Ibiglustat (L-Malic acid)

Cat. No.: HY-16743A

CAS No.: 1629063-78-0

Molecular Formula: $C_{24}H_{30}FN_3O_7S$

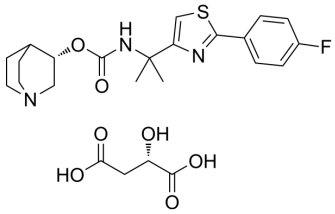
Molecular Weight: 523.57

Target: Glucosylceramide Synthase (GCS)

Pathway: Neuronal Signaling

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 : ≥ 100 mg/mL (191.00 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9100 mL	9.5498 mL	19.0996 mL
5 mM	0.3820 mL	1.9100 mL	3.8199 mL
10 mM	0.1910 mL	0.9550 mL	1.9100 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 (4.77 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.77 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.77 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ibiglustat (Venglustat) L-Malic acid is an orally active, brain-penetrant glucosylceramide synthase (GCS) inhibitor. Ibiglustat L-Malic acid can be used for the research of Gaucher disease type 3, Parkinson's disease associated with GBA mutations, Fabry disease, GM2 gangliosidosis, and autosomal dominant polycystic kidney disease[1][2].

IC_{50} & Target

Glucosylceramide synthase[1].

In Vitro	Ibiglustat (SAR402671) (1 μM, 15 days) L-Malic acid treated Fabry disease (FD) cells are close to the physiological level in untreated WT cells in GL-3 levels, suggesting that Ibiglustat L-Malic acid can prevent additional GL-3 accumulation and could serve to ameliorate the abundant levels of this sphingolipid in FD cardiomyocytes[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- FASEB J. 2020 Dec;34(12):15922-15945.
- bioRxiv. 2024 Jan 29.

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REFERENCES

[1]. Iva Stojkovska, et al. Molecular mechanisms of α-synuclein and GBA1 in Parkinson's disease. Cell Tissue Res. 2017.

[2]. Itier JM, et al. Effective clearance of GL-3 in a human iPSC-derived cardiomyocyte model of Fabry disease. J Inherit Metab Dis. 2014 Nov;37(6):1013-22.

[3]. Viel C, et al. Preclinical pharmacology of glucosylceramide synthase inhibitor venglustat in a GBA-related synucleinopathy model. Sci Rep. 2021;11(1):20945. Published 2021 Oct 22.

[4]. Peterschmitt MJ, et al. Pharmacokinetics, Pharmacodynamics, Safety, and Tolerability of Oral Venglustat in Healthy Volunteers. Clin Pharmacol Drug Dev. 2021;10(1):86-98.

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