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# A COMPREHENSIVE REVIEW ARTICLE ON FINERENONE

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**ABSTRACT**

*Finerenone is a Novel selective Non-steroidal Mineralocorticoids Receptor Antagonist. Finerenone is indicated for the treatment of Chronic kidney disease (stage 3 and stage 4) associated with type 2 diabetes in adults. Finerenone blocks the MR receptor so that the progression of chronic kidney disease is control. Finerenone belongs to the BCS class-II, low solubility and low half life and bioavailbility.*

**KEYWORDS** : *Finerenone, Chronic kidney disease, Solubility, BCS Class.*

## INTRODUCTION

Finerenone belongs to BCS class –II (Low solubility and High Permeability).Finerenone having a molecular weight about 378 gm/mol and about 211-218´C. Finerenone is chemically called as (S)-4-(3-cyano 5-methoxyphenyl)-5-ethoxy-2, 8-dimethyl-1, 4-dihydro-1, 6-naph thyridine-3-carboxamide. Finerenone (BAY94-8862) is a novel non steroidal MRA with more potential than spironolactone and greater affinity than eplerenone in vitro. Finerenone structural activity has a strong binding mode within Mineralocorticoid Receptor MR. Finerenone is white crystalline powder and odourless.

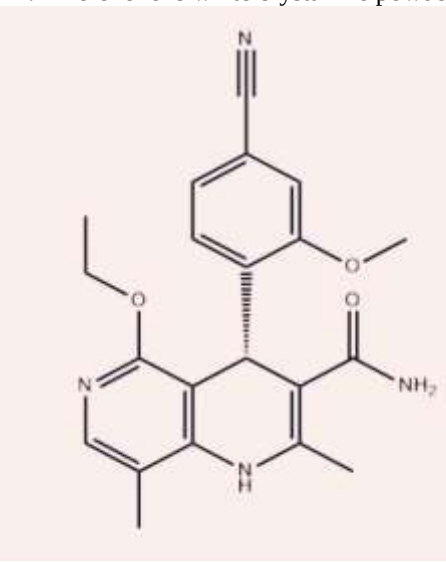


**Image No.1 structure of Finerenone**

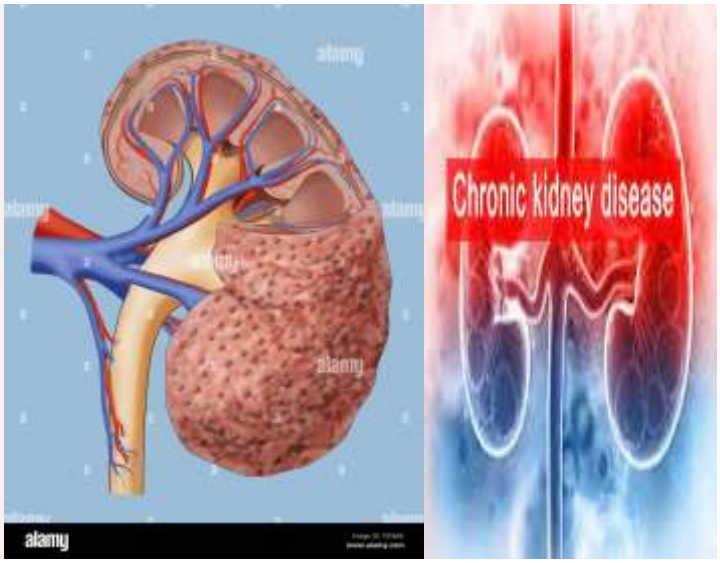


**Image No.2 & 3 Kidney damage in Chronic kidney disease**

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## FINERENONE PHYSICOCHEMICAL, PHARMACOKINETICS AND PHARMACODYNAMICS PROPERTIES.

Finerenone has a physicochemical property like high lipophilicity and Polarity, low solubility which govern high plasma protein binding, transport, tissue penetration and distribution. Finerenone has a low dissolution and having a less bioavailability which show at 10, 20mg dose in twice in day as per requirement of patients and its stored in a room temperature.

Finerenone is metabolized by CYP3A4 (90%) and CYP2C8 (10%). However, renal dysfunction alters clearance of the medication. Patients with different creatinine clearances (CrCl) had the same maximum serum concentration, but elimination half-life was prolonged in those with worse kidney function: < 30 ml/min/m2 (3.0 h), 30–50 ml/min/m2 (2.88 h), 50–80 ml/min/m2 (2.34 h), and > 80 ml/min/m2 (2.23 h). Given its significant protein binding capacity, Finerenone is also impacted by serum albumin levels; thus, hypoalbuminemia (e.g., nephrotic syndrome, malnutrition) may result in increased blood levels of the drug . The investigator found that Finerenone was rapidly absorbed during fasting condition (more absorbed with 10-40 mg PEG) with a median time to maximum plasma concentration (t max) of 0.5–1 h, exhibiting dose-linear pharmacokinetics and rapid elimination from plasma (geometric mean terminal half-life (t½) of 1.7– 2.83 h). In the postprandial state, the elimination rate from plasma was affected, but not the absorption. They also concluded that Finerenone did not influence laboratory parameters such as urinary electrolytes, serum aldosterone, and AGII. Finerenone was found to be tolerable with favorable pharmacokinetics despite the prandial state.

## COMPARISON BETWEEN NONSTEROIDAL MR ANTAGONIST (FINERENONE) AND STEROIDAL MR ANTAGONISTS

As detailed in a recent scholarly review, there are many distinct differences between the nonsteroidal MRA (finerenone) and steroidal MRAs (spironolactone and eplerenone) . They differ with respect to the mode of MR antagonism, tissue distribution, pharmacokinetics, effects on cofactor recruitment, and effects on inflammation and fibrosis in rodent models of cardiac fibrosis and CKD. For the purposes of the present review, 3 attributes will be highlighted:

1. Pharmacokinetics: Finerenone has no active metabolites and a short half-life and low solubility. In contrast, spironolactone is a prodrug with multiple active metabolites with long half-lives. Eplerenone has no active metabolites but has a half-life of 4–6 h
2. Effect on inflammation and fibrosis: Finerenone (at equi-natriuretic doses to eplerenone) manifests strong inhibition of inflammation and fibrosis
3. Effect on cofactor recruitment in the absence of aldosterone in vitro: Finerenone acts as an inverse agonist (inhibits cofactor binding in the absence of aldoste rone). In contrast, both spironolactone and eplerenone act as partial agonists for cofactor recruitment.
4. Animal studies showed that finerenone has anti-inflammatory and anti-fibrotic effects and consequently beneficial cardiorenal effects.

## MECHANISMS OF ACTION

Finerenone inhibits the effects of mineralocorticoids like aldosterone and cortisol when the MR is overactivated, possibly reducing inflammation and fibrosis in the heart and kidney. Aldosterone is produced when the renin-angiotensin-aldosterone system pathway is activated, and this pathway has a role in regulating blood pressure and sodium and fluid retention.

## ADVERSED EFFECT

Finerenone may cause electrolyte imbalances that must be resolved by a healthcare professional. In the case of potassium, patients taking Finerenone may experience a higher level of potassium in the blood. Symptoms that correlate to this clinical finding include nausea, weakness, chest pain and loss of movement.[14] Another common electrolyte imbalance which may occur for patients on Finerenone is that patients may have low sodium, which can manifest as headaches, confusion, weakness and feeling off balance for patients.

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## MARKETED FORMULATION


**Finerenone (Kerendia) 10,20 mg**

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