Elafibranor

Cat. No.: HY-16737

CAS No.: 923978-27-2

Molecular Formula: $C_{22}H_{24}O_4S$

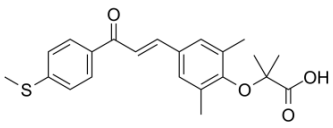
Molecular Weight: 384.49

Target: PPAR

Pathway: Cell Cycle/DNA Damage

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 33 mg/mL (85.83 mM)

H_2O : < 0.1 mg/mL (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.6008 mL	13.0042 mL	26.0085 mL
5 mM	0.5202 mL	2.6008 mL	5.2017 mL
10 mM	0.2601 mL	1.3004 mL	2.6008 mL

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

In Vivo

1. Add each solvent one by one: 1% DMSO >> 99% saline
   Solubility: 0.57 mg/mL (1.48 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.17 mg/mL (5.64 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.17 mg/mL (5.64 mM); Clear solution
4. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: 2.87 mg/mL (7.46 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description Elafibranor (GFT505) is a PPARα/δ agonist with EC_{50}s of 45 and 175 nM, respectively.

IC_{50} & Target	PPAR-α	PPAR-δ

	45 nM (EC50)	175 nM (EC50)
In Vitro	Elafibranor (GFT505) is being developed as a dual PPAR-α/PPAR-δ agonist for the treatment of T2DM and non-alcoholic fatty liver disease. Elafibranor has an active metabolite, GFT1007, and both have potent agonist activity for PPAR-α and to a lesser extent for PPAR-δ[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.	
In Vivo	Elafibranor is well tolerated and does not cause weight gain or cardiac events, but does produce a mild, reversible increase in serum creatinine. Elafibranor improves glucose homeostasis, and lipid metabolism and reduces inflammation[2]. Elafibranor (GFT505) treatment improves glucose control and plasma lipids in diabetic db/db mice. A significant dose-dependent reduction of hepatic expression of the key gluconeogenic enzymes glucose 6-phosphatase (G6Pase), PEPCK, and fructose 1,6-bisphosphatase 1 (FBP1) is observed with Elafibranor. Elafibranor does not induce cardiac adverse effects of PPARγ-activating agonists in monkeys[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.	

CUSTOMER VALIDATION

- Int J Mol Sci. 2019 Apr 2;20(7). pii: E1629.
- Cells. 2020 Apr 14;9(4):964.
- Mol Pharmacol. 2018 Jun;93(6):563-574.
- PLoS One. 2020 Dec 16;15(12):e0243911.
- Hepatology Communications. 2020 Nov 29.

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REFERENCES

[1]. Liu ZM, et al. Early investigational drugs targeting PPAR-α for the treatment of metabolic disease.Expert Opin Investig Drugs. 2015 May;24(5):611-21.

[2]. Ratziu V, et al. Elafibranor, an Agonist of the Peroxisome Proliferator-Activated Receptor-α and -δ, Induces Resolution of Nonalcoholic Steatohepatitis Without Fibrosis Worsening. Gastroenterology. 2016 May;150(5):1147-1159.

[3]. Hanf R, et al. The dual peroxisome proliferator-activated receptor alpha/delta agonist GFT505 exerts anti-diabetic effects in db/db mice without peroxisome proliferator-activated receptor gamma-associated adverse cardiac effects.Diab Vasc Dis Res. 2014 No

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