Telaglenastat

Cat. No.: HY-12248

CAS No.: 1439399-58-2

Molecular Formula: $C_{26}H_{24}F_3N_7O_3S$

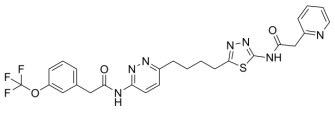
Molecular Weight: 571.57

Target: Autophagy; Glutaminase

Pathway: Autophagy; Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (87.48 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.7496 mL	8.7478 mL	17.4957 mL
5 mM	0.3499 mL	1.7496 mL	3.4991 mL
10 mM	0.1750 mL	0.8748 mL	1.7496 mL

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

In Vivo

1. Add each solvent one by one: 20% HP-β-CD/10 mM citrate pH 2.0
   Solubility: 10 mg/mL (17.50 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 20% SBE-β-CD/10 mM Trisodium citrate adjusted to pH 2.0 with HCL
   Solubility: 5 mg/mL (8.75 mM); Clear solution; Need ultrasonic and adjust pH to 2 with 1M HCl and heat to 55°C
3. Add each solvent one by one: 70% PEG300 >> 30% (20% SBE-β-CD in saline)
   Solubility: 4 mg/mL (7.00 mM); Suspended solution; Need ultrasonic and warming and heat to 55°C

BIOLOGICAL ACTIVITY

Description

Telaglenastat (CB-839) is a first-in-class, selective, reversible and orally active glutaminase 1 (GLS1) inhibitor. Telaglenastat selectively inhibits GLS1 splice variants KGA (kidney-type glutaminase) and GAC (glutaminase C) compared to GLS2. The IC_{50}s are 23 nM and 28 nM for endogenous glutaminase in mouse kidney and brain, respectively. Telaglenastat inuduces autophagy and has antitumor activity[1].

IC_{50} & Target

IC50: 23 nM (GLS1 in kidney), 28 nM (GLS1 in brain), >1 μM (GLS2 in liver)[1]

In Vitro

Telaglenastat (CB-839) (0.1-1000 nM; 72 hours) has antiproliferative activity in HCC1806 and MDA-MB-231 cells with IC_{50}s of 49 nM and 26 nM, respectively[1].

Telaglenastat (CB-839) (1 μM; 72 hours) activates caspase 3/7 and induces apoptosis in MDA-MB-231 and HCC1806 cells[1].

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

Cell Proliferation Assay[1]

Cell Line:	HCC1806, MDA-MB-231 cells
Concentration:	0.1, 1, 10, 100, 1000 nM
Incubation Time:	72 hours
Result:	Has a potent effect on the proliferation of the two TNBC cell lines (IC_{50} of 49 nM and 26 nM for HCC1806 and MDA-MB-231 cells).

Apoptosis Analysis[1]

Cell Line:	MDA-MB-231, HCC1806 cells
Concentration:	1 μM
Incubation Time:	72 hours
Result:	Caspase 3/7 activation.

In Vivo

Telaglenastat (CB-839) (200 mg/kg; p.o.; twice daily for 28 days) has antitumor activity in xenograft models of TNBC[1].

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

Animal Model:	Female nu/nu mice with age 4–6 weeks (TNBC patient-derived xenograft model)[1]
Dosage:	200 mg/kg
Administration:	Oral administration; twice daily for 28 days
Result:	Suppressed tumor growth by 61% relative to vehicle control at the end of study.

CUSTOMER VALIDATION

- Science. 2022 Mar 18;375(6586):1254-1261.
- Gastroenterology. 2024 Jan 24:S0016-5085(24)00064-7.
- Cell Metab. 2023 Jan 3;35(1):200-211.e9.
- Cancer Discov. 2017 Apr;7(4):380-390.
- Mol Cell. 2022 May 19;82(10):1821-1835.e6.

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REFERENCES

[1]. Gross MI, et al. Antitumor activity of the glutaminase inhibitor CB-839 in triple-negative breast cancer. Mol Cancer Ther. 2014 Apr;13(4):890-901.

[2]. Biancur DE, et al. Compensatory metabolic networks in pancreatic cancers upon perturbation of glutaminemetabolism. Nat Commun. 2017 Jul 3;8:15965.

[3]. Zhou WJ, et al. Estrogen inhibits autophagy and promotes growth of endometrial cancer by promoting glutamine metabolism. Cell Commun Signal. 2019 Aug 20;17(1):99.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA