Fezagepras sodium

Cat. No.: HY-100775

CAS No.: 1254472-97-3

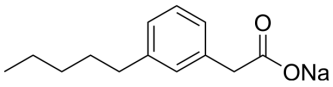
Molecular Formula: $C_{13}H_{17}NaO_2$

Molecular Weight: 228.26

Target: Free Fatty Acid Receptor; GPR84

Pathway: GPCR/G Protein

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

H_2O : ≥ 100 mg/mL (438.10 mM)
DMSO : ≥ 64 mg/mL (280.38 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.3810 mL	21.9048 mL	43.8097 mL
5 mM	0.8762 mL	4.3810 mL	8.7619 mL
10 mM	0.4381 mL	2.1905 mL	4.3810 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 120 mg/mL (525.72 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (9.11 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (9.11 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (9.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Fezagepras (Setogepram) sodium acts as an orally active agonist for GPR40 and as an antagonist or inverse agonist for GPR84[1]. Fezagepras sodium decreases renal, liver and pancreatic fibrosis[1][2]. Fezagepras sodium exerts anti-fibrotic, anti-inflammatory and anti-proliferative actions[2].

IC_{50} & Target

GPR40, GPR84[1]

In Vitro

Fezagepras sodium (500 μM; 24 hours) inhibits TGF-β (10 ng/mL)-activated human hepatic stellate cells (HSCs) proliferation [2].

Fezagepras sodium (250 or 500 μM; 24 hours) dose-dependently arrests HSCs at the G0/G1 phase without inducing apoptosis [2].

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

Cell Proliferation Assay[2]

Cell Line:	HSCs
Concentration:	250 or 500 μM
Incubation Time:	24 hours
Result:	Inhibited TGF-β-activated HSC proliferation. TGF-β (10 ng/mL) increased HSC proliferation by 10%.

Cell Cycle Analysis[2]

Cell Line:	HSCs
Concentration:	250 μM, 500 μM
Incubation Time:	24 hours
Result:	Inhibited cell cycle progression.

In Vivo

Fezagepras sodium (100 mg/kg/day; gavage from 8-20 weeks of age) markedly decreases hyperglycemia and markedly improvea glucose tolerance in type 2 diabetes eNOS$^{-/-}$db/db mice[1].·

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

Animal Model:	Type 2 diabetes eNOS$^{-/-}$db/db mice[1]
Dosage:	100 mg/kg/day
Administration:	Given via daily gavage from 8-20 weeks
Result:	Compared with vehicle-treated mice, hyperglycemia was markedly decreased, and glucose tolerance was markedly improved.

REFERENCES

[1]. Li Y, et al. Fatty acid receptor modulator PBI-4050 inhibits kidney fibrosis and improves glycemic control. JCI Insight. 2018 May 17;3(10). pii: 120365.

[2]. Grouix B, et al. PBI-4050 Reduces Stellate Cell Activation and Liver Fibrosis through Modulation of Intracellular ATP Levels and the Liver Kinase B1/AMP-Activated Protein Kinase/Mammalian Target of Rapamycin Pathway. J Pharmacol Exp Ther. 2018 Oct;367(1):71-81.

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