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Effect of Finerenone on Albuminuria in Type 1 Diabetes by Baseline HbA_{1c} Level and Diabetes Duration: An Exploratory Analysis of the FINE-ONE Trial

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Finerenone Reduced Albuminuria Over 6 Months and Was Well Tolerated in Adults With Type 1 Diabetes (T1D) and Chronic Kidney Disease Regardless of Baseline HbA_{1c} Levels or Diabetes Duration

Methods

Prespecified analysis of FINE-ONE

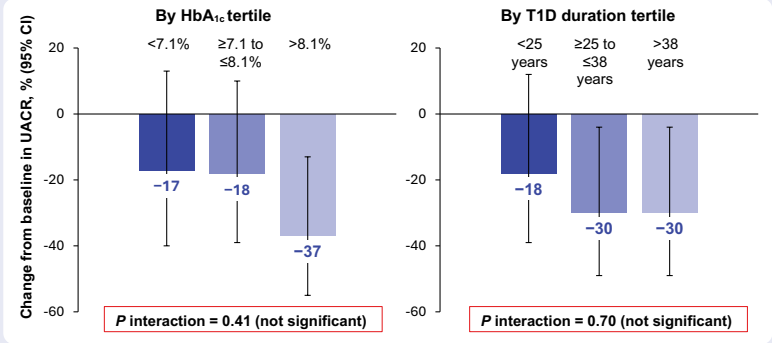
R
- Finerenone (10 or 20 mg once daily) *n* = 120*
- Placebo *n* = 120*

We analyzed **UACR change from baseline** over 6 months by baseline **HbA_{1c} and T1D duration tertile**

	HbA_{1c}, %	T1D duration, years
Tertile 1	<7.1	<25
Tertile 2	≥7.1 to ≤8.1	≥25 to ≤38
Tertile 3	>8.1	>38

Finerenone reduced urinary albumin-to-creatinine ratio (UACR) more than placebo across all HbA_{1c} and T1D duration tertiles

Difference between finerenone and placebo:



Incidence of adverse events and serious adverse events, including hyperkalemia, were **similar across HbA_{1c} tertiles**

*120 of 120 patients randomly assigned to finerenone and 120 of 122 patients randomly assigned to placebo in FINE-ONE had HbA_{1c} measurements at baseline and were included in this analysis.

ARTICLE HIGHLIGHTS

- **Why did we undertake this study?**
  We conducted this analysis to determine whether the efficacy and safety of finerenone differed according to baseline hemoglobin A_{1c} (HbA_{1c}) level, a proxy of glycemic control, or diabetes duration in adults with type 1 diabetes and chronic kidney disease.
- **What is the specific question(s) we wanted to answer?**
  Do the albuminuria-lowering effect and safety profile of finerenone vary across baseline HbA_{1c} and diabetes duration tertiles?
- **What did we find?**
  Finerenone reduced urinary albumin-to-creatinine ratio compared with placebo and was well tolerated irrespective of HbA_{1c} level or diabetes duration.
- **What are the implications of our findings?**
  These results suggest that finerenone provides kidney-protective benefits in type 1 diabetes regardless of glycemic control or diabetes duration, supporting its potential role across a broad patient population.

Effect of Finerenone on Albuminuria in Type 1 Diabetes by Baseline HbA_{1c} Level and Diabetes Duration: An Exploratory Analysis of the FINE-ONE Trial

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OBJECTIVE

To evaluate whether the efficacy and safety of finerenone varied by baseline hemoglobin A_{1c} (HbA_{1c}) level, a proxy of glycemic control, and diabetes duration in people with type 1 diabetes and chronic kidney disease (CKD).

RESEARCH DESIGN AND METHODS

Adults with type 1 diabetes, urinary albumin-to-creatinine ratio (UACR) 200 to <5,000 mg/g, and estimated glomerular filtration rate (eGFR) 25 to <90 mL/min/1.73 m^2 were randomly assigned (one to one) to finerenone or placebo. UACR change from baseline over 6 months by baseline HbA_{1c} and diabetes duration was analyzed.

RESULTS

Baseline HbA_{1c} was available for 240 of 242 participants; mean (SD) HbA_{1c}, diabetes duration, and eGFR were 7.6% (1.1%; 60 [12] mmol/mol), 32.0 (14.2) years, and 58.9 (19.2) mL/min/1.73 m^2, respectively. At 6 months, HbA_{1c} (95% CI) remained unchanged (finerenone +0.03% [−0.14%, 0.20%]; placebo 0.00% [−0.12%, 0.11%]; between-group difference +0.04% [−0.17%, 0.24%]; *P* = 0.74). Over 6 months, median UACR decreased from 574.6 to 373.5 mg/g with finerenone and from 506.4 to 475.6 mg/g with placebo, corresponding to a −25% placebo-corrected change (95% CI −35%, −13%; *P* = 0.0001). Treatment effects were consistent across HbA_{1c} tertiles (<7.1%, ≥7.1% to ≤8.1%, and >8.1%), with placebo-corrected UACR changes (95% CIs) of −17% (−40%, 13%), −18% (−39%, 10%), and −37% (−55%, −13%), respectively (*P* interaction = 0.41). Effects were similarly consistent across diabetes duration tertiles (*P* interaction = 0.70). Overall safety and incidence of hyperkalemia were similar across HbA_{1c} tertiles.

CONCLUSIONS

In adults with type 1 diabetes and CKD, finerenone reduced UACR and was well tolerated irrespective of HbA_{1c} level or diabetes duration.

Type 1 diabetes affects an estimated 9–10 million individuals worldwide, with >500,000 new cases diagnosed annually and global incidence increasing by up to 4% per year between 2019 and 2025 (1). Chronic kidney disease (CKD) develops in up to 40% of

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ORIGINAL ARTICLE

people with type 1 diabetes and is associated with increased risks of kidney failure, cardiovascular disease, and mortality (2–5).

Despite the high kidney and cardiovascular risks in type 1 diabetes, few therapies beyond renin-angiotensin system blockade have been evaluated in large outcome trials (6–8). In contrast, several agents, including the nonsteroidal mineralocorticoid receptor antagonist finerenone, have demonstrated kidney and cardiovascular protections in people with type 2 diabetes and CKD (7–10). Subsequent analyses showed that with finerenone, these benefits were largely mediated by reductions in urinary albumin-to-creatinine ratio (UACR), establishing albuminuria as a key pathway linking treatment responses to clinical outcomes (11). Because albuminuria plays a similar role in the pathophysiology and progression of CKD in types 1 and 2 diabetes, UACR provides a bridging biomarker to translate kidney-protective effects across people living with diabetes (8,11–14). Therefore, a short-term reduction in UACR with finerenone is expected to translate into long-term benefits in clinical kidney end points in patients with type 1 diabetes. FINE-ONE (Finerenone Efficacy and Safety in Chronic Kidney Disease and Type One Diabetes) was the first successful phase 3 clinical trial in >30 years in people with type 1 diabetes and CKD and applied this bridging biomarker concept, demonstrating that finerenone significantly reduced UACR over 6 months compared with placebo (11,15).

Poor glycemic control and longer diabetes duration are independently associated with higher albuminuria levels, faster decline in kidney function, and increased cardiovascular risk in people with type 1 diabetes. Large registry and cohort studies, as well as clinical trials, have demonstrated graded relationships between hemoglobin A_{1c} (HbA_{1c}) and mortality and between cumulative glycemic exposure and development and progression of kidney disease, with higher HbA_{1c} levels associated with more rapid progression of CKD (16–19). In addition, histopathological studies have shown that higher HbA_{1c} levels are associated with structural kidney changes, including glomerular and tubular basement membrane thickening and mesangial expansion (20). Together, these findings suggest that individuals with elevated HbA_{1c} or long-standing diabetes may have more advanced underlying kidney disease. Furthermore, studies in diabetes indicate that longer disease duration and more advanced disease may be associated with attenuated treatment responses, suggesting that cumulative disease burden could influence responsiveness to therapy (21). Determining whether the kidney-protective effects of finerenone are consistent across the spectrum of glycemic control and diabetes duration is therefore clinically important.

Here, we report a prespecified post hoc exploratory analysis of the FINE-ONE trial evaluating the effect of finerenone on UACR across tertiles of baseline HbA_{1c} and diabetes duration to assess the consistency of treatment effects in clinically relevant subgroups of people with type 1 diabetes and CKD.

RESEARCH DESIGN AND METHODS

Study Design

The FINE-ONE trial was a randomized, prospective, double-blind, global, multicenter phase 3 study evaluating once-daily finerenone versus placebo in individuals with type 1 diabetes and CKD. The trial design and primary results have been published previously (11,15). The study protocol was approved by an independent review board or independent ethics committee at each participating site, and all participants provided written informed consent. An independent data and safety monitoring committee was established to oversee participant safety and overall study conduct. FINE-ONE was conducted in accordance with the principles of the Declaration of Helsinki and the International Council for Harmonisation Guidelines for Good Clinical Practice. The trial is registered on ClinicalTrials.gov (NCT05901831).

Participants

Eligible individuals were aged ≥18 years with a diagnosis of type 1 diabetes, defined as continuous insulin treatment initiated within 1 year of diagnosis or, for individuals with onset after the age of 35 years, documented history of type 1 diabetes autoantibodies, hospitalization for ketoacidosis, or undetectable C-peptide. Participants were required to have been receiving a stable dose of an ACE inhibitor or angiotensin receptor blocker for at least 4 weeks before screening (maximally tolerated doses were not required) and have an HbA_{1c} level <10.0% (<86 mmol/mol), an estimated glomerular filtration rate (eGFR) of ≥25 to <90 mL/min/1.73 m^2, and a UACR of ≥200 to <5,000 mg/g (≥22.6 to <565 mg/mmol). Participants were excluded if they had a diagnosis of type 2 diabetes or a serum potassium level >4.8 mmol/L

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*A complete list of the FINE-ONE investigators can be found in the supplementary material online.

or had used a glucagon-like peptide 1 receptor agonist or sodium–glucose cotransporter 1/2 inhibitor within 8 weeks before screening.

Random Assignment

Participants were randomly assigned one to one to finerenone or matching placebo using an interactive response technology. On day 1, finerenone (or matching placebo) was administered orally in addition to standard care (either an ACE inhibitor or angiotensin receptor blocker). The starting dose of finerenone was based on baseline eGFR; participants with an eGFR ≥60 mL/min/1.73 m^2 received 20 mg, whereas those with an eGFR ≥25 to <60 mL/min/1.73 m^2 received 10 mg. Dose uptitration to 20 mg (the target dose) was permitted after 30 days if serum potassium was ≤4.8 mmol/L and the decrease in eGFR was <30% compared with the rate in the previous visit. Corresponding sham uptitrations were performed in the placebo group.

Study Visits and Assessments

After random assignment, participants attended scheduled study visits at months 1, 3, and 6, with a follow-up visit at month 7 (~30 days after treatment discontinuation). At each visit, blood and urine samples were collected for assessment of UACR, eGFR, and serum potassium, except at month 1, when UACR was not collected. UACR was measured using three first-morning void urine samples collected on the 2 days preceding the visit and on the morning of the visit, with analysis performed by a central laboratory. Serum potassium was assessed at every scheduled visit using both central and local measurements. HbA_{1c} was measured in a protocolized manner at baseline and months 3 and 6, with all samples analyzed centrally. Diabetes duration was determined from the documented date of diagnosis in the medical record and recorded at study entry.

Study Outcomes

The full list of study outcomes in the FINE-ONE trial has been reported previously (15). The primary efficacy outcome was the relative change in UACR over a period of 6 months, and secondary safety outcomes included the number of patients with adverse events, serious adverse events that started or worsened after the first dose of study medication and up to 3 days after any temporary or permanent interruption of study intervention, and adverse events leading to treatment discontinuation. Hyperkalemia and hypoglycemia were captured based on investigator-reported adverse events using Medical Dictionary for Regulatory Activities preferred terms corresponding to hyperkalemia, blood potassium increase, or hypoglycemia, respectively. Hyperkalemia events could be informed by central or local potassium measurements, using serum or plasma samples depending on country-specific laboratory practice. In this prespecified analysis, we evaluated the impact of baseline HbA_{1c} and diabetes duration on the relative change in UACR over a period of 6 months. We also report the incidence of adverse events up to month 6 by baseline HbA_{1c} and evaluated change in HbA_{1c} from baseline to months 3 and 6.

Statistical Analyses

The primary efficacy analysis evaluated the geometric mean ratio for the change from baseline in log-transformed UACR over the 6-month study period. This analysis was conducted in the full-analysis population, which included all participants who underwent random assignment. Treatment effect was analyzed with the use of a mixed model for repeated measures, which included treatment group, visit, treatment-by-visit interaction, and log-transformed baseline UACR as covariates and log-transformed baseline-by-visit interaction to characterize baseline-specific results over time (full details have been reported previously) (11).

Prespecified exploratory analyses evaluated treatment effects on UACR across tertiles of baseline HbA_{1c} (<7.1% [<54 mmol/mol], ≥7.1% to ≤8.1% [≥54 to ≤65 mmol/mol], and >8.1% [>65 mmol/mol]) and type 1 diabetes duration (<25, ≥25 to ≤38, and >38 years). These analyses were conducted in all patients with a baseline HbA_{1c} measurement. These subgroup analyses used an extension of the primary mixed model for repeated measures that additionally included fixed effects for subgroup and an interaction term, which included visit, treatment, and subgroup. Additional exploratory analyses evaluated treatment effects across the continuous ranges of baseline HbA_{1c} and diabetes duration using models incorporating restricted cubic splines and their interactions with treatment and visit. No adjustments were made for multiple comparisons, given the exploratory nature of these analyses. A two-sided P value <0.05 was considered to indicate statistical significance.

Safety analyses were performed in the safety analysis set, defined as all randomly assigned participants who received at least one dose of study medication. Adverse events, serious adverse events, and hyperkalemia were summarized descriptively by treatment group and baseline HbA_{1c} tertile. Data analyses were performed using R software.

Data and Resource Availability

The availability of the data underlying this publication will be determined according to Bayer's commitment to the European Federation of Pharmaceutical Industries and Associations/Pharmaceutical Research and Manufacturers of America principles for responsible clinical trial data sharing. This pertains to scope, time point and process of data access. As such, Bayer commits to sharing, on request from qualified scientific and medical researchers, patient-level clinical trial data, study-level clinical trial data, and protocols from clinical trials in patients for medicines and indications approved in the U.S. and European Union (EU) as necessary for conducting legitimate research. This applies to data on new medicines and indications that have been approved by the U.S. and EU regulatory agencies on or after 1 January 2014. Interested researchers can use www.vivli.org to request access to anonymized patient-level data and supporting documents from clinical studies to conduct further research that can help advance medical science or improve patient care. Information on the Bayer criteria for listing studies and other relevant information is provided in the member section of the portal. Data access will be granted to anonymized patient-level data, protocols, and clinical study reports after approval by an independent scientific review panel. Bayer is not involved in the decisions made by the independent review panel. Bayer will take all necessary measures to ensure that patient privacy is safeguarded.

RESULTS

Baseline Characteristics

Baseline HbA_{1c} and diabetes duration were available for 240 of 242 participants enrolled in FINE-ONE (Table 1). Overall, the

Table 1—Baseline characteristics by baseline HbA_{1c} tertile

	Mean (SD)*						
	Tertile 1†		Tertile 2†		Tertile 3†		
	Finerenone (*n* = 31)	Placebo (*n* = 45)	Finerenone (*n* = 43)	Placebo (*n* = 41)	Finerenone (*n* = 46)	Placebo (*n* = 34)	Total (*N* = 240)
Age, years	51.5 (13.9)	51.3 (13.3)	53.7 (16.0)	50.8 (13.9)	48.8 (12.3)	53.2 (12.2)	51.5 (13.6)
Female sex, *n* (%)	10 (32.3)	15 (33.3)	15 (34.9)	12 (29.3)	16 (34.8)	16 (47.1)	84 (35.0)
Race, *n* (%)							
White	21 (67.7)	32 (71.1)	32 (74.4)	32 (78.0)	32 (69.6)	24 (70.6)	173 (72.1)
Asian	7 (22.6)	12 (26.7)	8 (18.6)	8 (19.5)	8 (17.4)	5 (14.7)	48 (20.0)
Black or African American	1 (3.2)	1 (2.2)	2 (4.7)	0	6 (13.0)	5 (14.7)	15 (6.2)
Other‡	2 (6.5)	0	1 (2.3)	1 (2.4)	0	0	4 (1.7)
Ethnicity, *n* (%)							
Hispanic or Latino	3 (9.7)	3 (6.7)	4 (9.3)	4 (9.8)	4 (8.7)	2 (5.9)	20 (8.3)
Diabetes duration, years	33.3 (14.7)	32.4 (15.6)	36.7 (14.5)	31.8 (14.7)	26.7 (11.6)	31.4 (12.9)	32.0 (14.2)
HbA_{1c}							
%	6.5 (0.5)	6.5 (0.5)	7.5 (0.3)	7.5 (0.3)	8.9 (0.5)	8.8 (0.4)	7.6 (1.1)
mmol/mol	48 (5.5)	48 (5.5)	58 (3.3)	58 (3.3)	74 (5.5)	73 (4.4)	60 (12.0)
Range, min–max							
%	5.2–7.0	4.8–7.0	7.1–8.1	7.1–8.1	8.2–10.0	8.2–9.7	4.8–10.0
mmol/mol	33–53	29–53	54–65	54–65	66–86	66–83	29–86
Body weight, kg§	78.2 (15.9)	76.0 (19.9)	80.6 (20.8)	86.3 (23.5)	83.3 (18.5)	77.1 (23.2)	80.4 (20.6)
BMI, kg/m^2§	27.2 (5.2)	26.3 (5.9)	28.0 (5.9)	28.5 (5.9)	27.8 (5.1)	27.1 (8.0)	27.5 (6.0)
eGFR, mL/min/1.73 m^2‖	56.2 (17.8)	56.7 (19.6)	59.0 (20.5)	59.2 (16.6)	60.9 (19.7)	60.9 (21.4)	58.9 (19.2)
eGFR distribution, *n* (%), mL/min/1.73 m^2							
<45	10 (32.3)	14 (31.1)	11 (25.6)	9 (22.0)	11 (23.9)	8 (23.5)	63 (26.2)
45 to <60	9 (29.0)	10 (22.2)	12 (27.9)	10 (24.4)	11 (23.9)	9 (26.5)	61 (25.4)
≥60	12 (38.7)	21 (46.7)	20 (46.5)	22 (53.7)	24 (52.2)	17 (50.0)	116 (48.3)
UACR, median (IQR), mg/g	622 (282–1,066)	500 (248–1,401)	566 (400–924)	484 (325–1,079)	604 (307–1,301)	570 (320–913)	549 (298–1,217)
UACR distribution, *n* (%), mg/g							
<300	9 (29.0)	17 (37.8)	8 (18.6)	10 (24.4)	12 (26.1)	7 (20.6)	63 (26.2)
300–1,000	14 (45.2)	14 (31.1)	25 (58.1)	20 (48.8)	18 (39.1)	19 (55.9)	110 (45.8)
>1,000	8 (25.8)	14 (31.1)	10 (23.3)	11 (26.8)	16 (34.8)	8 (23.5)	67 (27.9)
Systolic blood pressure, mmHg	134.7 (17.6)	133.6 (19.2)	137.3 (15.9)	137.1 (15.3)	137.0 (14.6)	131.4 (18.9)	135.3 (16.9)
Serum potassium, mmol/L	4.7 (0.4)	4.5 (0.5)	4.6 (0.4)	4.6 (0.4)	4.6 (0.4)	4.6 (0.4)	4.6 (0.4)

IQR, interquartile range. *Data are mean (SD) unless otherwise indicated. †Tertile 1, <7.1% (<54 mmol/mol); tertile 2, ≥7.1% to ≤8.1% (≥54 and ≤65 mmol/mol); tertile 3, >8.1% (>65 mmol/mol). ‡Two participants in the finerenone group were Native American or Native Alaskan, and race or ethnic group was not reported for one participant in each group. §Data were missing for two participants across the HbA_{1c} subgroups: one in the finerenone group (tertile 2) and one in the placebo group (tertile 1). ‖Calculated with the use of the Chronic Kidney Disease Epidemiology Collaboration equation (25).

mean age was 51.5 years; 84 participants (35.0%) were female, and 173 (72.1%) were White. Mean (SD) baseline HbA_{1c}, diabetes duration, and eGFR were 7.6% (1.1%; 60 [12.0] mmol/mol), 32 (14.2) years, and 58.9 (19.2) mL/min/1.73 m^2, respectively. Median UACR was 549 mg/g overall. Baseline characteristics were generally similar across baseline HbA_{1c} and diabetes duration tertiles and between treatment groups. By HbA_{1c} tertile, median UACR values for finerenone and placebo were 622 and 500 mg/g in tertile 1, 566 and 484 mg/g in tertile 2, and 604 and 570 mg/g in tertile 3, respectively. By diabetes duration tertile, median UACR values for finerenone and placebo were 684 and 551 mg/g in tertile 1, 566 and 513 mg/g in tertile 2, and 511 and 443 mg/g in tertile 3, respectively (Supplementary Table 1).

HbA_{1c} Effect

Change from baseline in HbA_{1c} during the 6-month study period is shown in Fig. 1*A*. At month 6, HbA_{1c} remained stable in both treatment groups. The mean change from baseline was +0.03% (95% CI −0.14%, 0.20%) in the finerenone group and 0.00% (95% CI −0.12%, 0.11%) in the placebo group, corresponding to a between-group difference of +0.04% (95% CI −0.17%, 0.24%; $P = 0.74$).

Treatment Effects on UACR by Baseline HbA_{1c} and Diabetes Duration Tertile

Over the 6-month study period, median UACR decreased from 574.6 to 373.5 mg/g with finerenone and from 506.4 to 475.6 mg/g with placebo, corresponding to a placebo-corrected UACR change of

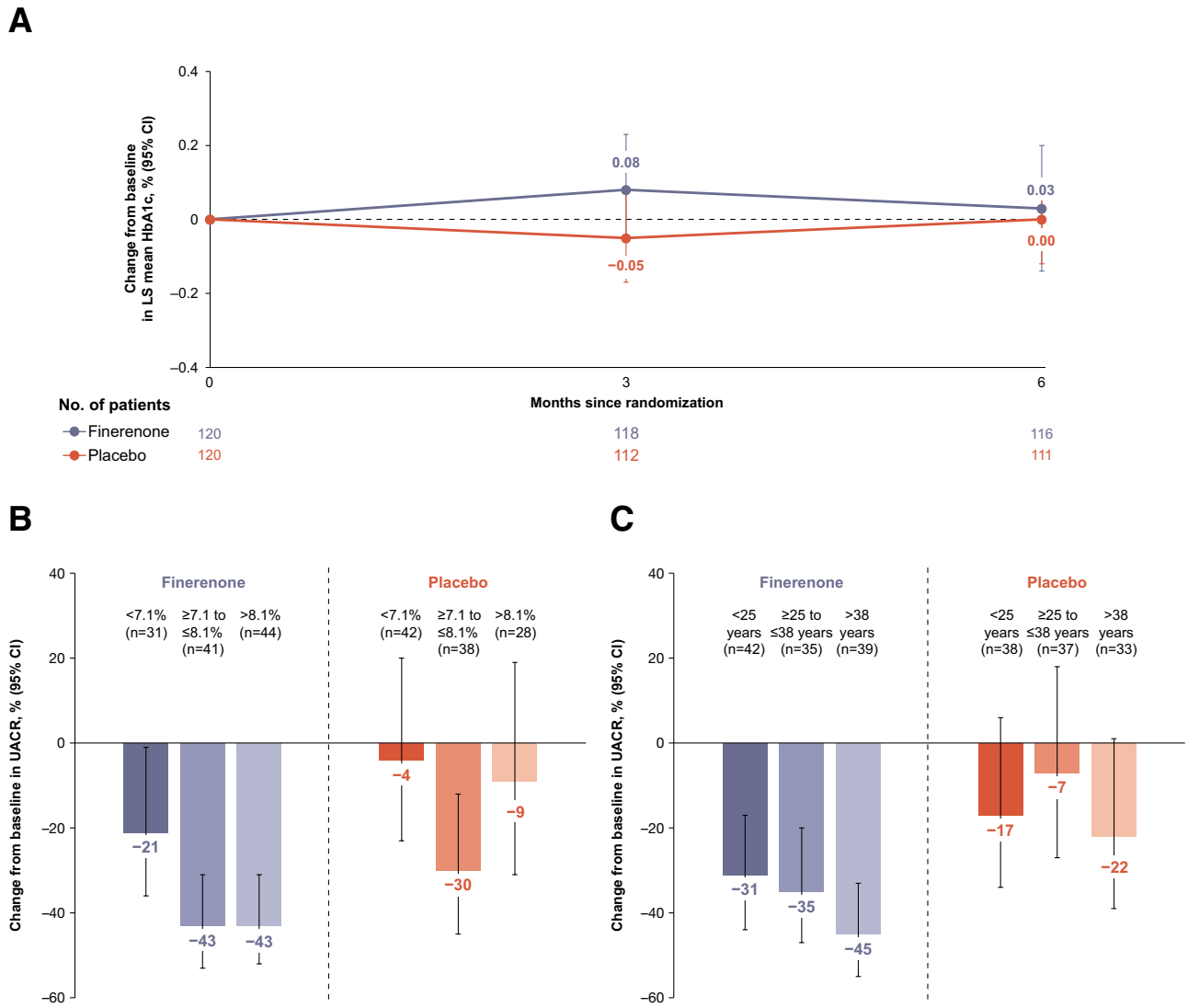

Figure 1—*A–C*: Overall change in HbA_{1c} (*A*) and UACR responses by baseline HbA_{1c} (*B*) and diabetes duration (*C*) tertiles over the 6-month study period. *A*: Least squares (LS) mean change from baseline in HbA_{1c} (%) over the 6-month study period for finerenone and placebo. Left *y*-axis represents HbA_{1c} as percentage, and right *y*-axis represents HbA_{1c} in mmol/mol. *B*: Percentage change from baseline to month 6 in UACR by baseline HbA_{1c} tertile (<7.1%, ≥7.1% to ≤8.1%, and >8.1%) for each treatment group. *C*: Percentage change from baseline to month 6 in UACR by diabetes duration tertile (<25, ≥25 to ≤38, and >38 years) for each treatment group. Error bars indicate 95% CIs. Log-transformed UACR ratios from baseline through month 6 were analyzed using mixed model for repeated measures including fixed effects for treatment group, visit, treatment-by-visit interaction, baseline log-transformed UACR, and baseline-by-visit interaction, with unstructured within-patient covariance matrix across visits. Model estimates were back-transformed and are presented as geometric mean ratio. Subgroup analyses were performed using extension of this model that additionally included fixed effects for subgroup and corresponding interaction terms.

−25% (95% CI −35%, −13%; *P* = 0.0001) (15). Finerenone reduced UACR from baseline across all HbA_{1c} tertiles. The placebo-corrected treatment effects (95% CI) were −17% (−40%, 13%; *P* = 0.230), −18% (−39%, 10%; *P* = 0.183), and −37% (−55%, −13%; *P* = 0.006) in tertiles 1, 2, and 3, respectively (Fig. 1*B* and Table 2). The treatment effect did not differ significantly across HbA_{1c} tertiles (*P* interaction = 0.41).

Finerenone also reduced UACR across all diabetes duration tertiles (<25, ≥25 to ≤38, and >38 years) over 6 months. The placebo-corrected treatment effects (95% CI) were −18% (−39%, 12%; *P* = 0.212), −30% (−49%, −4%; *P* = 0.029), and −30% (−49%, −4%; *P* = 0.029) in tertiles 1, 2, and 3, respectively (Fig. 1*C* and Supplementary Table 2). There was no significant interaction between treatment effects and diabetes duration tertile (*P* interaction = 0.70).

Finerenone reduced UACR across the full range of baseline HbA_{1c} and type 1 diabetes duration when modeled as continuous variables, with no significant interactions between treatment effect and baseline HbA_{1c} (*P* interaction = 0.27) or diabetes duration (*P* interaction = 0.27) (Fig. 2*A* and *B*).

Safety

Adverse events by HbA_{1c} tertile are shown in Supplementary Table 3. Across tertiles, the overall rates of adverse events and serious adverse events were similar between treatment groups. The frequency of treatment-emergent hyperkalemia was higher with finerenone than with placebo. Investigator-reported hyperkalemia occurred in two (6.7%), five (11.6%), and five (10.9%) participants receiving finerenone in tertiles

Table 2—Treatment effect by HbA_{1c} tertile: percentage change of UACR over 6-month study period versus baseline

	% (95% CI)			
	Finerenone	Placebo	Difference between finerenone and placebo	*P*
Tertile 1 (*n* = 73)*	−21 (−36, −1)	−4 (−23, 20)	−17 (−40, 13)	0.230
Tertile 2 (*n* = 79)*	−43 (−53, −31)	−30 (−45, −12)	−18 (−39, 10)	0.183
Tertile 3 (*n* = 72)*	−43 (−52, −31)	−9 (−31, 19)	−37 (−55, −13)	0.006
Overall (*n* = 224)	−34 (−40, −27)	−12 (−21, −2)	−25 (−35, −13)	0.0001†

*Tertile 1, <7.1% (<54 mmol/mol); tertile 2, ≥7.1% to ≤8.1% (≥54 and ≤65 mmol/mol); tertile 3, >8.1% (>65 mmol/mol). †Derived from the primary FINE-ONE analyses (full analysis set, *N* = 242) that used multiple imputation (15).

1, 2, and 3, respectively, and in two (4.4%), two (4.9%), and zero participants receiving placebo. Treatment discontinuation because of hyperkalemia was infrequent, occurring in none of the 120 participants receiving placebo and in two of 119 participants receiving finerenone (1.7%), with the two events occurring in tertiles 2 and 3. Treatment-emergent diabetic ketoacidosis was infrequent across HbA_{1c} tertiles. Two events occurred in participants receiving finerenone, both in tertile 3, and three events occurred with placebo, with one event in each tertile. Treatment-emergent hypoglycemia was also infrequent, with two events in the finerenone group (one each in tertiles 2 and 3) and seven events in the placebo group (two, two, and three events in tertiles 1, 2, and 3, respectively).

CONCLUSIONS

This prespecified subgroup analysis of the FINE-ONE trial showed that the benefits of finerenone in adults with type 1 diabetes and CKD were consistent regardless of baseline HbA_{1c} level or diabetes duration. In line with the overall FINE-ONE results (15), finerenone reduced UACR more than placebo across all HbA_{1c} and diabetes duration tertiles, with consistent effects observed across the continuous ranges of baseline HbA_{1c} and diabetes duration.

Decades of observational research in type 1 diabetes have shown that poor glycemic control, longer diabetes duration, and higher levels of albuminuria are each strong predictors of progressive kidney disease and cardiovascular complications (16–18,22). Long-term cohort studies and clinical trials have consistently demonstrated that elevated HbA_{1c} is associated with more rapid progression of kidney disease, even after accounting for other risk factors (17–19). Similarly, duration of type 1 diabetes has emerged as a major determinant of both kidney and cardiovascular risks, with earlier onset linked to markedly higher lifetime rates of cardiovascular events and premature mortality (16,18). However, CKD may also develop in individuals with relatively good glycemic control, suggesting that nonglycemic mechanisms, such as hypertension and genetic predisposition, among others, may contribute substantially to kidney disease progression in some patients. Against this background, the consistent albuminuria-lowering effect of finerenone across varying levels of HbA_{1c} and disease duration suggests that key metabolic and historical disease factors do not materially modify short-term therapeutic response. Overall, these results support the potential use

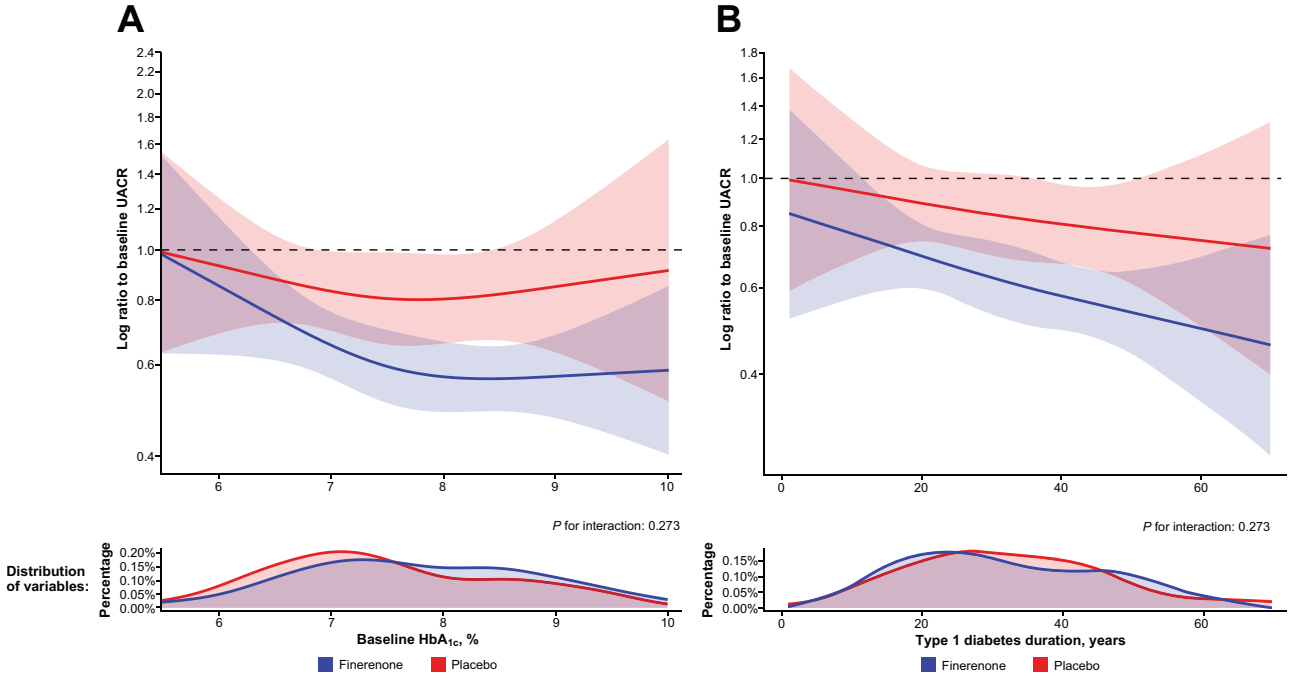

Figure 2—*A* and *B*: Effect of finerenone on UACR across continuous baseline HbA_{1c} (*A*) and type 1 diabetes duration (*B*). Associations are shown of baseline HbA_{1c} (*A*) and type 1 diabetes duration (*B*) with change from baseline in UACR over 6-month study period for finerenone and placebo. Log ratio from baseline UACR shown on *y*-axis, and baseline HbA_{1c} (*A*) and type 1 diabetes duration (*B*) shown on *x*-axis. Analyses were based on mixed model for repeated measures evaluating treatment effects across continuous range of baseline HbA_{1c} and diabetes duration using restricted cubic splines and their interactions with treatment and visit, adjusting for baseline log-transformed UACR and baseline-by-visit interaction, with unstructured within-patient covariance matrix across visits.

of finerenone across the broad clinical spectrum of adults with type 1 diabetes and CKD. Although tests for interaction were not statistically significant, numerically greater reductions in UACR were observed in participants with higher HbA_{1c} levels and longer diabetes duration. These observations should not be overinterpreted; however, they may suggest that individuals at higher risk derive at least comparable, if not greater, benefit.

The albuminuria-lowering effect of finerenone observed across HbA_{1c} and diabetes duration tertiles in FINE-ONE is consistent with findings from FIDELIO-DKD (Finerenone in Reducing Kidney Failure and Disease Progression in Diabetic Kidney Disease), a large phase 3 trial conducted in people with type 2 diabetes and CKD (9). A post hoc analysis of FIDELIO-DKD demonstrated similar reductions in UACR across HbA_{1c} subgroups, with no modification of treatment effect by baseline glycemic control (23). Additionally, pooled analyses of the FIDELIO-DKD and FIGARO-DKD (Finerenone in Reducing Cardiovascular Mortality and Morbidity in Diabetic Kidney Disease) trials showed that the effects of finerenone on cardiovascular outcomes and CKD progression were consistent across categories of baseline HbA_{1c} and diabetes duration (24). Together, these data suggest that the albuminuria-lowering and kidney and cardiovascular protective effects of finerenone are robust across varying metabolic profiles in CKD associated with diabetes. The present findings extend this observation from type 2 to type 1 diabetes.

Moreover, the incidence of adverse events and serious adverse events was similar between treatment groups across HbA_{1c} tertiles. Importantly, there was no excess occurrence of diabetic ketoacidosis in the finerenone group compared with the placebo group, and rates of hypoglycemia were not increased. Together, these findings support the glycemic and metabolic safety of finerenone in people with type 1 diabetes and CKD.

Although the subgroup analysis reported here was prespecified, the number of participants per subgroup was small. The study was not powered to detect differences or interaction effects across tertiles, and the lack of statistically significant interaction should be interpreted in the context of limited statistical power. Moreover, the relatively short 6-month treatment period constrained the ability to evaluate longer-term kidney outcomes; this is a limitation associated with the challenges of conducting a large, long-term trial in this population. However, in long-term studies in type 2 diabetes, effects of finerenone on UACR stabilized after ~4 months (9,10).

In conclusion, this prespecified analysis of the FINE-ONE trial demonstrated that finerenone reduced UACR compared with placebo, irrespective of baseline HbA_{1c} level or diabetes duration, and was well tolerated. These findings indicate that neither glycemic control, as reflected by HbA_{1c}, nor duration of type 1 diabetes materially modified the short-term response to finerenone. Together with prior evidence highlighting the strong prognostic importance of HbA_{1c}, diabetes duration, and albuminuria in this population, the present results support the potential applicability of finerenone across diverse clinical profiles of adults with type 1 diabetes and CKD.

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