Prucalopride

Cat. No.: HY-14151

CAS No.: 179474-81-8

Molecular Formula: $C_{18}H_{26}ClN_3O_3$

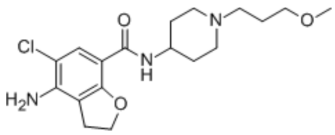
Molecular Weight: 367.87

Target: 5-HT Receptor; Apoptosis; Autophagy

Pathway: GPCR/G Protein; Neuronal Signaling; Apoptosis; Autophagy

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (135.92 mM; Need ultrasonic)

DMSO : ≥ 31 mg/mL (84.27 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.7184 mL	13.5918 mL	27.1835 mL
	5 mM	0.5437 mL	2.7184 mL	5.4367 mL
	10 mM	0.2718 mL	1.3592 mL	2.7184 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 50 mg/mL (135.92 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Prucalopride is an orally active, selective and specific 5-HT 4 receptor agonist (high affinity), with pK_is of 8.6 and 8.1 for human 5-HT4a/4b receptors, respectively. Prucalopride improves intestinal motility by promoting regeneration of the intestinal nervous system in rats. Prucalopride also shows anticancer activity by blocking of the PI3K/AKT/mTor signaling pathway. Prucalopride can be used in studies of chronic constipation, pseudo-intestinal obstruction and cancer[1][2][3].

IC_{50} & Target

5-HT_{4A} Receptor	5-HT_{4B} Receptor
8.6 (pKi)	8.1 (pKi)

In Vitro

Prucalopride (10 μM; 24, 48, 72 h) shows anti proliferative activity in A549 cells[1].

Prucalopride induces autophagy and apoptosis, decreases the expression of the phosphorylated protein kinase B (AKT) and

mammalian target of rapamycin (mTor) in A549/A427 cells[1].

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

Cell Proliferation Assay[1]

Cell Line:	A549 cells
Concentration:	10 μM
Incubation Time:	24, 48, 72 h
Result:	Repressed lung cancer cells proliferation.

In Vivo

Prucalopride (5, 10 μg/kg; p.o.; single daily for 2 weeks) shortens the colonic transit time in DM model, promotes the regeneration of colonic neural stem cells and neurons[2].

Prucalopride (5, 10 μg/kg; p.o.; single daily for 2 weeks) promotes the differentiation of colonic neural stem cells, activates the expression of glial proteins and promotes the recovery of neuronal injury to a certain extent[2].

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

Animal Model:	Sprague dawley rats (diabetes mellitus (DM) model)[2].
Dosage:	5 or 10 μg/kg
Administration:	Oral gavage; single daily for 2 weeks.
Result:	Accelerated colonic movement and shortened the colonic transit time, and markedly increased the expression levels of Ki67. Increased expression of SOX10 in the columnar epithelial nuclei and enteraden (when at 5 μg/kg), and in the columnar epithelial cells, the nuclei of lamina propria cells and enteraden (when at 10 μg/kg). Significantly increased Nestin expression, which concentrated in columnar epithelial cells and the mesenchyme. (Nestin:a marker of enteric neural stem cells in the ENS).

CUSTOMER VALIDATION

- Biochem Biophys Res Commun. 2021 Apr 6;556:16-22.

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REFERENCES

[1]. Chen M, et al. Prucalopride inhibits lung cancer cell proliferation, invasion, and migration through blocking of the PI3K/AKT/mTor signaling pathway. Hum Exp Toxicol. 2020 Feb;39(2):173-181.

[2]. Wang Y, et al. Prucalopride might improve intestinal motility by promoting the regeneration of the enteric nervous system in diabetic rats. Int J Mol Med. 2022 Jul;50(1):87.

[3]. Briejer MR, et al. The in vitro pharmacological profile of prucalopride, a novel enterokinetic compound. Eur J Pharmacol. 2001 Jun 29;423(1):71-83.

Caution: Product has not been fully validated for medical applications. For research use only.

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