TH-PO1240

# Finerenone in patients with focal segmental glomerulosclerosis: A FIND-CKD analysis

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## Background

- Focal segmental glomerulosclerosis (FSGS) encompasses heterogeneous glomerular diseases characterized by podocyte injury and segmental glomerular scarring[1]
  - FSGS is associated with impaired quality of life and increased risk of kidney failure[1]
- Proteinuria is a hallmark of FSGS and an important marker of disease progression[1,2]
  - Reduction in proteinuria is associated with preservation of kidney function and a lower long-term risk of kidney failure[1,2]
- Among adults with nondiabetic chronic kidney disease (CKD) in the FIND-CKD trial, finerenone slowed estimated glomerular filtration rate (eGFR) decline, reduced the risk of a composite kidney–cardiovascular (CV) outcome and reduced urinary protein-to-creatinine ratio (UPCR) vs placebo[3]
- This prespecified FIND-CKD subanalysis examined the efficacy and safety of finerenone in participants with nondiabetic CKD and investigator-reported FSGS at baseline

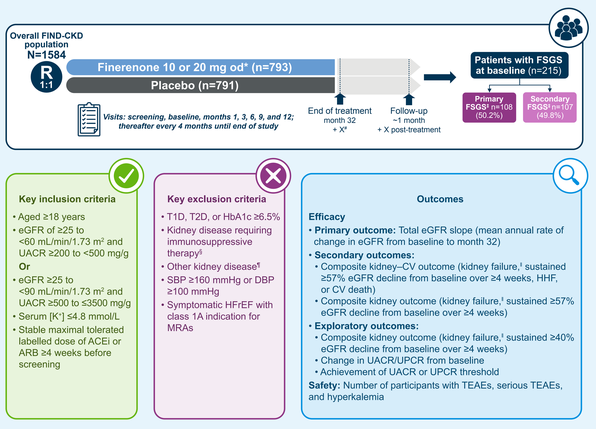
## Methods

- FIND-CKD was a phase III, international, prospective, randomized, double-blind, placebo-controlled trial in adults with nondiabetic CKD (NCT05047263)[3,4]
- The study design, inclusion, and exclusion criteria for the FIND-CKD trial and outcomes assessed in this subanalysis are shown in **Figure 1**
  - Participants included in this subanalysis had investigator-reported FSGS at baseline

### Statistical analysis

- Efficacy analyses were conducted in the full analysis set, comprising all randomized participants with investigator-reported FSGS at baseline, analyzed according to randomized treatment assignment
- Safety analyses were conducted in the safety analysis set, comprising participants with FSGS who received at least one dose of study treatment

**Figure 1.** Study design and outcomes




*10 mg od: eGFR ≥25 to <60 mL/min/1.73 m²; 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; ‡primary FSGS is considered an immune-mediated podocytopathy typically treated with immunosuppression, whereas secondary FSGS is generally managed with supportive therapies, including renin–angiotensin system blockade; §lupus nephritis, anti-neutrophil cytoplasmic antibody-associated vasculitis or any other kidney disease within 6 months prior to screening; ¶known autosomal dominant or recessive cystic kidney disease; ‖kidney failure was defined as eGFR <15 mL/min/1.73 m², initiation of chronic dialysis or kidney transplantation

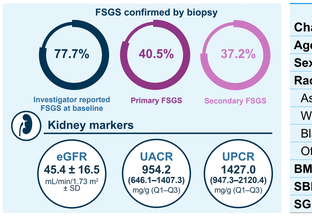
ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CV, cardiovascular; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; FSGS, focal segmental glomerulosclerosis; HbA1c, glycated hemoglobin; HFrEF, heart failure with reduced ejection fraction; HHF, hospitalization for heart failure; [K⁺], potassium concentration; MRA, mineralocorticoid receptor antagonist; od, once daily; R, randomized; SBP, systolic blood pressure; T1D, type 1 diabetes; T2D, type 2 diabetes; TEAE, treatment-emergent adverse event; UACR, urinary albumin-to-creatinine ratio; UPCR, urinary protein-to-creatinine ratio

## Results

### Baseline characteristics

- Of 1584 participants randomized in FIND-CKD, 215 (13.6%) had investigator-reported FSGS at baseline, with 45.6% (n=98) receiving finerenone and 54.4% (n=117) receiving placebo (**Table 1**)
  - A total of 167 (77.7%) had FSGS confirmed by kidney biopsy
  - Overall, 108 participants (50.2%) had primary FSGS and 107 (49.8%) had secondary FSGS
- At baseline, mean eGFR in participants with FSGS was 45.4 mL/min/1.73 m²; median urinary albumin-to-creatinine ratio (UACR) and UPCR were 954.2 mg/g and 1427.0 mg/g, respectively

**Table 1.** Baseline characteristics of participants with FSG




| Characteristic | FSGS at baseline (n=215) |
|---|---|
| **Age**, years, mean ± SD | 52.6 ± 14.0 |
| **Sex**, male, n (%) | 142 (66.0) |
| **Race**, n (%) | |
| Asian | 89 (41.4) |
| White | 111 (51.6) |
| Black | 4 (1.9) |
| Other* | 11 (5.1) |
| **BMI**, kg/m², mean ± SD | 28.5 ± 6.1 |
| **SBP**, mmHg, mean ± SD | 128.8 ± 14.7 |
| **SGLT-2i use**, n (%) | 59 (27.4) |

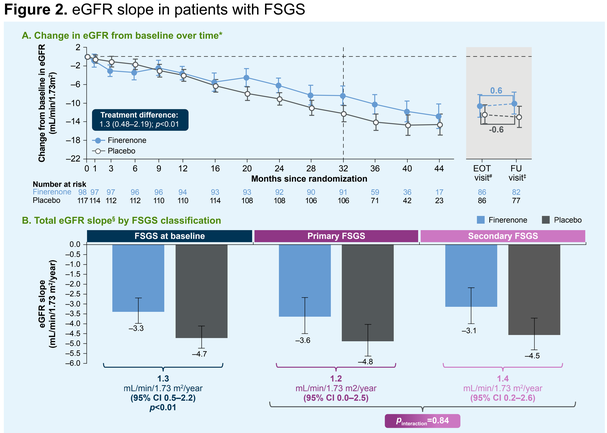
*Other includes participants for whom multiple races were recorded

BMI, body mass index; eGFR, estimated glomerular filtration rate; FSGS, focal segmental glomerulosclerosis; Q1, first quartile; Q3, third quartile; SBP, systolic blood pressure; SD, standard deviation; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UACR, urinary albumin-to-creatinine ratio; UPCR, urinary protein-to-creatinine ratio

### Total eGFR slope in the overall FSGS subgroup and by FSGS classification

- From baseline to month 32, finerenone reduced total eGFR decline by 1.3 mL/min/1.73 m²/year (95% confidence interval [CI] 0.5–2.2; $p<0.01$) (**Figure 2A**)
- The effect of finerenone on total eGFR slope was consistent in participants with primary and secondary FSGS ($p$-interaction=0.84; **Figure 2B**)

**Figure 2.** eGFR slope in patients with FSGS



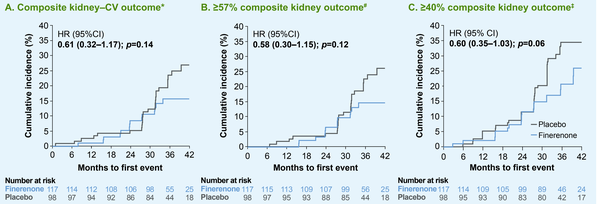

*eGFR slope from baseline to month 32 was estimated using multiple imputation. Error bars represent 95% CIs; †the EOT visit took place at the end of the treatment phase, when the last participants who had undergone randomization had received finerenone or placebo for 32 months; ‡the follow-up visit took place approximately 4 weeks after the last administration of finerenone or placebo; §total slope was defined as mean annual rate of change in eGFR from baseline to month 32 and was analyzed using a two-slope linear spline mixed-effects model with a fixed change point at month 3. Fixed effects included treatment, time, treatment-by-time interaction, baseline eGFR, screening UACR stratum and baseline SGLT-2i use; random effects included intercept, acute slope, and chronic slope. Treatment-by-subgroup interaction terms assessed heterogeneity between primary and secondary FSGS

CI, confidence interval; eGFR, estimated glomerular filtration rate; EOT, end of treatment; FSGS, focal segmental glomerulosclerosis; FU, follow-up; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UACR, urinary albumin-to-creatinine ratio

### Composite time-to-event outcomes

- Finerenone numerically reduced the risk of the composite kidney–CV outcome by 39% (hazard ratio [HR]=0.61 [95% CI 0.32–1.17]; $p=0.14$) and the ≥57% composite kidney outcome by 42% (HR=0.58 [95% CI 0.30–1.15]; $p=0.12$) versus placebo (**Figures 3A** and **3B**)
  - The incidence of hospitalization for heart failure or CV death was low, with 5 events reported overall (finerenone, n=2; placebo, n=3)
- Similar results were obtained for the exploratory ≥40% composite kidney outcome (HR=0.60 [95% CI 0.35–1.03]; $p=0.06$; **Figure 3C**)

**Figure 3.** Cumulative incidence of the kidney–CV and kidney composite outcomes



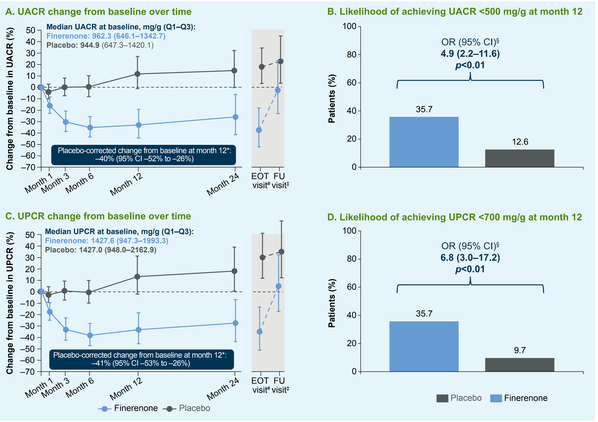

*The kidney–CV composite comprised sustained ≥57% eGFR decline from baseline for ≥4 weeks, kidney failure (defined as eGFR <15 mL/min/1.73 m², initiation of chronic dialysis or kidney transplantation), HHF, or CV death; †the kidney composite comprised sustained ≥57% eGFR decline from baseline for ≥4 weeks or kidney failure; ‡the exploratory kidney composite comprised sustained ≥40% eGFR decline from baseline for ≥4 weeks or kidney failure. HRs and two-sided 95% CIs were estimated using stratified Cox proportional-hazards regression; $p$-values were obtained using stratified log-rank tests

CI, confidence interval; CV, cardiovascular; eGFR, estimated glomerular filtration rate; HHF, hospitalization for heart failure; HR, hazard ratio

### UACR and UPCR

- Finerenone reduced UACR by 40% vs placebo from baseline to month 12 (95% CI 26%–52%; **Figure 4A**)
  - Participants receiving finerenone were more likely to achieve UACR <500 mg/g at month 12 compared with placebo (odds ratio [OR]=4.9 [95% CI 2.2–11.6]; $p<0.01$; **Figure 4B**)
- Finerenone reduced UPCR by 41% vs placebo from baseline to month 12 (95% CI 26%–53%; **Figure 4C**)
  - Participants receiving finerenone were more likely to achieve UPCR <700 mg/g at month 12 (OR=6.8 [95% CI 3.0–17.2]; $p<0.01$) (**Figure 4D**)

**Figure 4.** Changes in UACR and UPCR from baseline over time and achievement of thresholds at month 12



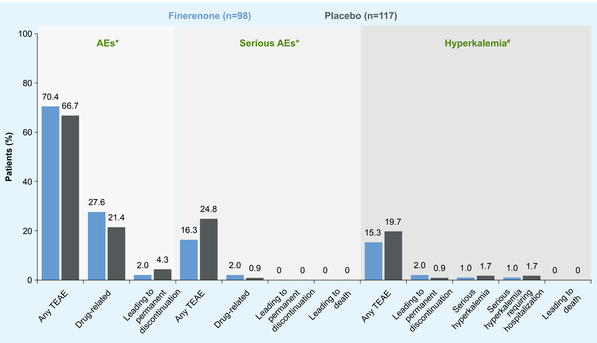

*Month 12 values are model-estimated percentage changes from baseline; †the EOT visit took place at the end of the treatment phase, when the last participants who had undergone randomization had received finerenone or placebo for 32 months; ‡the follow-up visit took place approximately 4 weeks after the last administration of finerenone or placebo; §ORs were estimated using logistic regression adjusted for log-transformed baseline UACR or UPCR. An OR >1 favors finerenone

CI, confidence interval; EOT, end of treatment; FU, follow-up; OR, odds ratio; Q1, first quartile; Q3, third quartile; UACR, urinary albumin-to-creatinine ratio; UPCR, urinary protein-to-creatinine ratio

### Safety

- Among participants with FSGS, the incidence of treatment-emergent adverse events was generally balanced between treatment arms (**Figure 5**)
  - Hyperkalemia was reported in 15 (15.3%) participants receiving finerenone and 23 (19.7%) receiving placebo
  - Serious hyperkalemia and events requiring hospitalization or permanent treatment discontinuation were uncommon

**Figure 5.** Safety outcomes in patients with FSGS




*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 TEAEs. A serious AE could fulfill more than one reason for seriousness; #hyperkalemia was an AESI and defined as any investigator-reported AEs with MedDRA codes corresponding to the preferred terms hyperkalemia or blood potassium increased

AE, adverse event; AESI, adverse event of special interest; FSGS, focal segmental glomerulosclerosis; MedDRA, Medical Dictionary for Regulatory Activities; TEAE, treatment-emergent adverse event

## Conclusions

- Finerenone significantly slowed total eGFR decline from baseline to month 32 versus placebo in participants with FSGS, with a consistent treatment effect in primary and secondary FSGS
- Kidney and CV outcome HRs favored finerenone but the differences were not nominally significant
- Finerenone reduced UACR and UPCR at month 12 and increased the likelihood of achieving UACR <500 mg/g or UPCR <700 mg/g versus placebo
- Finerenone was well tolerated, with a safety profile that was broadly comparable with placebo

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