Telaglenastat hydrochloride

Cat. No.: HY-12248A

CAS No.: 1874231-60-3

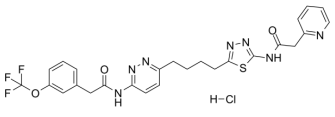
Molecular Formula: $C_{26}H_{25}ClF_3N_7O_3S$

Molecular Weight: 608.04

Target: Glutaminase; Autophagy

Pathway: Metabolic Enzyme/Protease; Autophagy

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Telaglenastat (CB-839) hydrochloride is a first-in-class, selective, reversible and orally active glutaminase 1 (GLS1) inhibitor. Telaglenastat hydrochloride 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 hydrochloride inudces autophagy and has antitumor activity[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

- Cancer Discov. 2017 Apr;7(4):380-390.
- Mol Cell. 2019 Oct 3;76(1):148-162.e7.
- Clin Cancer Res. 2019 Jul 1;25(13):4079-4090.
- Theranostics. 2020 Feb 10;10(8):3488-3502.
- Elife. 2020 Nov 2;9:e60151.

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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.

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