KERENDIA- finerenone tablet, film coated
KERENDIA- finerenone tablet
Bayer HealthCare Pharmaceuticals Inc.

HIGHLIGHTS OF PRESCRIBING INFORMATION
These highlights do not include all the information needed to use KERENDIA safely and effectively. See full prescribing information for KERENDIA.

KERENDIA® (finerenone) tablets, for oral use
Initial U.S. Approval: 2021

RECENT MAJOR CHANGES

Indications and Usage (1)	9/2026
Dosage and Administration (2.3)	9/2026

INDICATIONS AND USAGE

Kerendia is a non-steroidal mineralocorticoid receptor antagonist (nsMRA) indicated to:

- reduce the risk of sustained estimated glomerular filtration rate (eGFR) decline, end-stage kidney disease, cardiovascular death, non-fatal myocardial infarction, and hospitalization for heart failure in adults with chronic kidney disease (CKD) associated with Type 2 diabetes mellitus (T2DM). (1)
- reduce urinary albumin-to-creatinine ratio (UACR), which is expected to reduce the risk of sustained eGFR decline and end-stage kidney disease in adults with CKD associated with Type 1 diabetes mellitus (T1DM). (1)
- reduce the risk of cardiovascular death, hospitalization for heart failure, and urgent heart failure visits in adults with heart failure with left ventricular ejection fraction (LVEF) ≥ 40%. (1)

DOSAGE AND ADMINISTRATION

- The recommended starting dosage is 10 mg or 20 mg orally once daily based on eGFR and serum potassium thresholds. (2.1, 2.2)
- Increase dosage after 4 weeks to the target dose of 20 mg once daily for CKD associated with T2DM or T1DM based on eGFR and serum potassium thresholds. (2.3)
- Increase dosage after 4 weeks to the target dose of 20 mg or 40 mg once daily for heart failure with LVEF ≥ 40% based on eGFR and serum potassium thresholds. (2.3)
- Tablets may be taken with or without food. (2.2)

DOSAGE FORMS AND STRENGTHS

Tablets: 10 mg, 20 mg, and 40 mg. (3)

CONTRAINDICATIONS

- Hypersensitivity to any component of this product. (4)
- Concomitant use with strong CYP3A4 inhibitors. (4, 7.1)
- Patients with adrenal insufficiency. (4)

WARNINGS AND PRECAUTIONS

- Hyperkalemia. Patients with decreased kidney function and higher baseline potassium levels are at increased risk. Monitor serum potassium levels and adjust dose as needed. (2.1, 2.2, 2.3, 5.1)
- Worsening of Renal Function in Patients with Heart Failure. Measure eGFR and adjust dose as needed. (2.1, 2.3, 6.1)

ADVERSE REACTIONS

Adverse reactions occurring in ≥ 1% of patients on Kerendia and more frequently than placebo are hyperkalemia, hypotension, and hyponatremia. (6.1)

To report SUSPECTED ADVERSE REACTIONS, contact Bayer HealthCare Pharmaceuticals Inc. at 1-888-842-2937 or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

DRUG INTERACTIONS

- Strong CYP3A4 Inhibitors: Use is contraindicated. (7.1)

- Grapefruit or grapefruit juice: Avoid concomitant use. (7.1)
- Moderate or weak CYP3A4 Inhibitors: Monitor serum potassium during drug initiation or dosage adjustment of either Kerendia or the moderate or weak CYP3A4 inhibitor, and adjust Kerendia dosage as appropriate. (7.1)
- Strong or moderate CYP3A4 Inducers: Avoid concomitant use. (7.1)
- Sensitive CYP2C8 substrates at Kerendia 40 mg: Monitor more frequently for adverse reactions. (7.2)

USE IN SPECIFIC POPULATIONS

Lactation: Breastfeeding not recommended. (8.2)

See 17 for PATIENT COUNSELING INFORMATION.

Revised: 9/2026

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FULL PRESCRIBING INFORMATION

1 INDICATIONS AND USAGE

Kerendia is indicated to:

- reduce the risk of sustained estimated glomerular filtration rate (eGFR) decline, end-stage kidney disease, cardiovascular death, non-fatal myocardial infarction, and hospitalization for heart failure in adults with chronic kidney disease (CKD) associated with Type 2 diabetes mellitus (T2DM) *[see Clinical Studies (14.1)]*.
- reduce urinary albumin-to-creatinine ratio (UACR), which is expected to reduce the risk of sustained eGFR decline and end-stage kidney disease in adults with CKD associated with Type 1 diabetes mellitus (T1DM) *[see Clinical Studies (14.2)]*.
- reduce the risk of cardiovascular death, hospitalization for heart failure, and urgent heart failure visits in adults with heart failure with left ventricular ejection fraction (LVEF) ≥ 40% *[see Clinical Studies (14.3)]*.

2 DOSAGE AND ADMINISTRATION

2.1 Prior to Initiation of Kerendia

Measure serum potassium levels and eGFR before initiation. Do not initiate treatment if serum potassium is > 5.0 mEq/L *[see Warnings and Precautions (5.1)].*

2.2 Recommended Starting Dosage

The recommended starting dose of Kerendia is based on eGFR and is presented in Table 1.

Table 1: Recommended Starting Dosage

eGFR (mL/min/1.73m^2)	Starting Dose
≥ 60	20 mg orally once daily
≥ 25 to < 60	10 mg orally once daily
< 25	Initiation is not recommended

For patients who are unable to swallow whole tablets, Kerendia may be crushed and mixed with water or soft foods such as applesauce immediately prior to use and administered orally *[see Clinical Pharmacology (12.3)]*.

2.3 Monitoring and Dosage Adjustment

CKD associated with T2DM or T1DM

The target daily dose of Kerendia is 20 mg orally.

Measure serum potassium 4 weeks after initiating treatment and adjust dose (see Table 2); if serum potassium levels are > 4.8 to 5.0 mEq/L, initiation of Kerendia treatment may be considered with additional serum potassium monitoring within the first 4 weeks based on clinical judgment and serum potassium levels *[see Warnings and Precautions (5.1)].* Measure serum potassium 4 weeks after a dose adjustment and periodically throughout treatment, and adjust the dose as needed (see Table 2) *[see Warnings and Precautions (5.1) and Drug Interactions (7.1)].*

Table 2: Dose Adjustment Based on Current Serum Potassium Concentration and Current Dose (CKD associated with T2DM or T1DM)

		Current Kerendia Dose	
		10 mg once daily	**20 mg once daily**
Current Serum Potassium (mEq/L)	**≤ 4.8**	Increase the dose to 20 mg once daily.*	Maintain 20 mg once daily.
	> 4.8 – 5.5	Maintain 10 mg once daily.	Maintain 20 mg once daily.
	> 5.5	Withhold Kerendia. Consider restarting at 10 mg once daily when serum potassium ≤ 5.0 mEq/L.	Withhold Kerendia. Restart at 10 mg once daily when serum potassium ≤ 5.0 mEq/L.

* If eGFR has decreased by more than 30% compared to previous measurement, maintain 10 mg dose.

Heart Failure with LVEF ≥ 40%

The target daily dose of Kerendia for heart failure (LVEF ≥ 40%) is dependent on renal function (eGFR) at initiation of Kerendia treatment (see Table 3).

- The target daily dose is 40 mg orally once daily if eGFR at initiation is ≥ 60 mL/min/1.73m^2.
- The target daily dose is 20 mg orally once daily if eGFR at initiation is ≥ 25 to < 60 mL/min/1.73m^2.

Measure serum potassium and eGFR 4 weeks after initiating treatment and adjust dose (see Table 3). Measure serum potassium and eGFR 4 weeks after a dose adjustment and monitor periodically throughout treatment, and adjust the dose as needed (see Table 3) *[see Warnings and Precautions (5.1 & 5.2) and Drug Interactions (7.1)].*

Table 3: Dose Adjustment Based on Current Serum Potassium Concentration, eGFR, and Current Dose (Heart Failure (LVEF ≥ 40%))

		Current Kerendia Dose		
		10 mg once daily	**20 mg once daily**	**40 mg once daily**
Current Serum Potassium (mEq/L)	**< 5.0**	Increase the dose to 20 mg once daily*	Maintain 20 mg once daily if eGFR < 60 mL/min/1.73m^2 at initiation. Otherwise increase the dose to 40 mg once daily*	Maintain 40 mg once daily.
	≥ 5.0 to < 5.5	Maintain current dose.		
	≥ 5.5 to < 6.0	Withhold Kerendia. Restart at 10 mg once daily when serum potassium < 5.5 mEq/L.	Decrease to 10 mg once daily.	Decrease to 20 mg once daily.
	≥ 6.0	Withhold Kerendia. Restart at 10 mg once daily when serum potassium < 5.5 mEq/L.†		

* If eGFR has decreased by more than 30% compared to previous measurement, maintain current dose.

† If repeated serum potassium measurements are ≥5.5 mEq/L, restart Kerendia at 10 mg once daily when serum potassium < 5.0 mEq/L.

2.4 Missed Doses

Direct a patient to take a missed dose as soon as possible after it is noticed, but only on the same day. If this is not possible, the patient should skip the dose and continue with the next dose as prescribed.

3 DOSAGE FORMS AND STRENGTHS

Kerendia is available as film-coated, oblong tablets in three strengths.

- 10 mg: pink, with "FI" on one side, "10" on the other side.
- 20 mg: yellow, with "FI" on one side, "20" on the other side.
- 40 mg: gray-orange, with "FI" on one side, "40" on the other side.

4 CONTRAINDICATIONS

Kerendia is contraindicated in patients:

- Who are hypersensitive to any component of this product *[see Adverse Reactions (6.2)]*.
- Who are receiving concomitant treatment with strong CYP3A4 inhibitors *[see Drug Interactions (7.1)].*
- With adrenal insufficiency.

5 WARNINGS AND PRECAUTIONS

5.1 Hyperkalemia

Kerendia can cause hyperkalemia *[see Adverse Reactions (6.1)]*.

The risk for developing hyperkalemia increases with decreasing kidney function and is greater in patients with higher baseline potassium levels or other risk factors for hyperkalemia. Measure serum potassium and eGFR in all patients before initiation of treatment with Kerendia and dose accordingly *[see Dosage and Administration (2.1)]*. Do not initiate Kerendia if serum potassium is > 5.0 mEq/L.

Measure serum potassium periodically during treatment with Kerendia and adjust dose accordingly *[see Dosage and Administration (2.3)]*. More frequent monitoring may be necessary for patients at risk for hyperkalemia, including those on concomitant medications that impair potassium excretion or increase serum potassium *[see Drug Interactions (7.1, 7.2)]*.

5.2 Worsening of Renal Function in Patients with Heart Failure

Kerendia can cause worsening of renal function in patients with heart failure. Rarely, severe events associated with worsening renal function, including events requiring hospitalization, have been observed *[see Adverse Reactions (6.1)]*.

Measure eGFR in all patients before initiation of treatment or with dose titration of Kerendia and dose accordingly *[see Dosage and Administration (2.1, 2.3)].* Initiation of Kerendia in patients with heart failure and an eGFR <25 mL/min/1.73m^2 is not recommended.

Measure eGFR periodically during maintenance treatment with Kerendia in patients with heart failure. Consider delaying up-titration or interrupting treatment with Kerendia in patients who develop clinically significant worsening of renal function.

6 ADVERSE REACTIONS

The following serious adverse reactions are discussed elsewhere in the labeling:

- Hyperkalemia *[see Warnings and Precautions (5.1)]*

6.1 Clinical Trials Experience

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice.

CKD associated with T2DM

The safety of Kerendia in patients with CKD associated with T2DM was evaluated in 2 randomized, double-blind, placebo-controlled, multicenter pivotal phase 3 studies, FIDELIO-DKD and FIGARO-DKD, in which a total of 6510 patients were treated with 10 or 20 mg once daily over a mean duration of 2.2 and 2.9 years, respectively.

Overall, serious adverse events occurred in 32% of patients receiving Kerendia and in 34% of patients receiving placebo in the FIDELIO-DKD study; the findings were similar in

the FIGARO-DKD study. Permanent discontinuations due to adverse events also occurred in a similar proportion of patients in the two studies (6-7% of patients receiving Kerendia and in 5-6% of patients receiving placebo).

The most frequently reported (≥ 10%) adverse reaction was hyperkalemia *[see Warnings and Precautions (5.1)].* Hospitalization due to hyperkalemia for the Kerendia group was 0.9% vs 0.2% in the placebo group across both studies. Hyperkalemia led to permanent discontinuation of treatment in 1.7% receiving Kerendia versus 0.6% of patients receiving placebo across both studies.

Table 4 shows adverse reactions that occurred more commonly on Kerendia than on placebo, and in at least 1% of patients treated with Kerendia.

Table 4: Adverse reactions reported in ≥ 1% of patients on Kerendia and more frequently than placebo (Pooled data from FIDELIO-DKD and FIGARO-DKD)

Adverse reactions	Kerendia N=6510 n (%)	Placebo N=6489 n (%)
Hyperkalemia	912 (14.0)	448 (6.9)
Hypotension	302 (4.6)	194 (3.0)
Hyponatremia	82 (1.3)	47 (0.7)

CKD associated with T1DM

The safety of Kerendia in patients with CKD associated with T1DM was evaluated in the randomized, double-blind, placebo-controlled, multicenter pivotal phase 3 study, FINE-ONE. In this study, 119 patients received Kerendia (10 mg or 20 mg) once daily over a mean duration of treatment of 5.9 months.

The overall safety profile of Kerendia in the FINE-ONE study was consistent with the adverse reactions reported in patients with CKD associated with T2DM (Table 4).

Heart Failure with LVEF ≥ 40%

The safety of Kerendia in patients with heart failure (LVEF ≥ 40%) was evaluated in the randomized, double-blind, placebo-controlled, multicenter pivotal phase 3 study, FINEARTS-HF, in which a total of 2,993 patients were treated with 10 mg, 20 mg, or 40 mg once daily of Kerendia with a mean duration of treatment of 2.3 years.

The overall safety profile of Kerendia in the FINEARTS-HF study was largely consistent with the adverse reactions reported in patients with CKD and T2DM (Table 4). However, adverse reactions related to worsening renal function were reported more frequently in the Kerendia group (18%) compared with placebo (12%) in FINEARTS-HF. The most frequently reported adverse reactions included renal impairment (7% vs. 4%), eGFR decreased (5% vs. 4%), acute kidney injury (4% vs. 2%) and renal failure (3% vs. 2%). The majority of events were reported to be mild to moderate. These events led to dose modifications in 9% of patients receiving Kerendia versus 4% of patients receiving placebo. Hospitalization due to events related to worsening of renal function for the Kerendia group was 2.0% versus 1.3% in the placebo group.

Laboratory Test

Initiation of Kerendia may cause an initial small increase in blood creatinine levels (mean change <0.1 mg/dL) and a small decrease in eGFR (mean change 2-3 mL/min) that occurs within the first 4 weeks of starting therapy and then stabilizes. These changes were reversible after treatment discontinuation. Initiation of Kerendia may also cause a small increase in serum uric acid. This increase appears to attenuate over time.

6.2 Postmarketing Experience

The following additional adverse reactions have been reported in postmarketing experience with finerenone. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to estimate their frequency reliably or to establish a causal relationship to drug exposure:

Hypersensitivity: Angioedema, Rash and Urticaria

7 DRUG INTERACTIONS

7.1 Effect of Other Drugs on Kerendia

Strong CYP3A4 Inhibitors

Kerendia is a CYP3A4 substrate. Concomitant use with a strong CYP3A4 inhibitor increases finerenone exposure *[see Clinical Pharmacology (12.3)]*, which may increase the risk of Kerendia adverse reactions. Concomitant use of Kerendia with strong CYP3A4 inhibitors is contraindicated *[see Contraindications (4)]*. Avoid concomitant intake of grapefruit or grapefruit juice.

Moderate and Weak CYP3A4 Inhibitors

Kerendia is a CYP3A4 substrate. Concomitant use with a moderate or weak CYP3A4 inhibitor increases finerenone exposure *[see Clinical Pharmacology (12.3)]*, which may increase the risk of Kerendia adverse reactions. Monitor serum potassium during drug initiation or dosage adjustment of either Kerendia or the moderate or weak CYP3A4 inhibitor, and adjust Kerendia dosage as appropriate *[see Dosage and Administration (2.3) and Drug Interactions (7.2)]*.

Strong and Moderate CYP3A4 Inducers

Kerendia is a CYP3A4 substrate. Concomitant use of Kerendia with a strong or moderate CYP3A4 inducer decreases finerenone exposure *[see Clinical Pharmacology (12.3)],* which may reduce the efficacy of Kerendia. Avoid concomitant use of Kerendia with strong or moderate CYP3A4 inducers.

7.2 Effect of Kerendia on Other Drugs

CYP2C8 Substrates

Kerendia is a weak CYP2C8 inhibitor at 40 mg. Kerendia increases exposure of CYP2C8 substrates at 40 mg dose *[see Clinical Pharmacology (12.3)]*, which may increase the risk of adverse reactions related to these substrates. Monitor patients more frequently for adverse reactions caused by sensitive CYP2C8 substrates if Kerendia 40 mg is co-administered with such substrates since minimal concentration changes may lead to serious adverse reactions.

7.3 Drugs That Affect Serum Potassium

More frequent serum potassium monitoring is warranted in patients receiving concomitant therapy with drugs or supplements that increase serum potassium *[see Dosage and Administration (2.3) and Warnings and Precautions (5.1)].*

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

There are no available data on Kerendia use in pregnancy to evaluate for a drug-associated risk of major birth defects, miscarriage, or adverse maternal or fetal outcomes. Animal studies have shown developmental toxicity at exposures about 2 times those expected in humans *(see Data)*. The clinical significance of these findings is unclear.

The estimated background risk of major birth defects and miscarriage for the indicated population is unknown. All pregnancies have a background risk of birth defect, loss or other adverse outcomes. In the U.S. general population, the estimated background risk of major birth defects and miscarriage in clinically recognized pregnancies is 2 to 4% and 15 to 20%, respectively.

Data

Animal Data

In the embryo-fetal toxicity study in rats, finerenone resulted in reduced placental weights and signs of fetal toxicity, including reduced fetal weights and retarded ossification at the maternal toxic dose of 10 mg/kg/day corresponding to an $AUC_{unbound}$ of at least 7 times that in humans. At 30 mg/kg/day, the incidence of visceral and skeletal variations was increased (slight edema, shortened umbilical cord, slightly enlarged fontanelle) and one fetus showed complex malformations including a rare malformation (double aortic arch) at an $AUC_{unbound}$ of about 10 times that in humans at the 40 mg dose and about 25 times that in humans at the 20 mg dose. The doses free of any findings (low dose in rats, high dose in rabbits) provide safety margins of 4 to 5 times for the $AUC_{unbound}$ expected in humans.

When rats were exposed during pregnancy and lactation in the pre- and postnatal developmental toxicity study, increased pup mortality and other adverse effects (lower pup weight, delayed pinna unfolding) were observed at about 2 or 4 times the $AUC_{unbound}$ expected in humans at the dose of 40 mg and 20 mg, respectively. In addition, the offspring showed slightly increased locomotor activity, but no other neurobehavioral changes starting at about 2 or 4 times the $AUC_{unbound}$ expected in humans at the dose of 40 mg and 20 mg, respectively. The dose free of findings provides a safety margin of about 2 times for the $AUC_{unbound}$ expected in humans for the 20 mg dose and is in the therapeutic range for the 40 mg dose.

8.2 Lactation

Risk Summary

There are no data on the presence of finerenone or its metabolite in human milk, the

effects on the breastfed infant or the effects of the drug on milk production. In a pre- and postnatal developmental toxicity study in rats, increased pup mortality and lower pup weight were observed at about 2 times the $AUC_{unbound}$ expected in humans. These findings suggest that finerenone is present in rat milk *[see Use in Specific Populations (8.1) and Data].* When a drug is present in animal milk, it is likely that the drug will be present in human milk. Because of the potential risk to breastfed infants from exposure to Kerendia, avoid breastfeeding during treatment and for 1 day after treatment.

8.4 Pediatric Use

The safety and efficacy of Kerendia have not been established in patients below 18 years of age.

8.5 Geriatric Use

Of the 6510 patients who received Kerendia in the FIDELIO-DKD and FIGARO-DKD studies, 55% were 65 years and older, and 14% were 75 years and older. Of the 2993 patients who received Kerendia in the FINEARTS-HF study, 79% were 65 years and older, and 43% were 75 years and older. Of the 119 patients who received Kerendia in the FINE-ONE study, 22% were 65 years and older, and 3% were 75 years and older. No overall differences in safety or efficacy were observed between these patients and younger patients. No dose adjustment is required.

8.6 Hepatic Impairment

Avoid use of Kerendia in patients with severe hepatic impairment (Child Pugh C).

No dosage adjustment is recommended in patients with mild or moderate hepatic impairment (Child Pugh A or B).

Consider additional serum potassium monitoring in patients with moderate hepatic impairment (Child Pugh B) *[see Dosage and Administration (2.3) and Clinical Pharmacology (12.3)].*

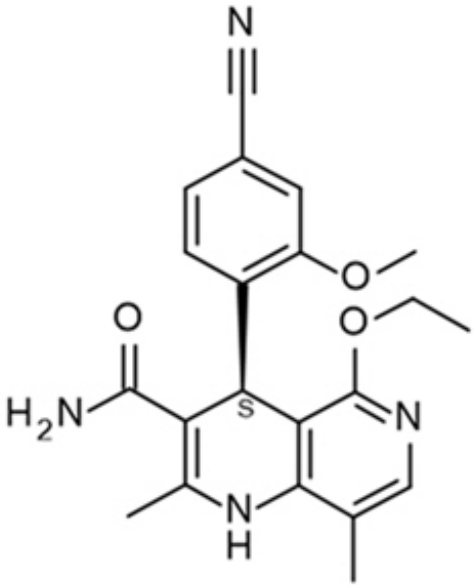
10 OVERDOSAGE

In the event of suspected overdose, immediately interrupt Kerendia treatment. The most likely manifestation of overdose is hyperkalemia. If hyperkalemia develops, standard treatment should be initiated.

Finerenone is unlikely to be efficiently removed by hemodialysis given its fraction bound to plasma proteins of about 90%.

11 DESCRIPTION

Kerendia contains finerenone, a nonsteroidal mineralocorticoid receptor antagonist. Finerenone's chemical name is (*4S*)-4-(4-cyano-2-methoxyphenyl)-5-ethoxy-2,8-dimethyl-1,4-dihydro-1,6-naphthyridine-3-carboxamide. The molecular formula is $C_{21}H_{22}N_4O_3$ and the molecular weight is 378.43 g/mol. The structural formula is:



Finerenone is a white to yellow crystalline powder. It is practically insoluble in water; and sparingly soluble in 0.1 M HCl, ethanol, and acetone.

Each Kerendia tablet contains 10 mg, 20 mg, or 40 mg of finerenone. The inactive ingredients of Kerendia are lactose monohydrate, cellulose microcrystalline, croscarmellose sodium, hypromellose, magnesium stearate, and sodium lauryl sulfate. The film coating contains hypromellose, titanium dioxide and talc, in addition to ferric oxide red (10 mg and 40 mg strength tablets) or ferric oxide yellow (20 mg and 40 mg strength tablets).

12 CLINICAL PHARMACOLOGY

12.1 Mechanism of Action

Finerenone is a nonsteroidal, selective antagonist of the mineralocorticoid receptor (MR), which is activated by aldosterone and cortisol and regulates gene transcription. Finerenone blocks MR mediated sodium reabsorption and MR overactivation in both epithelial (e.g., kidney) and nonepithelial (e.g., heart, and blood vessels) tissues. MR overactivation is thought to contribute to fibrosis and inflammation. Finerenone has a high potency and selectivity for the MR and has no relevant affinity for androgen, progesterone, estrogen, and glucocorticoid receptors.

12.2 Pharmacodynamics

In ARTS DN, a randomized, double-blind, placebo-controlled, multicenter phase IIb dose finding study in adults with CKD and T2DM, the placebo-corrected relative reduction in UACR at Day 90 was 25% and 38% in patients treated with finerenone 10 mg and 20 mg once daily, respectively. In patients treated with Kerendia, the mean systolic blood pressure decreased by 3 mmHg and the mean diastolic blood pressure decreased by 1-2 mmHg at month 1, remaining stable thereafter.

In FIDELIO-DKD and FIGARO-DKD, randomized, double-blind, placebo-controlled, multicenter studies in adult patients with CKD associated with T2DM, the placebo-corrected relative reduction in UACR in patients randomized to finerenone was 31% (95% CI 29-34%), and 32% (95% CI 30-35%), respectively, at Month 4 and remained

stable for the duration of the trial.

Cardiac Electrophysiology

At a dose 2 times the maximum approved recommended dose, finerenone does not prolong the QT interval to any clinically relevant extent.

12.3 Pharmacokinetics

Finerenone exposure increased proportionally over a dose range of 1.25 to 80 mg (0.06 to 4 times the maximum approved recommended dosage). Steady state of finerenone was achieved after 2 days of dosing. The estimated steady-state geometric mean $C_{max,md}$ was 166 µg/L and steady-state geometric mean $AUC_{\tau,md}$ was 718 µg.h/L following administration of finerenone 20 mg to patients.

Absorption

Finerenone is completely absorbed after oral administration but undergoes metabolism resulting in absolute bioavailability of 44%. Finerenone C_{max} was achieved between 0.5 and 1.25 hours after dosing.

Effect of Food

There was no clinically significant effect on finerenone AUC following administration with high fat, high calorie food.

Distribution

The volume of distribution at steady-state (Vss) of finerenone is 52.6 L. Plasma protein binding of finerenone is 92%, primarily to serum albumin, in vitro.

Elimination

The terminal half-life of finerenone is about 2 to 3 hours, and the systemic blood clearance is about 25 L/h.

Metabolism

Finerenone is primarily metabolized by CYP3A4 (90%) and to a lesser extent by CYP2C8 (10%) to inactive metabolites.

Excretion

About 80% of the administered dose is excreted in urine (<1% as unchanged) and approximately 20% in feces (< 0.2% as unchanged).

Specific Populations

There are no clinically significant effects of age (18 to 86 years), sex, race/ethnicity (White, Asian, Black, and Hispanic), or weight (54 to 126 kg) on the pharmacokinetics of finerenone.

Renal Impairment

There were no clinically relevant differences in finerenone AUC or C_{max} values in patients with eGFR 15 to < 90 mL/min/1.73m^2 compared to eGFR ≥ 90 mL/min/1.73m^2. For dosing recommendations based on eGFR and serum potassium levels *[see Dosage and Administration (2)]*.

Hepatic Impairment

There was no clinically significant effect on finerenone exposure in cirrhotic patients with mild hepatic impairment (Child Pugh A).

Finerenone mean AUC was increased by 38% and C_{max} was unchanged in cirrhotic patients with moderate hepatic impairment (Child Pugh B) compared to healthy control subjects.

The effect of severe hepatic impairment (Child Pugh C) on finerenone exposure was not studied.

Drug Interaction Studies

Clinical Studies and Model-Informed Approaches

Strong CYP3A Inhibitors: Concomitant use of itraconazole (strong CYP3A4 inhibitor) was predicted to increase finerenone AUC by > 400%.

Moderate CYP3A Inhibitors: Concomitant use of erythromycin (moderate CYP3A4 inhibitor) increased finerenone mean AUC and C_{max} by 248% and 88%, respectively. Concomitant use of verapamil (moderate CYP3A4 inhibitor) increased finerenone mean AUC and C_{max} by 170% and 122%, respectively.

Weak CYP3A Inhibitors: Concomitant use of amiodarone (weak CYP3A4 inhibitor) increased finerenone AUC by 21%.

Strong or Moderate CYP3A Inducers: Concomitant use of efavirenz (moderate CYP3A4 inducer) and rifampicin (strong CYP3A4 inducer) was predicted to decrease finerenone AUC by 80% and 90%, respectively.

CYP3A4 Substrates: Concomitant use of multiple finerenone 40 mg doses once daily with midazolam (sensitive CYP3A4 substrate) increased the mean AUC by 31% with no effect on C_{max}. There were no clinically significant differences in the pharmacokinetics of midazolam when used concomitantly with multiple finerenone 20 mg doses once daily.

CYP2C8 Substrates: Concomitant use of multiple finerenone 40 mg doses once daily with repaglinide (sensitive CYP2C8 substrate) increased the mean AUC and C_{max} of repaglinide by 59% and 30%, respectively. There were no clinically significant differences in the pharmacokinetics of repaglinide when used concomitantly with multiple finerenone 20 mg doses once daily.

Other Drugs: No clinically significant differences in the pharmacokinetics of the following drugs were observed or predicted when used concomitantly with finerenone: S-warfarin (CYP2C9 substrate), digoxin (P-gp substrate), and rosuvastatin (BCRP and OATP substrate). There was no clinically significant difference in finerenone pharmacokinetics when used concomitantly with gemfibrozil (strong CYP2C8 inhibitor).

13 NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility

Finerenone was non-genotoxic in an in vitro bacterial reverse mutation (Ames) assay, the in vitro chromosomal aberration assay in cultured Chinese hamster V79 cells, or the in vivo micronucleus assay in mice.

In 2-year carcinogenicity studies, finerenone did not show a statistically significant

increase in tumor response in Wistar rats or in CD1 mice. In male mice, Leydig cell adenoma was numerically increased at a dose representing 10 times the $AUC_{unbound}$ in humans and is not considered clinically relevant. Finerenone did not impair fertility in male rats but impaired fertility in female rats at 9 times AUC to the maximum human exposure.

14 CLINICAL STUDIES

14.1 CKD associated with T2DM

FIDELIO-DKD (NCT: 02540993) and FIGARO-DKD (NCT: 02545049) studies were randomized, double-blind, placebo-controlled, multicenter studies in adults with CKD associated with T2DM. In FIDELIO-DKD, patients were required to either have an UACR of 30 to < 300 mg/g, eGFR 25 to < 60 mL/min/1.73m^2 and diabetic retinopathy, or an UACR of ≥ 300 mg/g and an eGFR 25 to < 75 mL/min/1.73m^2 to qualify for enrollment. In FIGARO-DKD, patients needed to have an UACR of 30 mg/g to < 300 mg/g and an eGFR 25 to 90 mL/min/1.73m^2, or an UACR of ≥ 300 mg/g and an eGFR ≥ 60 mL/min/1.73m^2.

Both trials excluded patients with known significant non-diabetic kidney disease. All patients were to have a serum potassium ≤ 4.8 mEq/L at screening and be receiving standard of care background therapy, including a maximum tolerated labeled dose of an angiotensin-converting enzyme inhibitor (ACEi) or angiotensin receptor blocker (ARB). Patients with a clinical diagnosis of chronic heart failure with reduced ejection fraction and persistent symptoms (New York Heart Association class II to IV) were excluded. The starting dose of Kerendia was based on screening eGFR (10 mg once daily in patients with an eGFR of 25 to < 60 mL/min/1.73m^2 and 20 mg once daily in patients with an eGFR ≥ 60 mL/min/1.73m^2). The dose of Kerendia could be titrated during the study, with a target dose of 20 mg daily.

The primary objective of the FIDELIO-DKD study was to determine whether Kerendia reduced the incidence of a sustained decline in eGFR of ≥ 40%, kidney failure (defined as chronic dialysis, kidney transplantation, or a sustained decrease in eGFR to < 15 mL/min/1.73m^2), or renal death. The secondary outcome was a composite of time to first occurrence of CV death, non-fatal MI, non-fatal stroke or hospitalization for heart failure. The primary objective of the FIGARO-DKD study was to determine whether Kerendia reduced the time to first occurrence of CV death, non-fatal MI, non-fatal stroke or hospitalization for heart failure. The secondary outcome was a composite of time to kidney failure, a sustained decline in eGFR 40% or more compared to baseline over at least 4 weeks, or renal death.

In FIDELIO-DKD, a total of 5674 patients were randomized to receive Kerendia (N=2833) or placebo (N=2841) and were followed for a median of 2.6 years. The mean age of the study population was 66 years, and 70% of patients were male. This global trial population was 63% White, 25% Asian, and 5% Black. At baseline, the mean eGFR was 44 mL/min/1.73m^2, with 55% of patients having an eGFR < 45 mL/min/1.73m^2. Median UACR was 852 mg/g, mean glycated hemoglobin A1c (HbA1c) was 7.7%, and the mean blood pressure was 138/76 mmHg. Approximately 46% of patients had a history of atherosclerotic cardiovascular disease and 8% had a history of heart failure. At baseline, 99.8% of patients were treated with an ACEi or ARB. Approximately 97% were on an antidiabetic agent (insulin [64.1%], biguanides [44%], glucagon-like peptide-1 [GLP-1]

receptor agonists [7%], sodium-glucose cotransporter 2 [SGLT2] inhibitors [5%]), 74% were on a statin, and 57% were on an antiplatelet agent.

In FIGARO-DKD, a total of 7352 patients were randomized to receive Kerendia (N=3686) or placebo (N=3666) and were followed for 3.4 years. As compared to FIDELIO-DKD, baseline eGFR was higher in FIGARO-DKD (mean eGFR 68, with 62% of patients having an eGFR ≥ 60 mL/min/1.73m^2) and median UACR was lower (308 mg/g). Otherwise, baseline patient characteristics and background therapies were similar in the two trials.

In FIDELIO-DKD, Kerendia reduced the incidence of the primary composite endpoint of a sustained decline in eGFR ≥ 40%, kidney failure, or renal death (HR 0.82, 95% CI 0.73-0.93, p=0.001) as shown in Table 5 and Figure 1. The treatment effect reflected a reduction in a sustained decline in eGFR ≥ 40% and progression to kidney failure. There were few renal deaths during the trial. Kerendia also reduced the incidence of the secondary composite endpoint of cardiovascular (CV) death, non-fatal myocardial infarction (MI), non-fatal stroke or hospitalization for heart failure (HR 0.86, 95% CI 0.75-0.99, p=0.034) as shown in Table 5 and Figure 3. The treatment effect reflected a reduction in CV death, non-fatal MI, and hospitalization for heart failure. The treatment effect on the primary and secondary composite endpoints was generally consistent across subgroups.

In FIGARO-DKD, Kerendia reduced the incidence of the primary composite endpoint of CV death, non-fatal MI, non-fatal stroke or hospitalization for heart failure (HR 0.87, 95% CI 0.76-0.98, p=0.026) as shown in Table 5 and Figure 4. The treatment effect was mainly driven by an effect on hospitalization for heart failure, though CV death also contributed to the treatment effect. The treatment effect on the primary composite endpoint was generally consistent across subgroups, including patients with and without pre-existing cardiovascular disease. The findings for the renal composite endpoint are shown in Table 5 and Figure 2.

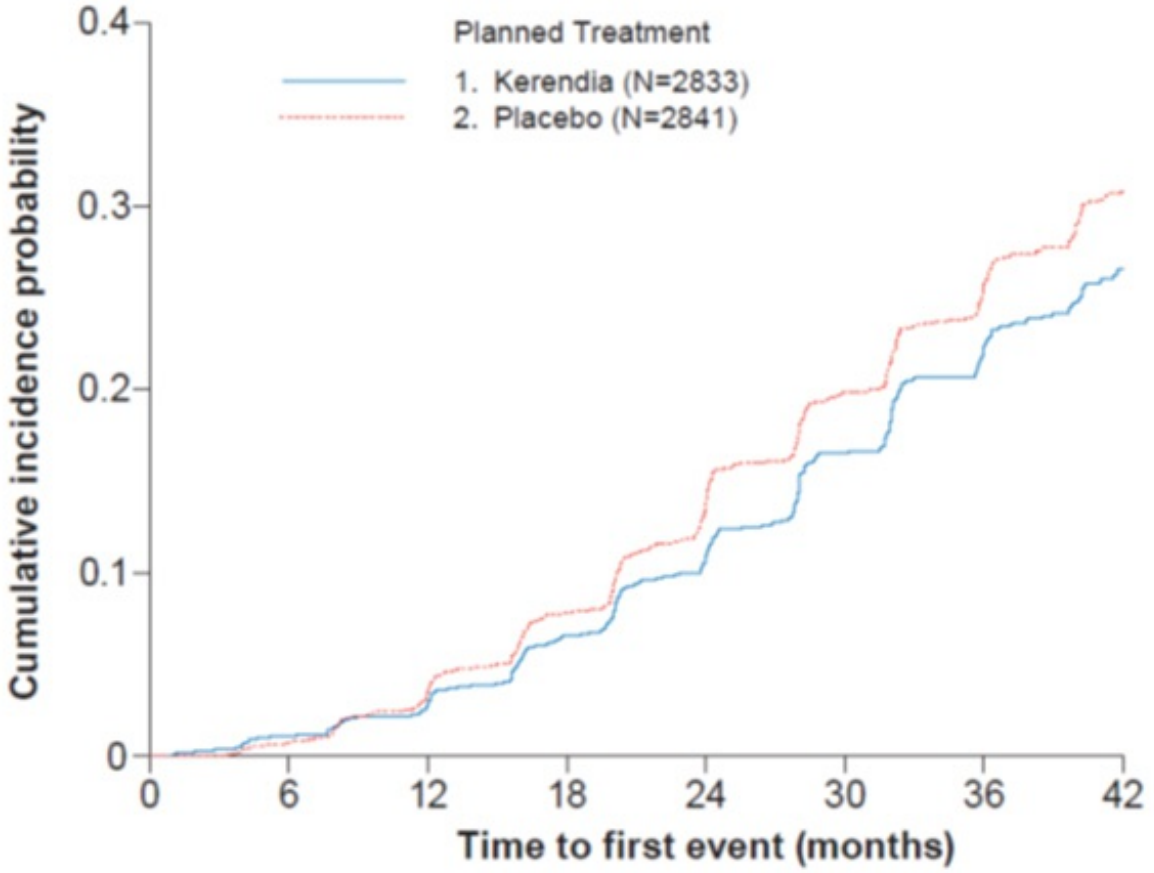
Table 5: Analysis of the Primary and Secondary Time-to-Event Endpoints (and their Individual Components) in Phase 3 Studies FIDELIO-DKD and FIGARO-DKD

	FIDELIO-DKD				**FIGARO-DKD**			
	Kerendia N=2833	**Placebo N=2841**	**Treatment Effect Kerendia / Placebo**		**Kerendia N=3686**	**Placebo N=3666**	**Treatment Effect Kerendia / Placebo**	
Time-to-event Endpoints:	**Event Rate (100 pt-yr)**	**Event Rate (100 pt-yr)**	**Hazard Ratio (95% CI)**	**p-value**	**Event Rate (100 pt-yr)**	**Event Rate (100 pt-yr)**	**Hazard Ratio (95% CI)**	**p-value**
Composite of kidney failure, sustained eGFR decline ≥ 40% or renal death	7.6	9.1	0.82 [0.73; 0.93]	0.001	3.2	3.6	0.87 [0.76; 1.01]	-
Kidney failure	3.0	3.4	0.87 [0.72;	-	0.4	0.5	0.72 [0.49;	-

			1.05]				1.05]	
Sustained eGFR decline ≥ 40%	7.2	8.7	0.81 [0.72; 0.92]	-	3.1	3.5	0.87 [0.75; >1.00]	-
Renal death	-	-	-	-	-	-	-	-
Composite of CV death, non-fatal MI, non-fatal stroke or hospitalization for heart failure	5.1	5.9	0.86 [0.75; 0.99]	0.034	3.9	4.5	0.87 [0.76; 0.98]	0.026
CV death	1.7	2.0	0.86 [0.68; 1.08]	-	1.6	1.8	0.90 [0.73; 1.08]	-
Non-fatal MI	0.9	1.2	0.80 [0.58; 1.09]	-	0.9	0.8	0.99 [0.76; 1.32]	-
Non-fatal stroke	1.2	1.2	1.03 [0.76; 1.38]	-	0.9	0.9	0.97 [0.74; 1.26]	-
Hospitalization for heart failure	1.9	2.2	0.86 [0.68; 1.08]	-	1.0	1.4	0.71 [0.56; 0.90]	-

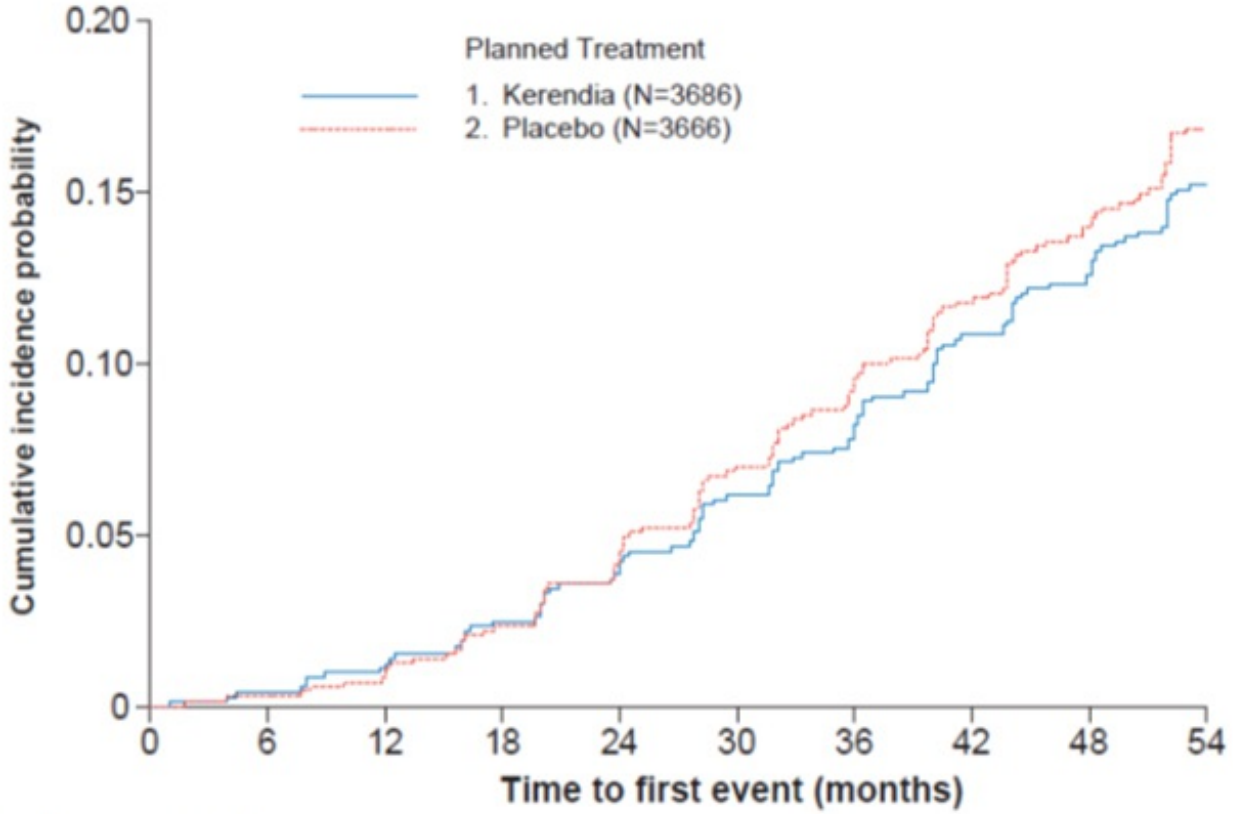
p-value: two-sided p-value from stratified logrank test
CI = confidence interval, CV = cardiovascular, eGFR = estimated glomerular filtration rate, MI = myocardial infarction, N = number of subjects, n = number of subjects with event, pt-yr = patient year.
NOTE: Time to first event was analyzed in a Cox proportional hazards model. For patients with multiple events, only the first event contributed to the composite endpoint. Sums of the numbers of first events for the single components do not add up to the numbers of events in the composite endpoint.

Figure 1: Time to first occurrence of kidney failure, sustained decline in eGFR ≥ 40% from baseline, or renal death in the FIDELIO-DKD study



No. of patients at risk								
Kerendia	2833	2705	2607	2397	1808	1274	787	441
Placebo	2841	2724	2586	2379	1758	1248	792	453

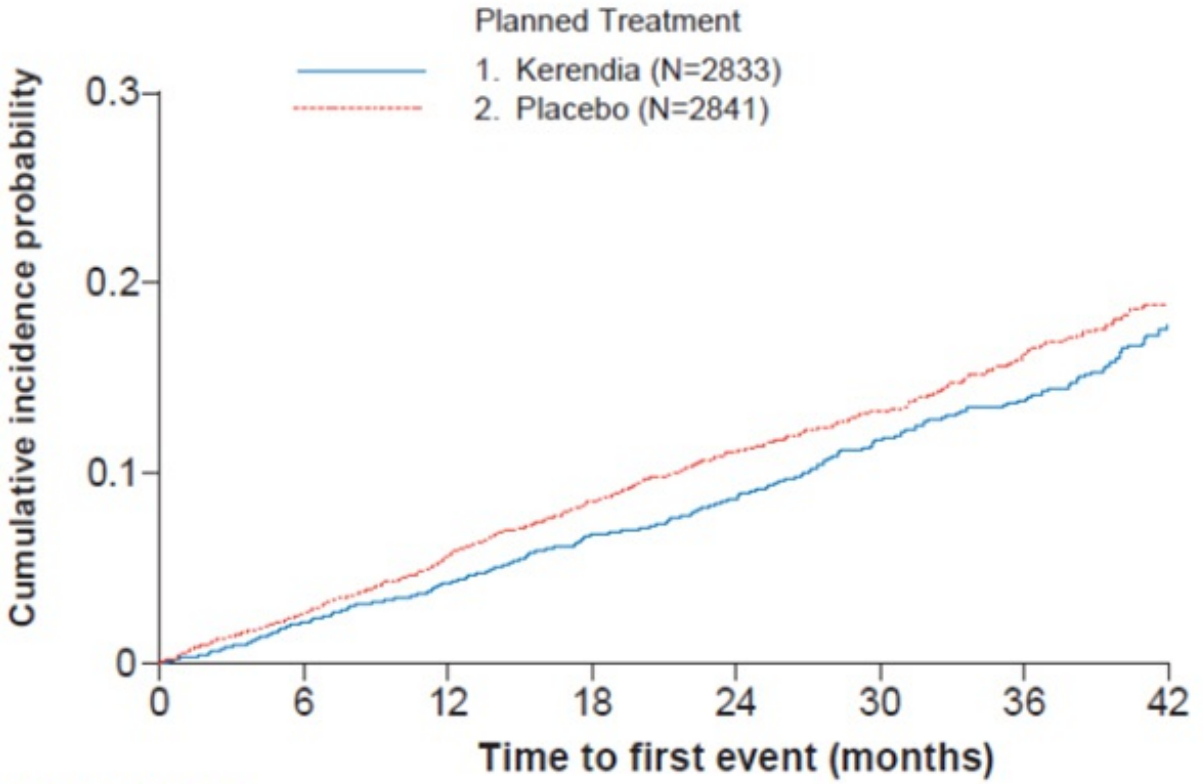
Figure 2: Time to first occurrence of kidney failure, sustained decline in eGFR ≥ 40% from baseline, or renal death in the FIGARO-DKD study



No. of patients at risk

Kerendia	3686	3550	3445	3301	3131	2540	1940	1481	908	480
Placebo	3666	3546	3436	3282	3096	2507	1933	1443	907	485

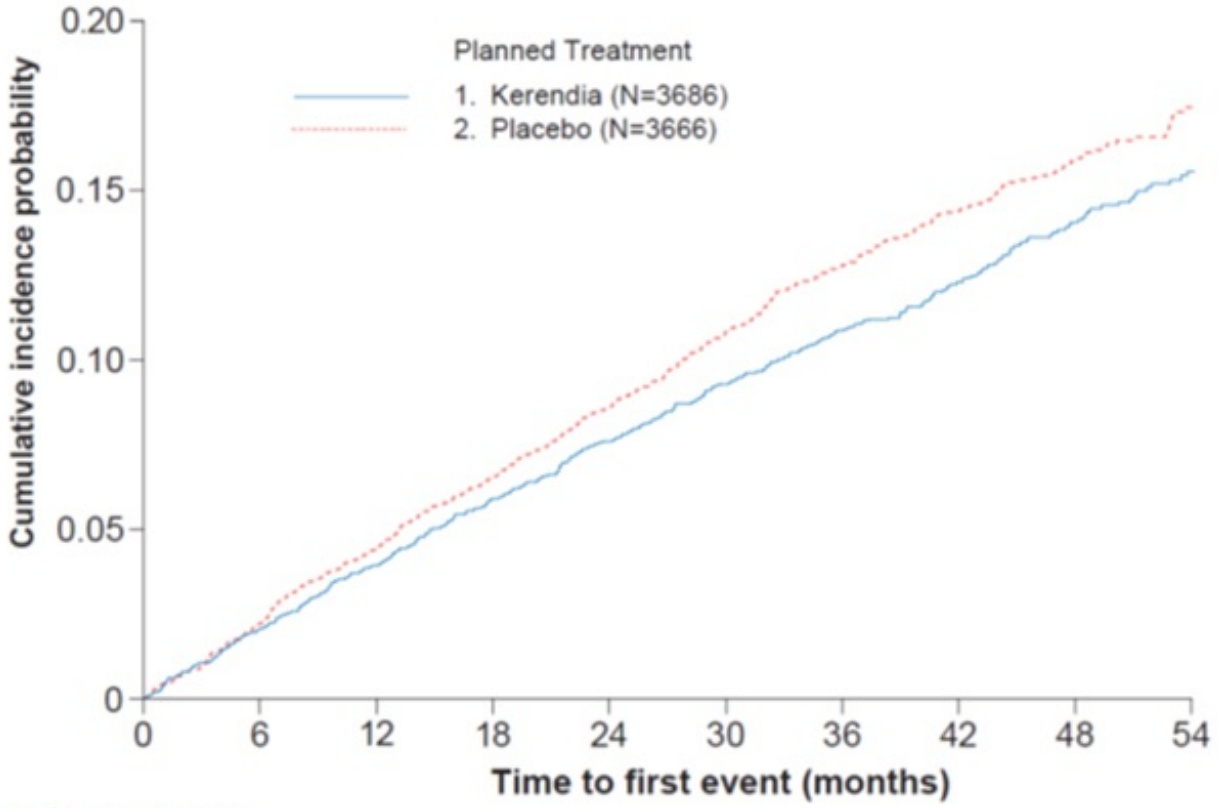
Figure 3: Time to first occurrence of CV death, non-fatal myocardial infarction, non-fatal stroke or hospitalization for heart failure in the FIDELIO-DKD study



No. of patients at risk

Kerendia	2833	2760	2688	2582	2017	1488	984	537
Placebo	2841	2753	2653	2549	1969	1475	951	536

Figure 4: Time to first occurrence of CV death, non-fatal myocardial infarction, non-fatal stroke or hospitalization for heart failure in the FIGARO-DKD study



No. of patients at risk

Kerendia	3686	3600	3517	3427	3320	2781	2184	1712	1093	598
Placebo	3666	3577	3479	3389	3267	2730	2125	1657	1076	585

14.2 CKD associated with T1DM

Use of Kerendia for this indication is supported by evidence from adequate and well-controlled studies in adults with CKD associated with T2DM (FIDELIO-DKD and FIGARO-DKD) with additional pharmacodynamic and safety data in adults with CKD associated with T1DM. Kerendia improved renal outcomes and reduced UACR in adults with CKD associated with T2DM in the FIDELIO-DKD and FIGARO-DKD studies. Therefore, the effect on UACR was used to infer a reduction in the risk of sustained eGFR decline and end-stage kidney disease in adults with CKD associated with T1DM.

FINE-ONE (NCT: 05901831) was a randomized, double-blind, placebo-controlled, multicenter study in 242 adults with CKD associated with T1DM. Patients were required to have an UACR ≥200 mg/g to <5000 mg/g at screening, HbA1c <10%, eGFR 25 to <90 mL/min/1.73m^2, and serum potassium ≤4.8 mEq/L, be continuously treated with insulin, and be receiving a stable dose of an angiotensin-converting enzyme inhibitor (ACEi) or angiotensin receptor blocker (ARB) as standard of care. Patients treated with sodium glucose co-transporter 2 (SGLT2) inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists were excluded from the study. Patients were randomized to receive either Kerendia (N=120) or placebo (N=122) over 6 months. The starting and target dose of Kerendia was the same as the FIDELIO-DKD and FIGARO-DKD studies.

The primary endpoint in FINE-ONE was the change in log-transformed UACR from baseline (evaluated as the ratio to baseline) averaged over Months 3 and 6. Treatment with Kerendia reduced UACR compared to placebo over 6 months (p=0.0001). At

Months 3 and 6, the adjusted relative reduction in UACR comparing Kerendia with placebo was 22% (ratio of Least Square Geometric Mean Ratio (LSGMR) of 0.78; 95% CI: 0.68, 0.90) and 28% (ratio of LSGMR of 0.72; 95% CI: 0.60, 0.86), respectively.

14.3 Heart Failure (LVEF ≥ 40%)

FINEARTS-HF (NCT: 04435626) was a randomized, double-blind, placebo-controlled, multicenter study in adult patients with heart failure (New York Heart Association [NYHA] class II–IV) with documented left ventricular ejection fraction (LVEF) ≥ 40%. Patients were required to have an eGFR ≥25 mL/min/1.73m^2 and serum potassium ≤5.0 mEq/L at screening and randomization and were receiving background heart failure medical treatment, including diuretics. The primary endpoint was the composite of cardiovascular (CV) death and total (first and recurrent) heart failure events comprised of hospitalization for heart failure and urgent heart failure visits.

In FINEARTS-HF, 6001 patients were analyzed; 3003 were randomized to Kerendia and 2998 were randomized to placebo and were followed for a median of 2.7 years. The study included 3247 (54%) patients with a heart failure event in the past 3 months, including 1219 (20%) patients randomized during the hospitalization or within 7 days from the heart failure event. Patients with eGFR ≥ 25 to <60 mL/min/1.73m^2 initiated Kerendia 10 mg and patients with eGFR ≥60 mL/min/1.73m^2 initiated Kerendia 20 mg, and both groups were titrated up to a target dose of Kerendia 20 mg and 40 mg, respectively. At month 6, approximately 65% of the patients reached their target dose. The study population was 79% White, 17% Asian, and 1.5% Black. The mean age at enrollment was 72 years and 46% of patients were female. At baseline, the mean LVEF was 53%, with 64% of patients having an LVEF ≥ 50%, and 69% and 30% of patients were NYHA class II and III, respectively. Mean blood pressure was 129/75 mmHg and mean body mass index (BMI) was 30 kg/m^2. The median NT-proBNP was 1041 pg/mL, the mean eGFR was 62 mL/min/1.73m^2 with 48% of patients having an eGFR <60 mL/min/1.73m^2. Atrial fibrillation was present on screening/baseline ECG for 38% of patients and 41% had diabetes mellitus. The majority of patients were on loop diuretics (87%), an angiotensin-converting enzyme inhibitor (ACEi) or angiotensin receptor blocker (ARB) (79%), or an angiotensin receptor neprilysin inhibitor (ARNI) (9%), and 14% were on sodium-glucose cotransporter 2 (SGLT2) inhibitors.

Kerendia reduced the risk of the primary composite endpoint compared to placebo (Relative Risk [RR] 0.84, 95% CI 0.74-0.95, p=0.007). See Table 6 and Figure 5 below. The Kerendia and placebo event curves separated early and continued to diverge over the study period, see Figure 5 below. Kerendia reduced the risk of the secondary endpoint of total heart failure events (hospitalization for HF or urgent HF visit) compared to placebo (RR 0.82, 95% CI 0.71-0.94, p=0.006). The treatment effect for the primary endpoint was consistent across all pre-specified subgroups, including sex, LVEF, NYHA class, eGFR, time since latest heart failure event, SGLT2 inhibitor therapy, and diabetes mellitus status.

Table 6: Analysis of the Primary Endpoint (and its Individual Components) and Secondary Endpoint in Phase 3 Study FINEARTS-HF

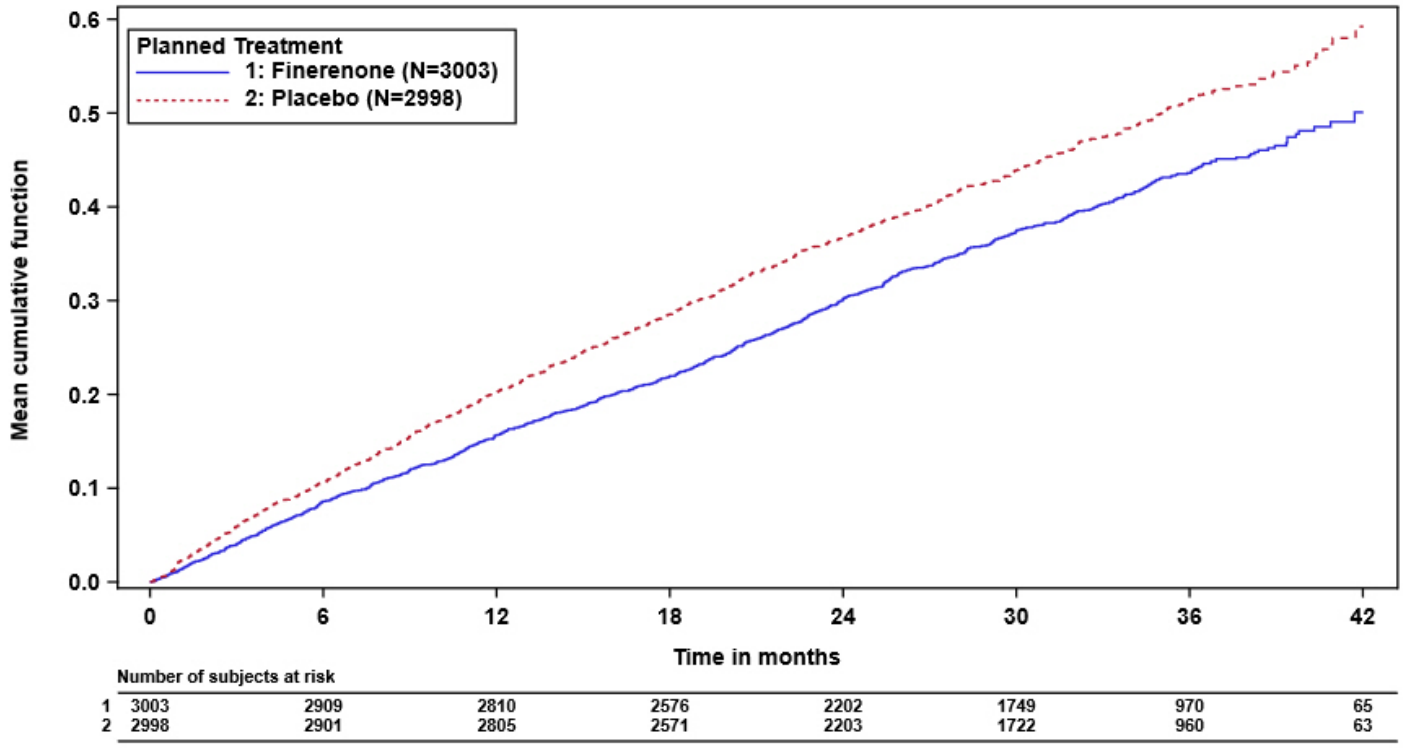
	FINEARTS-HF		
	Kerendia 10 or 20 or 40 mg OD	**Placebo N=2998**	**Treatment Effect Kerendia vs**

	OD N=3003		N=2998		Placebo	
Primary Efficacy Outcome and Components:	**Value**	**Event Rate (100 pt-yr)**	**Value**	**Event Rate (100 pt-yr)**	**Ratio or difference (95% CI)**	**p-value**
Primary composite of CV death and total HF events – E (n; %)	1083 (624; 20.8%)	14.9	1283 (719; 24.0%)	17.7	RR 0.84 (0.74, 0.95)	0.007
Total HF events – E (n; %)*	842 (479; 16.0%)	11.6	1024 (573; 19.1%)	14.1	RR 0.82 (0.71, 0.94)	0.006
CV death – n (%)	242 (8.1%)	3.3	260 (8.7%)	3.6	HR 0.93 (0.78, 1.11)	-

E = total number of events; n = number of patients with an event; RR = rate ratio; HR = hazard ratio; HF = Heart Failure; CV = Cardiovascular.

* Total HF events (hospitalization for HF or urgent HF visit) was a prespecified secondary endpoint.

Figure 5: Mean cumulative functions for primary composite endpoint of CV death and total HF events in Phase 3 study FINEARTS-HF



16 HOW SUPPLIED/STORAGE AND HANDLING

16.1 How Supplied

Kerendia is available as a film-coated tablet in three strengths. The 10 mg is a pink oblong tablet with "FI" on one side of tablet and "10" on the other side of tablet. The 20 mg tablet is a yellow oblong tablet with "FI" on one side of tablet and "20" on the other side of tablet. The 40 mg tablet is a gray-orange oblong tablet with "FI" on one side of tablet and "40" on the other side of tablet.

Kerendia Tablets 10 mg	
Bottle of 30 tablets	NDC 50419-540-01
Bottle of 90 tablets	NDC 50419-540-02
Hospital Blister Pack of 30 tablets	NDC 50419-540-06
Kerendia Tablets 20 mg	
Bottle of 30 tablets	NDC 50419-541-01
Bottle of 90 tablets	NDC 50419-541-02
Hospital Blister Pack of 30 tablets	NDC 50419-541-06
Kerendia Tablets 40 mg	
Bottle of 30 tablets	NDC 50419-542-01
Bottle of 90 tablets	NDC 50419-542-02
Hospital Blister Pack of 30 tablets	NDC 50419-542-06

16.2 Storage and Handling

Store at 20°C to 25°C (68°F to 77°F); excursions are permitted from 15°C to 30°C (59°F to 86°F*) [see USP Controlled Room Temperature].*

17 PATIENT COUNSELING INFORMATION

Advise patients of the need for periodic monitoring of serum potassium levels. Advise patients receiving Kerendia to consult with their physician before using potassium supplements or salt substitutes containing potassium *[see Warnings and Precautions (5.1)].*

Advise patients to avoid strong or moderate CYP3A4 inducers and to find alternative medicinal products with no or weak potential to induce CYP3A4 *[see Drug Interactions (7.1)].*

Avoid concomitant intake of grapefruit or grapefruit juice as it is expected to increase the plasma concentration of finerenone *[see Drug Interactions (7.1)].*

Advise women that breastfeeding is not recommended at the time of treatment with Kerendia and for 1 day after treatment *[see Use in Specific Populations (8.2)].*

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Manufactured for:

Bayer HealthCare Pharmaceuticals Inc.
Whippany, NJ 07981

Manufactured in Germany

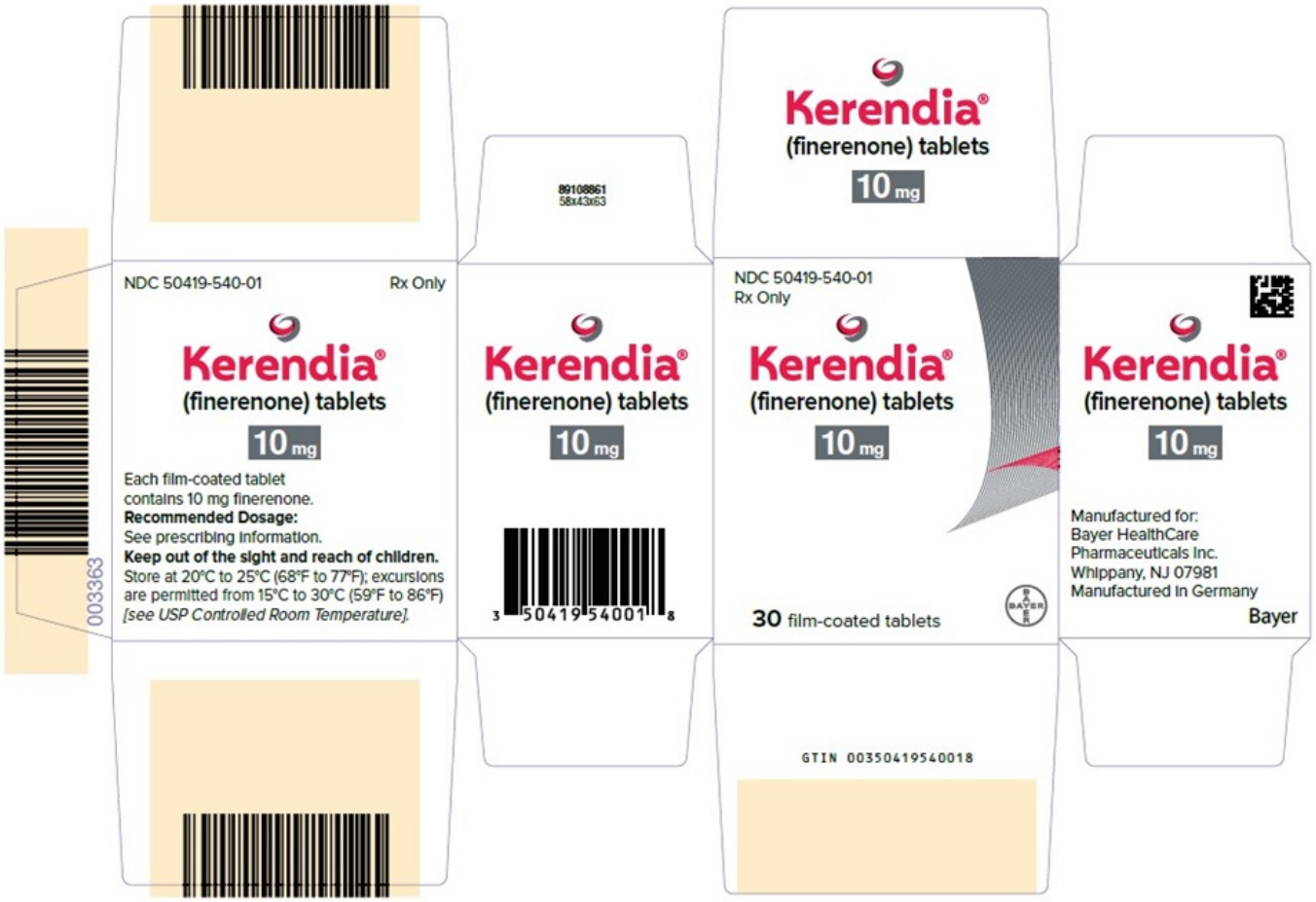
PRINCIPAL DISPLAY PANEL- 10 mg Tablet Carton

NDC 50419-540-01
Rx only

Kerendia®
(finerenone) tablets
10 mg

30 film-coated tablets

BAYER



PRINCIPAL DISPLAY PANEL- 20 mg Tablet Carton

NDC 50419-541-01
Rx only

Kerendia®
(finerenone) tablets
20 mg

30 film-coated tablets

BAYER



PRINCIPAL DISPLAY PANEL- 40 mg Tablet Carton

NDC 50419-542-01
Rx only

Kerendia®
(finerenone) tablets
40 mg

30 film-coated tablets

BAYER



KERENDIA

finerenone tablet, film coated

Product Information			
Product Type	HUMAN PRESCRIPTION DRUG	**Item Code (Source)**	NDC:50419-540
Route of Administration	ORAL		

Active Ingredient/Active Moiety		
Ingredient Name	**Basis of Strength**	**Strength**
FINERENONE (UNII: DE2O63YV8R) (FINERENONE - UNII:DE2O63YV8R)	FINERENONE	10 mg

Inactive Ingredients	
Ingredient Name	**Strength**
LACTOSE MONOHYDRATE (UNII: EWQ57Q8I5X)	
MICROCRYSTALLINE CELLULOSE (UNII: OP1R32D61U)	
CROSCARMELLOSE SODIUM (UNII: M28OL1HH48)	
HYPROMELLOSE, UNSPECIFIED (UNII: 3NXW29V3WO)	
MAGNESIUM STEARATE (UNII: 70097M6I30)	
SODIUM LAURYL SULFATE (UNII: 368GB5141J)	

Product Characteristics			
Color	PINK	**Score**	no score
Shape	OVAL	**Size**	4mm
Flavor		**Imprint Code**	10;FI
Contains			

Packaging

#	Item Code	Package Description	Marketing Start Date	Marketing End Date
1	NDC:50419-540-01	30 in 1 BOTTLE, PLASTIC; Type 0: Not a Combination Product	07/09/2021	
2	NDC:50419-540-02	90 in 1 BOTTLE, PLASTIC; Type 0: Not a Combination Product	07/09/2021	
3	NDC:50419-540-70	7 in 1 BOTTLE, PLASTIC; Type 0: Not a Combination Product	07/09/2021	
4	NDC:50419-540-06	10 in 1 BLISTER PACK; Type 0: Not a Combination Product	08/23/2025	

Marketing Information

Marketing Category	Application Number or Monograph Citation	Marketing Start Date	Marketing End Date
NDA	NDA215341	07/09/2021	

KERENDIA

finerenone tablet, film coated

Product Information			
Product Type	HUMAN PRESCRIPTION DRUG	**Item Code (Source)**	NDC:50419-541
Route of Administration	ORAL		

Active Ingredient/Active Moiety

Ingredient Name	Basis of Strength	Strength
FINERENONE (UNII: DE2O63YV8R) (FINERENONE - UNII:DE2O63YV8R)	FINERENONE	20 mg

Inactive Ingredients

Ingredient Name	Strength
LACTOSE MONOHYDRATE (UNII: EWQ57Q8I5X)	
MICROCRYSTALLINE CELLULOSE (UNII: OP1R32D61U)	
CROSCARMELLOSE SODIUM (UNII: M28OL1HH48)	
HYPROMELLOSE, UNSPECIFIED (UNII: 3NXW29V3WO)	
MAGNESIUM STEARATE (UNII: 70097M6I30)	
SODIUM LAURYL SULFATE (UNII: 368GB5141J)	

Product Characteristics			
Color	YELLOW	**Score**	no score
Shape	OVAL	**Size**	4mm
Flavor		**Imprint Code**	20;FI
Contains			

Packaging

#	Item Code	Package Description	Marketing Start Date	Marketing End Date
1	NDC:50419-541-01	30 in 1 BOTTLE, PLASTIC; Type 0: Not a Combination Product	07/09/2021	
2	NDC:50419-541-02	90 in 1 BOTTLE, PLASTIC; Type 0: Not a Combination Product	07/09/2021	
3	NDC:50419-541-70	7 in 1 BOTTLE, PLASTIC; Type 0: Not a Combination Product	07/09/2021	
4	NDC:50419-541-06	10 in 1 BLISTER PACK; Type 0: Not a Combination Product	08/23/2025	

Marketing Information

Marketing Category	Application Number or Monograph Citation	Marketing Start Date	Marketing End Date
NDA	NDA215341	07/09/2021	

KERENDIA

finerenone tablet

Product Information			
Product Type	HUMAN PRESCRIPTION DRUG	**Item Code (Source)**	NDC:50419-542
Route of Administration	ORAL		

Active Ingredient/Active Moiety

Ingredient Name	Basis of Strength	Strength
FINERENONE (UNII: DE2O63YV8R) (FINERENONE - UNII:DE2O63YV8R)	FINERENONE	40 mg

Inactive Ingredients

Ingredient Name	Strength
LACTOSE MONOHYDRATE (UNII: EWQ57Q8I5X)	
MICROCRYSTALLINE CELLULOSE (UNII: OP1R32D61U)	
CROSCARMELLOSE SODIUM (UNII: M28OL1HH48)	
HYPROMELLOSE, UNSPECIFIED (UNII: 3NXW29V3WO)	
MAGNESIUM STEARATE (UNII: 70097M6I30)	

SODIUM LAURYL SULFATE (UNII: 368GB5141J)	

Product Characteristics

Color	ORANGE (gray-orange)	**Score**	no score
Shape	OVAL	**Size**	11mm
Flavor		**Imprint Code**	40;FI
Contains			

Packaging

#	Item Code	Package Description	Marketing Start Date	Marketing End Date
1	NDC:50419-542-01	30 in 1 BOTTLE, PLASTIC; Type 0: Not a Combination Product	07/11/2025	
2	NDC:50419-542-02	90 in 1 BOTTLE, PLASTIC; Type 0: Not a Combination Product	07/11/2025	
3	NDC:50419-542-70	7 in 1 BOTTLE, PLASTIC; Type 0: Not a Combination Product	07/11/2025	
4	NDC:50419-542-06	10 in 1 BLISTER PACK; Type 0: Not a Combination Product	07/11/2025	

Marketing Information

Marketing Category	Application Number or Monograph Citation	Marketing Start Date	Marketing End Date
NDA	NDA215341	07/09/2021	

Labeler - Bayer HealthCare Pharmaceuticals Inc. (005436809)

Registrant - Bayer AG (314947622)

Establishment

Name	Address	ID/FEI	Business Operations
Bayer AG		314947622	MANUFACTURE(50419-540, 50419-541, 50419-542) , ANALYSIS(50419-540, 50419-541, 50419-542) , PARTICLE SIZE REDUCTION(50419-540, 50419-541, 50419-542) , PACK(50419-540, 50419-541, 50419-542)

Establishment

Name	Address	ID/FEI	Business Operations
Sharp Corporation		143696495	PACK(50419-540, 50419-541, 50419-542) , LABEL(50419-540, 50419-541, 50419-542)

Establishment

Name	Address	ID/FEI	Business Operations
Bayer AG		323208116	API MANUFACTURE(50419-540, 50419-541, 50419-542) , ANALYSIS(50419-540, 50419-541, 50419-542)

Establishment

Name	Address	ID/FEI	Business Operations
Bayer AG		342872971	PARTICLE SIZE REDUCTION(50419-540, 50419-541, 50419-542) , PACK(50419-540, 50419-541, 50419-542)

Establishment

Name	Address	ID/FEI	Business Operations
Bayer AG		314398484	ANALYSIS(50419-540, 50419-541, 50419-542)

Establishment

Name	Address	ID/FEI	Business Operations
AMPAC Fine Chemicals, Rancho Cordova		073903937	API MANUFACTURE(50419-540, 50419-541, 50419-542)

Establishment

Name	Address	ID/FEI	Business Operations
Delpharm Milano Srl		437999026	LABEL(50419-540, 50419-541, 50419-542) , PACK(50419-540, 50419-541, 50419-542)