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Efficacy and safety of finerenone in Asian patients with type 2 diabetes and chronic kidney disease: A FIDELITY analysis by baseline kidney function

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Introduction

- Type 2 diabetes (T2D) is a known cause of chronic kidney disease (CKD); CKD affects approximately 40% of patients with T2D[1]
- T2D and CKD are associated with high risks of cardiorenal morbidity and mortality, and these risks increase as CKD worsens[2]
- Regional analyses have shown that T2D and CKD are significant health burdens in Asia
  - Asian patients account for over 50% of the global diabetic population, with an expected prevalence of over 355 million by 2040[3,4]
  - Asia also has the greatest burden of diabetes-associated CKD worldwide, with age-standardised incidence rates increasing annually since 1990[4]
- CKD is defined by both low estimated glomerular filtration rate (eGFR) and raised albuminuria[5]
  - Albuminuria is one of the earliest signs of progressive CKD[6] and one of the strongest risk factors for end-stage kidney disease in Asian patients[7]
  - When considered together, high albuminuria and low eGFR are associated with a range of cardiovascular (CV) outcomes, as well as all-cause hospitalisation and early mortality[8,9]; this association is stronger as eGFR decreases and albuminuria levels increase[9]
- FIDELITY, a prespecified pooled analysis of two phase III trials, demonstrated that finerenone reduced the risk of adverse kidney and CV outcomes versus placebo in patients with CKD and T2D[2]
  - Similar reductions in CV and kidney outcomes were reported in Asian populations receiving finerenone despite these patients having more advanced CKD at baseline than the overall trial population[10,11]
- This FIDELITY post hoc subanalysis assessed the effect of finerenone on chronic eGFR slope and time to urine albumin-to-creatinine ratio (UACR) regression in Asian patient subgroups

Methods

Study design and patient population

- The prespecified FIDELITY pooled analysis combined individual patient-level data from the FIDELIO-DKD (NCT02540993) and FIGARO-DKD (NCT02545049) phase III, randomised, double-blind, placebo-controlled, multicentre clinical trials
  - The designs and results of these studies have been published previously[2,12–14]
- Race was reported by self-identification (Asian, White, Black or African American, American Indian or Alaska Native, or Native Hawaiian or Other Pacific Islander)
  - Patients who identified as multiple races were considered non-Asian
  - Patients from Asian and non-Asian countries and territories were included if they self-identified as Asian
  - The Asian countries included in this analysis were: China, Hong Kong, Japan, South Korea, Malaysia, The Philippines, Singapore, Thailand, Taiwan and Vietnam

Randomisation and masking

- Patients with CKD and T2D, and on maximum dose of renin–angiotensin system blockade were randomised 1:1 to receive once-daily oral finerenone (10 or 20 mg) or matching placebo
- Stratification factors for the prespecified FIDELITY pooled analysis included study (FIDELIO-DKD or FIGARO-DKD), region (North America, Europe, Asia, Latin America, other), albuminuria at screening (moderately or severely increased), eGFR at screening (25 to <45, 45 to <60 and ≥60 ml/min/1.73 m²) and a history of CV disease (present or absent)
- Up-titration of dose was allowed after 1 month if serum potassium levels were ≤4.8 mmol/l and eGFR was stable; down-titration was permitted as needed any time after treatment initiation

Outcomes

- Efficacy outcomes in this analysis included:
  - Chronic eGFR slopes from month 4 to permanent treatment discontinuation or the end of study
  - UACR change from baseline to month 4
  - Time to UACR regression from 'high' to 'normal', 'very high' to 'normal' and 'very high' to 'high'
- All UACR analyses were assessed by the following parameters at baseline: UACR (<300 and ≥300 mg/g); eGFR (<60 and ≥60 ml/min/1.73 m²); systolic blood pressure (SBP; <130, 130 to <160 and ≥160 mmHg); glycated haemoglobin (HbA1c; ≤7.5% and >7.5%); body mass index (BMI; <30 and ≥30 kg/m²); sodium-glucose co-transporter-2 inhibitor (SGLT-2i) use (yes and no); and glucagon-like peptide-1 receptor agonist (GLP-1RA) use (yes and no)
- Safety outcomes included treatment-emergent adverse events (TEAEs) and were assessed by eGFR subgroups (<60 and ≥60 ml/min/1.73 m²)

Statistical analysis

- Patient demographics and efficacy outcomes were conducted on the full analysis set, which comprised Asian patients without critical Good Clinical Practice (GCP) violations
- Safety outcomes were conducted on the safety analysis set, comprising Asian patients who were administered ≥1 dose of the study drug and were without critical GCP violations
- Chronic eGFR slope (month 4 to permanent treatment discontinuation or end of study) was evaluated using an analysis of covariance model
- Change in UACR was investigated based on summary statistics from baseline to month 4 (or closest visit to month 4)
- Regression from one albuminuria category to another was investigated by a Cox model with study, geographic region, eGFR and albuminuria category at screening, and a history of CV disease as stratification factors
- Albuminuria categories were defined as: 'very high' albuminuria for UACR ≥300 mg/g, 'high' albuminuria for UACR ≥30 to <300 mg/g, and 'normal' albuminuria for UACR <30 mg/g
- Treatment effects were expressed as the hazard ratio (HR) with corresponding 95% confidence intervals (CIs)
- All analyses were performed with the use of SAS software, version 9.4 (SAS Institute)

Results

Patient characteristics

- Of the 12,990 patients in the FIDELITY pooled analysis, 22.0% (n=2858) were Asian
- Baseline characteristics by baseline eGFR of ≥60 ml/min/1.73 m² and <60 ml/min/1.73 m² were mainly balanced between these subgroups, with some noticeable differences:
  - Patients with a baseline eGFR of ≥60 ml/min/1.73 m² were younger, had a shorter duration of diabetes, reported greater use of SGLT-2is at baseline, and had a higher UACR compared with patients with a baseline eGFR of <60 ml/min/1.73 m² (**Table 1**)

Table 1. Baseline demographic and clinical characteristics in the FIDELITY Asian subpopulation by baseline eGFR status

	Asian subpopulation (n=2858)	
Characteristics	**eGFR <60 ml/min/1.73 m² (n=1835)**	**eGFR ≥60 ml/min/1.73 m² (n=1023)**
Age, years, mean (SD)	64.4 (9.5)	58.5 (10.5)
Sex, male, n (%)	1340 (73.0)	765 (74.8)
SBP, mmHg, mean (SD)	134.0 (14.9)	134.6 (14.3)
DBP, mmHg, mean (SD)	73.7 (10.1)	77.5 (9.6)
BMI, kg/m², mean (SD)	27.0 (4.1)*	27.7 (4.4)
Duration of diabetes, years, mean (SD)	15.8 (8.9)*	13.7 (7.7)*
HbA1c, %, mean (SD)	7.5 (1.2)*	7.7 (1.3)*
Serum potassium, mmol/l, mean (SD)	4.3 (0.4)	4.2 (0.4)
eGFR, ml/min/1.73 m², mean (SD)	42.9 (9.7)	77.7 (13.9)
eGFR, ml/min/1.73 m², n (%)		
<25	37 (2.0)	0
25 to <45	970 (52.9)	0
45 to <60	828 (45.1)	0
≥60	0	1023 (100)
UACR, mg/g, median, IQR (Q1–Q3)	606.2 (210.3–1431.2)	659.1 (352.0–1220.1)
UACR, mg/g, n (%)		
<30	29 (1.6)	8 (0.8)
30 to <300	559 (30.5)	206 (20.1)
≥300	1247 (68.0)	809 (79.1)
Medication use at baseline, n (%)		
SGLT-2is	80 (4.4)	101 (9.9)
Alpha-glucosidase inhibitors	316 (17.2)	208 (20.3)

*n ≤9 missing

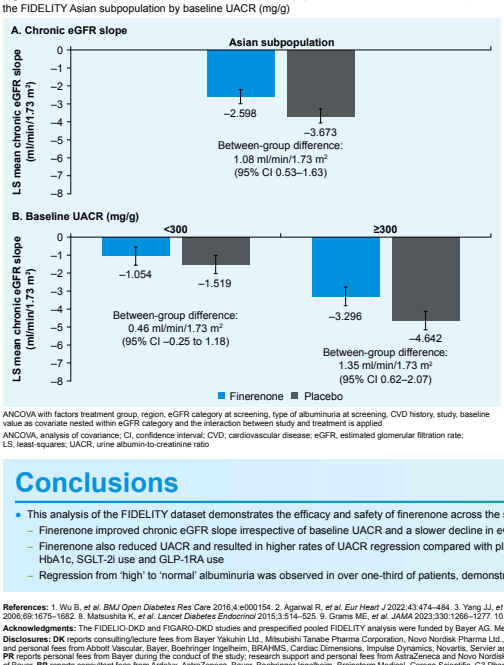
BMI, body mass index; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; HbA1c, glycated haemoglobin; IQR, interquartile range; SBP, systolic blood pressure; SD, standard deviation; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UACR, urine albumin-to-creatinine ratio

- From the Asian population, 921 of 1412 (65.2%) patients initiated treatment on finerenone 10 mg versus 491 of 1412 (34.8%) patients who initiated treatment on finerenone 20 mg
  - Out of the 921 patients who started on the 10-mg dose, a total of 159 patients (17.3%) were never up-titrated and 762 patients (82.7%) were up-titrated at least once

Chronic eGFR slope

- Finerenone improved chronic eGFR slope between month 4 and permanent treatment discontinuation (or end of study) compared with placebo in the Asian population (**Figure 1A**)
- When analysed by UACR subgroups, improvements in chronic eGFR slope were also observed with finerenone compared with placebo in the Asian subpopulation (**Figure 1B**); least-squares mean differences were:
  - In patients with baseline UACR <300 mg/g: 0.46 ml/min/1.73 m² (95% CI –0.25 to 1.18)
  - In patients with baseline UACR ≥300 mg/g: 1.35 ml/min/1.73 m² (95% CI 0.62–2.07)

Figure 1. (**A**) Chronic eGFR slope in the FIDELITY Asian subpopulation and (**B**) chronic eGFR slope in the FIDELITY Asian subpopulation by baseline UACR (mg/g)



ANCOVA with factors treatment group, region, eGFR category at screening, type of albuminuria at screening, CVD history, study, baseline value as covariate nested within eGFR category and the interaction between study and treatment is applied
ANCOVA, analysis of covariance; CI, confidence interval; CVD, cardiovascular disease; eGFR, estimated glomerular filtration rate; LS, least-squares; UACR, urine albumin-to-creatinine ratio

UACR reduction and regression

- In the overall Asian population, treatment with finerenone reduced UACR from baseline to month 4, representing a geometric mean reduction of 34% (**Table 2**)
  - Finerenone also reduced UACR from baseline to month 4 by 31–42% regardless of baseline eGFR and UACR, SBP, HbA1c, BMI, SGLT-2i use and GLP-1RA use (**Table 2**)

Table 2. Summary statistics for change in UACR from baseline to month 4 in the FIDELITY Asian subpopulation

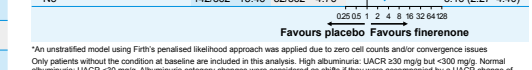
		Value at baseline		Value at month 4		Ratio to baseline at month 4	
		n	Geometric mean (SD)	n	Geometric mean (SD)	n	Geometric mean (SD)
Overall	Finerenone	1412	526.2 (3.4)	1383	345.0 (4.0)	1383	0.7 (1.9)
	Placebo	1446	555.0 (3.5)	1404	546.1 (3.7)	1404	1.0 (1.8)
eGFR (<60 ml/min/1.73 m²)	Finerenone	911	500.1 (3.7)	893	333.7 (4.2)	893	0.7 (1.9)
	Placebo	924	524.7 (3.8)	893	528.1 (4.0)	893	1.0 (1.8)
eGFR (≥60 ml/min/1.73 m²)	Finerenone	501	577.1 (2.9)	490	366.8 (3.5)	490	0.6 (2.0)
	Placebo	522	612.9 (2.9)	511	579.1 (3.2)	511	1.0 (1.7)
UACR (<300 mg/g)	Finerenone	401	107.0 (2.0)	395	71.6 (2.5)	395	0.7 (2.1)
	Placebo	401	110.3 (2.1)	393	119.0 (2.6)	393	1.1 (1.9)
UACR (≥300 mg/g)	Finerenone	1011	989.7 (2.0)	988	647.1 (2.6)	988	0.7 (1.9)
	Placebo	1045	1031.6 (2.1)	1011	987.3 (2.4)	1011	1.0 (1.7)
SBP (<130 mmHg)	Finerenone	521	457.7 (3.3)	512	317.3 (3.8)	512	0.7 (2.0)
	Placebo	538	456.9 (3.5)	530	462.8 (3.8)	530	1.0 (1.8)
SBP (130 to <160 mmHg)	Finerenone	856	561.5 (3.5)	837	358.0 (4.1)	837	0.7 (1.9)
	Placebo	866	613.2 (3.4)	833	595.7 (3.6)	833	1.0 (1.8)
SBP (≥160 mmHg)	Finerenone	35	855.2 (3.0)	34	493.1 (3.6)	34	0.6 (1.8)
	Placebo	42	856.3 (3.2)	41	795.3 (3.4)	41	0.9 (1.6)
HbA1c (≤7.5%)	Finerenone	817	487.6 (3.5)	800	326.3 (4.1)	800	0.7 (1.9)
	Placebo	830	526.2 (3.6)	808	515.9 (3.9)	808	1.0 (1.7)
HbA1c (>7.5%)	Finerenone	594	584.2 (3.3)	582	372.7 (3.8)	582	0.6 (1.9)
	Placebo	612	597.1 (3.3)	592	592.6 (3.4)	592	1.0 (1.7)
BMI (<30 kg/m²)	Finerenone	1115	532.0 (3.4)	1090	346.1 (3.9)	1090	0.7 (1.9)
	Placebo	1126	543.9 (3.6)	1093	536.0 (3.9)	1093	1.0 (1.7)
BMI (≥30 kg/m²)	Finerenone	295	509.2 (3.4)	291	342.8 (4.1)	291	0.7 (2.0)
	Placebo	320	595.8 (3.0)	311	583.1 (3.1)	311	1.0 (1.8)
SGLT-2i (yes)	Finerenone	91	429.8 (3.0)	91	258.3 (4.1)	91	0.6 (2.1)
	Placebo	90	442.2 (3.7)	87	431.0 (3.7)	87	1.0 (1.6)
SGLT-2i (no)	Finerenone	1321	533.6 (3.5)	1292	352.1 (3.9)	1292	0.7 (1.9)
	Placebo	1356	563.4 (3.5)	1317	554.7 (3.7)	1317	1.0 (1.8)
GLP-1RA (yes)	Finerenone	79	372.6 (3.8)	78	253.7 (4.0)	78	0.7 (1.9)
	Placebo	71	537.1 (3.1)	67	549.8 (3.4)	67	1.0 (1.9)
GLP-1RA (no)	Finerenone	1333	537.1 (3.4)	1305	351.4 (4.0)	1305	0.7 (1.9)
	Placebo	1375	555.9 (3.5)	1337	545.9 (3.7)	1337	1.0 (1.7)

BMI, body mass index; eGFR, estimated glomerular filtration rate; GLP-1RA, glucagon-like peptide-1 receptor agonist; HbA1c, glycated haemoglobin; SBP, systolic blood pressure; SD, standard deviation; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UACR, urine albumin-to-creatinine ratio

- Regression from 'high' to 'normal' albuminuria was seen in 39.5% of Asian patients treated with finerenone versus 14.8% receiving placebo (HR=3.04; 95% CI 2.21–4.18) (**Figure 2**)
  - This treatment effect was similar across the different baseline subgroups (**Figure 2**)
- Regression from 'very high' to 'normal' albuminuria (3.8% vs 1.7%; HR=2.27; 95% CI 1.28–4.05) and from 'very high' to 'high' albuminuria (42.3% vs 20.3%; HR=2.69; 95% CI 2.27–3.19) was seen more frequently in Asian patients treated with finerenone versus patients receiving placebo

Figure 2. UACR regression from 'high' to 'normal' albuminuria in the FIDELITY Asian subpopulation and by subgroups

	Finerenone		Placebo		
Outcome	**n/N**	**IR per 100 PY**	**n/N**	**IR per 100 PY**	**HR (95% CI)**
Overall Asian population	153/387	15.61	56/378	4.93	3.04 (2.21–4.18)
eGFR at baseline (ml/min/1.73 m²)					
<60	116/283	16.50	41/276	5.11	3.02 (2.09–4.35)
≥60	37/104	13.37	15/102	4.48	3.41 (1.76–6.61)
SBP at baseline (mmHg)					
<130	53/160	12.97	28/164	5.62	2.22 (1.34–3.70)
130 to <160	97/223	17.33	26/204	4.26	3.85 (2.46–6.03)
≥160	3/4	26.08	2/10	6.98	2.94 (0.41–21.25)*
HbA1c at baseline (%)					
≤7.5	101/239	16.65	36/233	5.18	2.73 (1.85–4.04)
>7.5	52/148	13.94	20/144	4.55	3.65 (2.06–6.46)
BMI at baseline					
<30 kg/m²	119/307	15.07	45/308	4.95	2.92 (2.05–4.16)
≥30 kg/m²	34/79	18.20	11/70	4.84	5.18 (2.12–12.66)
SGLT-2i at baseline					
Yes	11/26	20.03	1/24	1.23	8.29 (1.03–66.73)
No	142/361	15.35	55/354	5.21	2.83 (2.05–3.92)
GLP-1RA at baseline					
Yes	11/25	17.95	4/16	8.84	2.61 (0.65–10.38)
No	142/362	15.46	52/362	4.76	3.16 (2.27–4.40)



*An unstratified model using Firth's penalised likelihood approach was applied due to zero cell counts and/or convergence issues
Only patients without the condition at baseline are included in this analysis. High albuminuria: UACR ≥30 mg/g but <300 mg/g. Normal albuminuria: UACR <30 mg/g. Albuminuria category changes were considered as shifts if they were accompanied by a UACR change of ≥30% from baseline to each visit. Values after onset of ESKD are not considered for this analysis
BMI, body mass index; CI, confidence interval; eGFR, estimated glomerular filtration rate; ESKD, end-stage kidney disease; GLP-1RA, glucagon-like peptide-1 receptor agonist; HbA1c, glycated haemoglobin; HR, hazard ratio; IR, incidence rate; PY, patient-years; SBP, systolic blood pressure; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UACR, urine albumin-to-creatinine ratio

Safety

- Overall, TEAEs were similar between the finerenone and placebo groups
  - The incidence of treatment-emergent serious adverse events was slightly lower in patients receiving finerenone compared with placebo (**Table 3**)
- The overall safety profile of finerenone was generally consistent across predefined eGFR subgroups (**Table 3**)

Table 3. TEAEs in the FIDELITY Asian subpopulation according to baseline eGFR (safety analysis set)

	eGFR <60 ml/min/1.73 m²		eGFR ≥60 ml/min/1.73 m²	
TEAE, n (%)	**Finerenone (n=911)**	**Placebo (n=921)**	**Finerenone (n=502)**	**Placebo (n=520)**
Any TEAE	853 (93.6)	860 (93.4)	462 (92.0)	494 (95.0)
Leading to discontinuation	62 (6.8)	56 (6.1)	22 (4.4)	26 (5.0)
Any serious AE	322 (35.3)	368 (40.0)	182 (36.3)	204 (39.2)
Leading to discontinuation	22 (2.4)	29 (3.1)	8 (1.6)	14 (2.7)
Death	16 (1.8)	18 (2.0)	3 (0.6)	7 (1.3)
Any treatment-emergent hyperkalaemia	211 (23.2)	135 (14.7)	74 (14.7)	50 (9.6)
Leading to permanent treatment discontinuation	16 (1.8)	5 (0.5)	5 (1.0)	4 (0.8)
Leading to hospitalisation	14 (1.5)	1 (0.1)	1 (0.2)	0
Any treatment-emergent acute kidney injury	23 (2.5)	33 (3.6)	7 (1.4)	14 (2.7)
Leading to permanent treatment discontinuation	0	2 (0.2)	0	1 (0.2)
Leading to hospitalisation	11 (1.2)	14 (1.5)	3 (0.6)	4 (0.8)

AE, adverse event; eGFR, estimated glomerular filtration rate; TEAE, treatment-emergent adverse event

- Hyperkalaemia
  - Hyperkalaemia was more common in patients with lower eGFR compared with higher eGFR at baseline in both the finerenone (23.2% versus 14.7%, respectively) and placebo (14.7% versus 9.6%, respectively) groups, although the rate was low overall
  - However, rates of hyperkalaemia leading to permanent treatment discontinuation (≤1.8%) or hospitalisation (≤1.5%) were low for both finerenone-treated and placebo-treated patients, regardless of baseline eGFR
- Acute kidney injury
  - Acute kidney injury was more common in patients with lower eGFR compared with higher eGFR at baseline in both the finerenone (2.5% versus 1.4%, respectively) and placebo (3.6% versus 2.7%, respectively) groups, although the rate was low overall
  - However, rates of acute kidney injury leading to permanent treatment discontinuation (≤0.2%) or hospitalisation (≤1.5%) were low for both finerenone-treated and placebo-treated patients, regardless of baseline eGFR

Conclusions

- This analysis of the FIDELITY dataset demonstrates the efficacy and safety of finerenone across the spectrum of Asian patients with CKD and T2D
  - Finerenone improved chronic eGFR slope irrespective of baseline UACR and a slower decline in eGFR was seen even in patients with UACR ≥300 mg/g
  - Finerenone also reduced UACR and resulted in higher rates of UACR regression compared with placebo, irrespective of baseline eGFR, UACR, SBP, HbA1c, SGLT-2i use and GLP-1RA use
  - Regression from 'high' to 'normal' albuminuria was observed in over one-third of patients, demonstrating meaningful kidney protection with finerenone
- The overall incidence of TEAEs was similar between treatment arms in the Asian patients regardless of baseline eGFR levels
  - Incidence of treatment-emergent hyperkalaemia was higher in patients treated with finerenone, but rates of hyperkalaemia that led to hospitalisation and permanent treatment discontinuation were low in both treatment arms
- Overall, this FIDELITY analysis shows that finerenone reduced the risk of kidney disease progression and reduces albuminuria levels versus placebo, and demonstrated a clinically manageable safety profile in Asian patients across key baseline subgroups

References: 1. Wu B, *et al. BMJ Open Diabetes Res Care* 2016;4:e000154. 2. Agarwal R, *et al. Eur Heart J* 2022;43:474–484. 3. Yang JJ, *et al. JAMA Netw Open* 2019;2:e192696. 4. Deng Y, *et al. Front Endocrinol (Lausanne)* 2021;12:672350. 5. Kidney Disease: Improving Global Outcomes CKD Work Group. *Kidney Int* 2024;105:S117–S314. 6. Carrero JJ, *et al. Kidney Int* 2017;91:244–251. 7. de Zeeuw D, *et al. Kidney Int* 2006;69:1675–1682. 8. Matsushita K, *et al. Lancet Diabetes Endocrinol* 2015;3:514–525. 9. Grams ME, *et al. JAMA* 2023;330:1266–1277. 10. Koya D, *et al. Am J Nephrol* 2023;54:370–378. 11. Wada T, *et al. Kidney Diseases* 2025 (in press). 12. Ruilope LM, *et al. Am J Nephrol* 2019;50:345–356. 13. Bakris GL, *et al. Am J Nephrol* 2019;50:333–344. 14. Pitt B, *et al. N Engl J Med* 2021;385:2252–2263.

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