Mavodelpar

Cat. No.: HY-112597A

CAS No.: 1604815-32-8

Molecular Formula: $C_{31}H_{29}FNNaO_5$

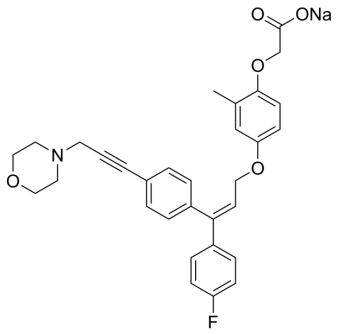
Molecular Weight: 537.55

Target: PPAR

Pathway: Cell Cycle/DNA Damage; Vitamin D Related/Nuclear Receptor

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (186.03 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.8603 mL	9.3015 mL	18.6029 mL
	5 mM	0.3721 mL	1.8603 mL	3.7206 mL
	10 mM	0.1860 mL	0.9301 mL	1.8603 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (4.65 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.65 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.65 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Mavodelpar (REN001) is a selective PPARδ agonist. Mavodelpar suppresses glomerular injury and renal fibrosis. Mavodelpar can be used for the research of primary mitochondrial myopathies (PMM) and long-chain fatty acid oxidation disorders (LC-FAOD)[1]. Mavodelpar is a click chemistry reagent, it contains an Alkyne group and can undergo copper-catalyzed azide-alkyne cycloaddition (CuAAc) with molecules containing Azide groups.

In Vivo

Mavodelpar (10 mg/kg; i.p., once daily, from 6 to 17 weeks of age) effectively suppresses glomerular injury and renal fibrosis, and decreases levels of fibrosis-related proteins[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male and female B6129SF1-Col4a3$^{-/-}$ mice[1]
Dosage:	10 mg/kg
Administration:	Intraperitoneal injection; 10 mg/kg, once daily, from 6 to 17 weeks of age
Result:	Suppressed proteinuria and blood urea nitrogen (BUN) levels. Reduced glomerular injury, renal fibrosis, phosho-Stat3 and connective tissue growth factor (CTGF) levels. Decreased the expression level of the activated fibroblast marker alpha-SMA and Collagen I and IV.

REFERENCES

[1]. Omachi K, et al. PPARδ agonism ameliorates renal fibrosis in an Alport syndrome mouse model. Kidney360. 2022 Nov 29.

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