Miglustat hydrochloride

Cat. No.: HY-17020A

CAS No.: 210110-90-0

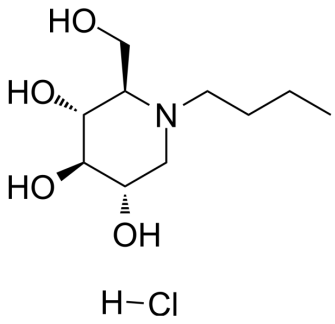
Molecular Formula: $C_{10}H_{22}ClNO_4$

Molecular Weight: 255.74

Target: Glucosylceramide Synthase (GCS)

Pathway: Neuronal Signaling

Storage: -20°C, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 65 mg/mL (254.16 mM; Need ultrasonic)
H_2O : ≥ 34 mg/mL (132.95 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.9102 mL	19.5511 mL	39.1022 mL
5 mM	0.7820 mL	3.9102 mL	7.8204 mL
10 mM	0.3910 mL	1.9551 mL	3.9102 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (391.02 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 3.25 mg/mL (12.71 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 3.25 mg/mL (12.71 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 3.25 mg/mL (12.71 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Miglustat (N-Butyldeoxynojirimycin) hydrochloride is an orally active and reversible ceramide glucosyltransferase inhibitor. Miglustat hydrochloride can be used for the research of type I gaucher disease[1][2].

In Vitro

Miglustat (200 μM; 2, 4 and 24 h) hydrochloride restores F508del-CFTR (cystic fibrosis transmembrane conductance regulator) function in cystic fibrosis (CF) bronchial epithelial IB3-1 and CuFi-1 cells. Miglustat hydrochloride reduces the

	inflammatory response to P. aeruginosa in both CF and non-CF bronchial cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Miglustat (0.2 mg/kg; oral administration; once) hydrochloride is able to rescue synaptic plasticity deficits, to restore ERKs activation and to counteract hyperexcitability[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	NPC1−/− mice[1]
Dosage:	0.2 mg/kg
Administration:	Oral administration; once
Result:	Was able to rescue synaptic plasticity deficits, to restore ERKs activation and to counteract hyperexcitability.

CUSTOMER VALIDATION

- Cell. 2019 Dec 12;179(7):1483-1498.e22.
- Cell Rep. 2022 Jul 5;40(1):111049.
- Preprints. 2023 Dec 20.

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REFERENCES

[1]. Maria Cristina Dechecchi, et al. Anti-inflammatory effect of miglustat in bronchial epithelial cells. J Cyst Fibros. 2008 Nov;7(6):555-65.

[2]. G D'Arcangelo, et al. Miglustat Reverts the Impairment of Synaptic Plasticity in a Mouse Model of NPC Disease. Neural Plast. 2016:2016:3830424.

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