2i	109/658	64.1	90/656	51.1	1.24 (0.94–1.64)	**0.223**
With SGLT-2i	26/135	80.9	15/135	43.2	1.99 (1.05–3.75)	
UACR ≤1000 mg/g	70/491	54.0	59/491	43.5	1.24 (0.87–1.75)	**0.539**
UACR >1000 mg/g	65/302	89.6	46/300	61.4	1.48 (1.01–2.16)	
Investigator-reported: TE* hyperkalemia leading to hospitalization						
Overall	**7/793**	**3.2**	**5/791**	**2.2**	**1.48 (0.47–4.67)**	
Without SGLT-2i	5/658	2.7	3/656	1.6	1.74 (0.42–7.28)	**0.671**
With SGLT-2i	2/135	5.7	2/135	5.6	1.08 (0.15–7.72)	
UACR ≤1000 mg/g	4/491	2.8	4/491	2.8	1.03 (0.26–4.14)	**0.377**
UACR >1000 mg/g	3/302	3.8	1/300	1.3	3.31 (0.34–31.97)	
Investigator-reported: TE* serious hyperkalemia						
Overall	**8/793**	**3.6**	**5/791**	**2.2**	**1.68 (0.55–5.14)**	
Without SGLT-2i	6/658	3.2	3/656	1.6	2.07 (0.52–8.28)	**0.573**
With SGLT-2i	2/135	5.7	2/135	5.6	1.08 (0.15–7.72)	
UACR ≤1000 mg/g	4/491	2.8	4/491	2.8	1.03 (0.26–4.14)	**0.254**
UACR >1000 mg/g	4/302	5.0	1/300	1.3	4.31 (0.48–38.67)	
Laboratory-reported: Time to first [K+] >5.5 mmol/L						
Overall	**146/776**	**66.8**	**95/776**	**41.6**	**1.59 (1.23–2.06)**	
Without SGLT-2i	127/643	69.2	87/643	45.8	1.50 (1.14–1.97)	**0.184**
With SGLT-2i	19/133	54.5	8/133	20.8	2.70 (1.18–6.18)	
UACR ≤1000 mg/g	73/478	52.6	58/487	40.1	1.31 (0.93–1.85)	**0.101**
UACR >1000 mg/g	73/298	91.5	37/289	44.1	2.04 (1.37–3.03)	
Laboratory-reported: Time to first [K+] >6.0 mmol/L						
Overall	**34/786**	**14.0**	**18/784**	**7.4**	**1.87 (1.05–3.31)**	
Without SGLT-2i	29/652	14.2	17/650	8.3	1.68 (0.92–3.06)	**0.279**
With SGLT-2i	5/134	13.0	1/134	2.5	5.11 (0.60–43.75)	
UACR ≤1000 mg/g	18/486	11.8	11/489	7.1	1.65 (0.78–3.49)	**0.604**
UACR >1000 mg/g	16/300	17.6	7/295	7.8	2.21 (0.91–5.37)	



*Adverse events that started or worsened after the first dose of study intervention up to 3 days after any temporary or permanent interruption of study intervention were considered TE

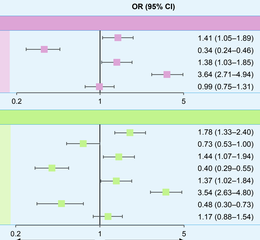
CI, confidence interval; HR, hazard ratio; [K+], potassium concentration; PY, patient-years; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; TE, treatment emergent; UACR, urinary albumin-to-creatinine ratio

Association between baseline characteristics and hyperkalemia

- Several associations were found between baseline characteristics and investigator-reported and laboratory-reported (serum [K+] >5.5 mmol/L) hyperkalemia (**Figure 4**)
  - Baseline serum [K+] >4.5 mmol/L had the strongest association with both investigator-reported and laboratory-reported hyperkalemia (odds ratio [OR]=3.64 [95% CI 2.71–4.94]; p<0.001, and OR=3.54 [95% CI 2.63–4.80]; p<0.001, respectively)
  - Baseline UACR >1000 mg/g increased the odds of both investigator-reported and laboratory-reported hyperkalemia (OR=1.38 [95% CI 1.03–1.85]; p=0.031, and OR=1.37 [95% CI 1.02–1.84]; p=0.039, respectively), as did baseline finerenone use (OR=1.41 [95% CI 1.05–1.89]; p=0.021, and OR=1.78 [95% CI 1.33–2.40]; p<0.001, respectively)
  - Baseline SBP >129 mmHg also increased the odds of laboratory-reported hyperkalemia (OR=1.44 [95% CI 1.07–1.94]; p=0.017)
  - A baseline eGFR ≥45 mL/min/1.73 m² was protective of both investigator-reported and laboratory-reported hyperkalemia (OR=0.34 [95% CI 0.24–0.46]; p<0.001, and OR=0.40 [95% CI 0.29–0.55]; p<0.001, respectively)
  - SGLT-2i use at baseline (OR=0.48 [95% CI 0.30–0.73]; p<0.001), and female sex (OR=0.73 [95% CI 0.53–1.00]; p=0.050) were also protective for laboratory-reported hyperkalemia

Figure 4. Association between baseline characteristics and hyperkalemia (multivariable logistic regression)

Baseline variable	Level	Events (n/N)	OR (95% CI)	p-value
Investigator-reported hyperkalemia				
Treatment group	Finerenone	135/793	1.41 (1.05–1.89)	0.021
eGFR (mL/min/1.73 m²)	≥45	55/740	0.34 (0.24–0.46)	<0.001
UACR (mg/g)	>1000	111/602	1.38 (1.03–1.85)	0.031
Serum [K+] (mmol/L)	>4.5	164/631	3.64 (2.71–4.94)	<0.001
HbA1c (%)*	>5.6	94/625	0.99 (0.75–1.31)	0.959
Laboratory-defined hyperkalemia ([K+] >5.5 mmol/L)				
Treatment group	Finerenone	146/776	1.78 (1.33–2.40)	<0.001
Sex	Female	71/526	0.73 (0.53–1.00)	0.050
SBP (mmHg)	>129	135/761	1.44 (1.07–1.94)	0.017
eGFR (mL/min/1.73 m²)	≥45	64/733	0.40 (0.29–0.55)	<0.001
UACR (mg/g)	>1000	110/587	1.37 (1.02–1.84)	0.039
Serum [K+] (mmol/L)	>4.5	156/606	3.54 (2.63–4.80)	<0.001
SGLT-2i use	Yes	27/266	0.48 (0.30–0.73)	<0.001
HbA1c (%)*	>5.6%	102/610	1.17 (0.88–1.54)	0.276



Covariates selected by backward elimination (likelihood-ratio p<0.1), treatment forced in. The competing risk of 'Death (any)' was counted as a nonevent

*Calculated using univariable logistic regression

BMI, body mass index; CI, confidence interval; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; [K+], potassium concentration; OR, odds ratio; SBP, systolic blood pressure; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UACR, urinary albumin-to-creatinine ratio

Conclusions

- In this subgroup analysis of the FIND-CKD trial, finerenone treatment was associated with an increase in serum [K+] compared with placebo
  - Baseline UACR and SGLT-2i use did not significantly impact treatment-emergent hyperkalemia outcomes
- The incidence of hyperkalemia in patients treated with finerenone in FIND-CKD (in patients with CKD without diabetes) was similar to that reported in FIDELIO-DKD (in patients with CKD and type 2 diabetes); indicating that the absence of diabetes does not confer further protection against hyperkalemia and suggesting the same monitoring rules should apply[3]
- Baseline serum [K+] >4.5 mmol/L, UACR >1000 mg/g, and SBP >129 mmHg were associated with higher odds of hyperkalemia
- Baseline eGFR ≥45 mL/min/1.73 m², SGLT-2i use, and female sex were protective of hyperkalemia

Acknowledgments

FIND-CKD was funded by Bayer AG. Medical writing assistance provided by OHMC and funded by Bayer AG.

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Disclosures

RA has received personal fees and nonfinancial support from Bayer Healthcare Pharmaceuticals; has received personal fees and non-financial support from Akebia Therapeutics, Boehringer Ingelheim, Eli Lilly, and Vifor Pharma; serves as a member of data safety monitoring committees for Chinook Therapeutics and Vertex Pharmaceuticals; serves as a member of steering committees of randomized trials for Akebia Therapeutics and Bayer; serves as Associate Editor for the *American Journal of Nephrology* and *Nephrology Dialysis Transplantation* and as an author for UpToDate; and has received research grants from the National Institutes of Health and US Veterans Administration. **DZC** has received honoraria from Boehringer Ingelheim–Lilly, Merck, AstraZeneca, Sanofi, Mitsubishi-Tanabe, AbbVie, Altimmune, Amgen, Bayer, Biobridge, BMS, CSL Behring, Gilead, GSK, Inversago, Janssen, Lexicon, Maze, Novartis, Novo Nordisk, Otsuka, Prometic, Vantage, and Youngene; and has received operational funding for clinical trials from AstraZeneca, Bayer, Boehringer Ingelheim–Lilly, CSL Behring, Janssen, Lexicon, Merck, Novo Nordisk, and Sanofi. **CSPL** has received research grants from the National Medical Research Council of Singapore, Novo Nordisk, and Roche Diagnostic; has served in advisory, consulting, and trial leadership roles for Alnylam Pharma, AnaCardio, Applied Therapeutics, AstraZeneca, Bayer, Boehringer Ingelheim, Boston Scientific, BridgeBio, Corteria, Corvexus Medical, CPC Clinical Research, Cytokinetics, Daiichi Sankyo, Eli Lilly, EvitaBio, ICON Clinical Research, Impulse Dynamics, Intellia Therapeutics, Janssen Research & Development, Klyv Therapeutics, Medscape/WebMD Global, Merck, Novartis, Novo Nordisk, Pfizer, Radcliffe Group, Ribocure, Roche, Tenax, and Us2.ai; has patent PCT/SG2016/050217 pending and patent US Patent No. US 10,702,247 B2; 10,631,828 B1; US 11,301,996 B2; US 11,446,009 B2; US 11,931,207 B2; US 12,001,939; US 12,400,762 B2; and is a co-founder and non-executive director of Us2.ai. **BLN** has received fees for advisory boards, scientific presentations, steering committee roles, travel, and publication support from AstraZeneca, Alexion, Bayer, Boehringer Ingelheim, Cornerstone Medical Education, CSL Behring, CSL Seqirus, the Limbic, Medscape, Menarini, MJH Life Sciences, Novo Nordisk, Otsuka, Proton Intelligence, Travere Therapeutics, and Vera Therapeutics. **VP** has received fees for an advisory board, steering committee or scientific presentation from AbbVie, Amgen, Astellas, AstraZeneca, Bayer, Boehringer Ingelheim, Chinook Therapeutics, CSL Vifor, Gilead, GSK, Janssen, Medimmune, Mitsubishi-Tanabe, Mundipharma, Novartis, Novo Nordisk, Otsuka, and Travere Therapeutics; has received speaker fees from Janssen; has served on a data and safety monitoring committee for Dimerix; has served on the board of directors for Garvan Institute, George Clinical, George Institute, Ingham Institute, Mindgardens Neuroscience Network, and Victor Chang Cardiac Research Institute; and has stock in George Clinical. **KRT** has received grants from Bayer, Benaroya Research Institute, Breakthrough T1D, CDC, Doris Duke Foundation, NCAT, NHLBI, NIDDK, NIH Director's Office, Otsuka, and Travere Therapeutics; has received consulting fees from Alnylam, AstraZeneca, Bayer, Boehringer Ingelheim, GSK, Novo Nordisk, ProKidney, and Roche; has received speaker fees from AstraZeneca, Bayer, Boehringer Ingelheim, Novo Nordisk, and Travere Therapeutics; has participated in an advisory board for AstraZeneca, George Clarke Institute, and NIDDK; and is a chair at the Diabetic Kidney Disease Collaborative and was a co-chair at ASN Kidney Week 2025 Program Committee. **PS** has received honoraria for advisory boards from Astellas, Bayer, Boehringer Ingelheim, F. Hoffmann-La Roche, PRO.MED.CS, SOBI, Specialty Therapeutics, TEVA, Value Research, and Vantive; has received speaker fees from AstraZeneca, Bayer, Boehringer Ingelheim, Genesis Pharma, Vantive, Vifor Fresenius, and Winmedica; has received research support from AstraZeneca, Boehringer Ingelheim, Genesis Pharma, Menarini, and Vianex; and is a member of trial committees for Alnylam/F. Hoffmann-La Roche, Bayer, and CSL Behring. **MB** is an employee of Bayer AG, Germany and reports ownership interest in Bayer AG. **HJLH** has received honoraria from Bayer for participation in the FINE-ONE clinical trial steering committee (honoraria paid to employer University of Groningen); has received grants from AstraZeneca, Bayer, Boehringer Ingelheim, Janssen, and Novo Nordisk; has received consulting fees from AstraZeneca, Alexion, Alnylam, Amgen, Bayer, Boehringer Ingelheim, BioCity Biopharmaceuticals, Dimerix, Eli Lilly, Novo Nordisk, Novartis, Roche, and Travere Therapeutics; has received speaker fees from AstraZeneca, Bayer, and Novo Nordisk; and has received support for attending meetings and/or travel from AstraZeneca and Eli Lilly.

Poster SA-PO0531 presented at the American Society of Nephrology (ASN) Kidney Week 2026, Denver, CO, USA, October 22–25, 2026