Ibiglustat succinate

Cat. No.: HY-16743B

CAS No.: 1629063-80-4

Molecular Formula: $C_{24}H_{30}FN_3O_6S$

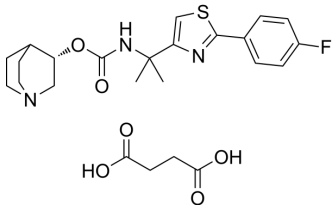
Molecular Weight: 507.57

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 : ≥ 250 mg/mL (492.54 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9702 mL	9.8509 mL	19.7017 mL
5 mM	0.3940 mL	1.9702 mL	3.9403 mL
10 mM	0.1970 mL	0.9851 mL	1.9702 mL

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

BIOLOGICAL ACTIVITY

Description

Ibiglustat (Venglustat) succinate is an orally active, brain-penetrant glucosylceramide synthase (GCS) inhibitor. Ibiglustat succinate 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].

In Vitro

Ibiglustat (SAR402671) succinate (1 μM, 15 days; Fabry disease (FD) cells) is close to the physiological level in untreated WT cells in GL-3 levels, suggesting that Ibiglustat succinate 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.

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REFERENCES

[1]. 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.

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

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

[4]. Itier JM, et al. Effective clearance of GL-3 in a human iPSC-derived cardiomyocyte model of Fabry disease. J Inherit Metab Dis. 2014;37(6):1013-1022.

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