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Finerenone in patients with chronic kidney disease by SGLT-2i treatment: A FIND-CKD analysis

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

Presented by Maria Jose Soler[12]

[1]Division of Nephrology, University Health Network, Toronto General Hospital, University of Toronto, Toronto, ON, Canada; [2]Division of Nephrology, Richard L. Roudebush VA Medical Center, Indianapolis, Indiana University School of Medicine, Indianapolis, IN, USA; [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]First Department of Nephrology, Aristotle University of Thessaloniki, Hippokration Hospital, Thessaloniki, Greece; [10]Department of Clinical Pharmacy and Pharmacology, University of Groningen, University Medical Center Groningen, Groningen, Netherlands; [11]Cardiology and Nephrology Clinical Development, Bayer AG, Wuppertal, Germany; [12]Nephrology and Transplantation Research Group, Vall d'Hebron Institut de Recerca (VHIR), Nephrology Department, Vall d'Hebron Hospital Universitari, Vall d'Hebron Barcelona Hospital Campus, Barcelona, Spain

Corresponding author: David Z. I. Cherney, Division of Nephrology, University Health Network, Toronto General Hospital, University of Toronto, Toronto, ON, Canada, David.Cherney@uhn.ca

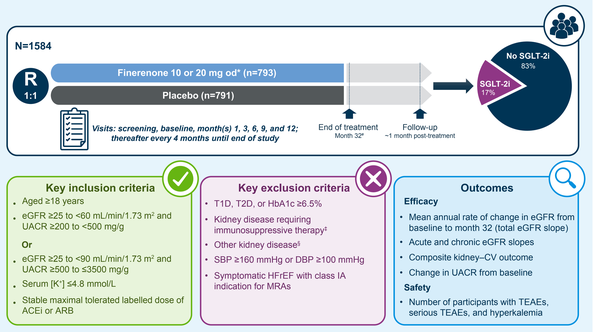
Background

- Management of chronic kidney disease (CKD) associated with type 2 diabetes (T2D) has evolved to incorporate complementary therapies with synergistic mechanisms, such as the nonsteroidal mineralocorticoid receptor antagonist finerenone and sodium-glucose co-transporter-2 inhibitors (SGLT-2is)[1]
  - SGLT-2is have been shown to reduce the risk of kidney and cardiovascular (CV) complications in patients with CKD, regardless of diabetes status[2,3]
  - Finerenone has been shown to slow CKD progression and improve kidney and CV outcomes in patients with CKD, with and without T2D[4–6]
- In the CONFIDENCE (NCT05254002) trial of patients with CKD and T2D, combination treatment with finerenone and SGLT-2is was shown to provide greater urinary albumin-to-creatinine ratio (UACR) reductions versus either monotherapy[7]
- However, the effect of combination treatment with finerenone and SGLT-2is has yet to be established in patients with nondiabetic CKD
- This prespecified subgroup analysis of the FIND-CKD (NCT05047263) trial examined the impact of background SGLT-2i use on kidney and CV risk reduction with finerenone in participants with nondiabetic CKD

Methods

- FIND-CKD randomized adults with nondiabetic CKD (estimated glomerular filtration rate [eGFR] 25 to <90 mL/min/1.73 m^2 and UACR ≥200 to ≤3500 mg/g) treated with a stable, maximum tolerated dose of renin–angiotensin system inhibitor 1:1 to finerenone (10 or 20 mg oral) or placebo[8]
- The study design, inclusion and exclusion criteria, and outcomes are shown in **Figure 1**
  - In the FIND-CKD trial, baseline SGLT-2i inhibitor use (yes vs no) was included as a stratification factor
  - In the current analysis, all outcomes were stratified by SGLT-2i use at baseline

Figure 1. FIND-CKD study design



*For 10 mg od: eGFR ≥25 to <60 mL/min/1.73 m^2; for 20 mg od: eGFR ≥60 mL/min/1.73 m^2; †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/dominant polycystic kidney disease.

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CV, cardiovascular; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; HFrEF, heart failure with reduced ejection fraction; [K+], potassium concentration; 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; TEAE, treatment-emergent adverse event; UACR, urinary albumin-to-creatinine ratio

Results

Baseline characteristics

- Of 1584 participants in FIND-CKD, 17% (n=270; 135 per treatment arm) were using an SGLT-2i at baseline
- Overall, baseline characteristics were broadly similar between participants receiving an SGLT-2i at baseline and those who were not (**Table 1**)
  - However, a greater proportion of participants receiving an SGLT-2i at baseline tended to be White males with higher median UACR compared with those not on an SGLT-2i at baseline

Table 1. Baseline characteristics according to SGLT-2i use

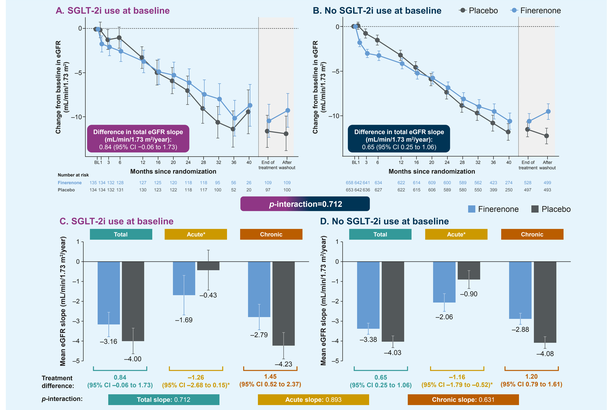
	SGLT-2i use at baseline (n=270)		No SGLT-2i use at baseline (n=1314)	
Characteristics	Finerenone (n=135)	Placebo (n=135)	Finerenone (n=658)	Placebo (n=656)
Age, years, mean ± SD	52.1 ± 14.9	54.0 ± 14.1	55.0 ± 14.0	55.0 ± 14.3
Sex, male, n (%)	**99 (73.3)**	**96 (71.1)**	**419 (63.7)**	**435 (66.3)**
Serum [K+], mmol/L, mean ± SD	4.5 ± 0.4	4.5 ± 0.4	4.5 ± 0.4	4.5 ± 0.4
eGFR, mL/min/1.73 m^2, mean ± SD	46.3 ± 16.9	45.0 ± 14.6	46.9 ± 16.1	47.0 ± 16.3
UACR, mg/g, median (Q1–Q3)	**873.6 (601.8–1368.0)**	**871.9 (648.6–1414.6)**	**818.7 (566.5–1234.5)**	**787.3 (568.6–1190.7)**
SBP, mmHg, mean ± SD	129.0 ± 14.3	127.4 ± 14.5	129.8 ± 13.8	129.6 ± 14.1
Primary cause of CKD, n (%)				
Hypertensive/ischemic nephropathy	30 (22.2)	25 (18.5)	204 (31.0)	200 (30.5)
Chronic glomerulonephritis	**94 (69.6)**	**96 (71.1)**	**352 (53.5)**	**361 (55.0)**
Other/unknown	11 (8.1)	14 (10.4)	102 (15.5)	95 (14.5)
Medication use, n (%)				
ACEi	40 (29.6)	39 (28.9)	190 (28.9)	166 (25.3)
ARB	95 (70.4)	96 (71.1)	465 (70.7)	489 (74.5)
Diuretics	20 (14.8)	26 (19.3)	122 (18.5)	122 (18.6)

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; [K+], potassium concentration; Q, quartile; SBP, systolic blood pressure; SD, standard deviation; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UACR, urinary albumin-to-creatinine ratio

Effect of SGLT-2i use at baseline on eGFR slope

- Finerenone reduced total eGFR decline versus placebo, with no evidence that this effect varied by SGLT-2i use at baseline (*p*-interaction=0.712)
  - The between-arm difference was 0.84 mL/min/1.73 m^2/year (95% CI –0.06 to 1.73; *p*=0.066) in participants receiving an SGLT-2i at baseline (**Figure 2A**) and 0.65 mL/min/1.73 m^2/year (95% CI 0.25 to 1.06; *p*=0.001) in participants not receiving an SGLT-2i at baseline (**Figure 2B**)
- As with the total eGFR slope, there was no significant heterogeneity in the effect of finerenone versus placebo on the acute or chronic eGFR slopes in participants with (**Figure 2C**) or without (**Figure 2D**) SGLT-2i use at baseline (*p*-interaction=0.893 and 0.631, respectively)
- Over the course of the FIND-CKD trial, participants receiving placebo were more likely to start an SGLT-2i (n/N=199/791, 25.2%) compared with finerenone-treated participants (n/N=144/793, 18.2%). In time-varying models taking into account initiation of SGLT-2i post-randomization, SGLT-2i exposure did not influence the effect of finerenone compared with placebo on total eGFR slope (*p*-interaction=0.745) (data not shown)

Figure 2. Change from baseline in eGFR according to SGLT-2i use at baseline



Mean change in eGFR is shown from baseline to month 40 for participants with (**A**) and without (**B**) SGLT-2i use. The annual rate of change in eGFR from baseline to month 32 (total slope), mean rate of change in eGFR from baseline to month 3 (acute slope), and mean annual rate of change in eGFR from month 3 to end of treatment (chronic slope) with (**C**) and without (**D**) SGLT-2i use entered as a time-varying on/off covariate

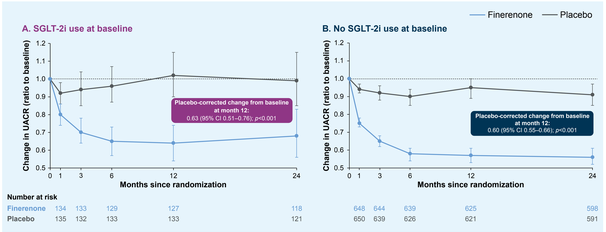
*Reported in mL/min/1.73 m^2/3 months

CI, confidence interval; eGFR, estimated glomerular filtration rate; SGLT-2i, sodium-glucose co-transporter-2 inhibitor

Effect of SGLT-2i use at baseline on UACR over time

- Finerenone reduced UACR versus placebo from baseline to month 12 regardless of SGLT-2i use at baseline (*p*-interaction=0.753)
  - At month 12, finerenone reduced UACR by 37% and 40% versus placebo in baseline SGLT-2i users and non-users, respectively (**Figures 3A and B**)

Figure 3. Change in UACR over time according to SGLT-2i use at baseline



Change in UACR from baseline to month 24 in participants with (**A**) or without (**B**) SGLT-2i use at baseline treated with finerenone or placebo

CI, confidence interval; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UACR, urinary albumin-to-creatinine ratio

Effect of SGLT-2i use at baseline on composite kidney–CV outcome

- Finerenone lowered the overall composite kidney–CV outcome versus placebo (hazard ratio [HR]=0.77; 95% CI 0.60–0.99) and was not modified by SGLT-2i use at baseline (*p*-interaction=0.548) (**Figure 4**)
  - SGLT-2i use at baseline did not affect the incidence of the composite kidney and/or CV outcomes with finerenone in participants with nondiabetic CKD (*p*-interaction>0.05)

Figure 4. Composite kidney and/or CV outcomes in participants treated with finerenone or placebo according to SGLT-2i use at baseline

Outcome	Finerenone Events, n/N	Finerenone Rate/1000 PY	Placebo Events, n/N	Placebo Rate/1000 PY	HR (95% CI)	*p*-interaction
Composite kidney–CV outcome (≥57% eGFR decline,* kidney failure,# HHF, or CV death)						
Overall	**110/793**	**46.9**	**134/791**	**58.3**	**0.77 (0.60–0.99)**	
With SGLT-2i	21/135	57.3	23/135	62.8	0.85 (0.47–1.57)	**0.548**
Without SGLT-2i	89/658	45.0	111/656	57.4	0.75 (0.57–1.00)	
Composite kidney outcome (≥57% eGFR decline,* kidney failure#)						
Overall	**104/793**	**44.2**	**128/791**	**54.2**	**0.78 (0.60–1.01)**	
With SGLT-2i	21/135	57.3	22/135	59.7	0.91 (0.49–1.68)	**0.448**
Without SGLT-2i	83/658	41.8	103/656	53.1	0.76 (0.57–1.01)	
Composite kidney outcome (≥40% eGFR decline,* kidney failure#)						
Overall	**168/793**	**73.6**	**206/791**	**92.8**	**0.76 (0.62–0.93)**	
With SGLT-2i	29/135	81.8	33/135	93.0	0.85 (0.51–1.41)	**0.519**
Without SGLT-2i	139/658	72.1	173/656	92.7	0.74 (0.59–0.93)	
Composite CV outcome (HHF or CV death)						
Overall	**10/793**	**3.9**	**16/791**	**6.4**	**0.60 (0.27–1.33)**	
With SGLT-2i	0/135	0.0	2/135	5.0	N/A‡	**N/A‡**
Without SGLT-2i	10/658	4.7	14/656	6.6	0.70 (0.31–1.57)	

0.25 0.5 1.0 2.0
Favors finerenone Favors placebo

Time-to-event outcomes were analyzed using stratified log-rank tests and stratified Cox proportional hazards models to estimate HRs with two-sided 95% CIs

*From baseline, sustained for ≥4 weeks; #defined as an eGFR of <15 mL/min/1.73 m^2, initiation of chronic dialysis, or kidney transplantation; ‡HR was not possible to calculate given that there were no events reported in the placebo arm of the SGLT-2i user group

CI, confidence interval; CV, cardiovascular; eGFR, estimated glomerular filtration rate; HHF, hospitalization for heart failure; HR, hazard ratio; PY, patient-years; SGLT-2i, sodium-glucose co-transporter-2 inhibitor

Adverse events according to SGLT-2i use at baseline

- Overall, the incidence of adverse events did not differ according to SGLT-2i use at baseline (*p*-interaction=0.835) (**Table 2**)
- The incidence of hyperkalemia was higher with finerenone than placebo in participants with (19.3% vs 11.1%) and without (16.6% vs 13.7%) SGLT-2i use at baseline, respectively
  - Risk of hyperkalemia was not modified by SGLT-2i use (*p*-interaction=0.223)
  - In finerenone-treated participants, hyperkalemia leading to treatment discontinuation or hospitalization was low (**Table 2**)

Table 2. AEs* by SGLT-2i use at baseline

	SGLT-2i use at baseline (n=270)		No SGLT-2i use at baseline (n=1314)	
AE, n (%)	Finerenone	Placebo	Finerenone	Placebo
Investigator-reported	**(n=135)**	**(n=135)**	**(n=658)**	**(n=656)**
Any AE*	88 (65.2)	88 (65.2)	456 (69.3)	431 (65.7)
Leading to permanent discontinuation	5 (3.7)	5 (3.7)	13 (2.0)	18 (2.7)
Any serious AE*	22 (16.3)	32 (23.7)	147 (22.3)	139 (21.2)
Leading to death	1 (0.7)	2 (1.5)	5 (0.8)	9 (1.4)
Any hyperkalemia#	26 (19.3)	15 (11.1)	109 (16.6)	90 (13.7)
Leading to permanent discontinuation	4 (3.0)	0	8 (1.2)	1 (0.2)
Any serious hyperkalemia#	2 (1.5)	2 (1.5)	6 (0.9)‡	3 (0.5)
Leading to hospitalization	2 (1.5)	2 (1.5)	5 (0.8)	3 (0.5)
Laboratory-defined	**(n=133)**	**(n=133)**	**(n=643)**	**(n=643)**
Hyperkalemia, serum [K+] >5.5 mmol/L	19 (14.3)	8 (6.0)	127 (19.8)	87 (13.5)

*AEs 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 treatment emergent; #hyperkalemia included AEs with MedDRA codes 10020646 (Hyperkalemia) and 10005725 (Blood potassium increased); ‡one serious hyperkalemia event in the finerenone group without SGLT-2i use at baseline was classified as life-threatening but did not require hospitalization

AE, adverse event; [K+], potassium concentration; MedDRA, Medical Dictionary for Regulatory Activities; SGLT-2i, sodium-glucose co-transporter-2 inhibitor

Conclusions

- Finerenone reduced eGFR decline, UACR, and the risk of kidney and CV outcomes in participants with nondiabetic CKD
- Estimates in SGLT-2i users were directionally consistent with the overall FIND-CKD trial but were imprecise; there was no evidence of heterogeneity between SGLT-2i users and non-users
- Adverse event profiles did not differ according to SGLT-2i use at baseline, with low rates of hyperkalemia-related discontinuation and hospitalization

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Disclosures

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