BCH

Cat. No.: HY-108540

CAS No.: 20448-79-7

Molecular Formula: $C_8H_{13}NO_2$

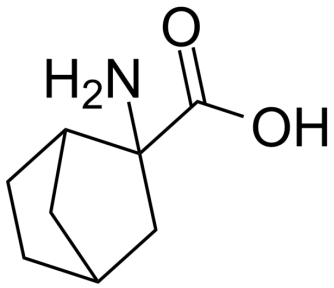
Molecular Weight: 155.19

Target: Apoptosis

Pathway: Apoptosis

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 20 mg/mL (128.87 mM; ultrasonic and warming and heat to 60°C)

DMSO : < 1 mg/mL (insoluble or slightly soluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.4437 mL	32.2186 mL	64.4371 mL
5 mM	1.2887 mL	6.4437 mL	12.8874 mL
10 mM	0.6444 mL	3.2219 mL	6.4437 mL

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

In Vivo

1. Add each solvent one by one: PBS

Solubility: 12.5 mg/mL (80.55 mM); Clear solution; Need ultrasonic and warming and heat to 60°C

BIOLOGICAL ACTIVITY

Description

BCH (2-Amino-2-norbornanecarboxylic acid) is a selective and competitive inhibitor of large neutral amino acid transporter 1 (LAT1) significantly inhibit cellular uptake of amino acids and mTOR phosphorylation, which induces the suppression of cancer growth and apoptosis[1][2][3].

IC_{50} & Target

LAT1[1]

In Vitro

BCH (1-100 mM; 3 days; KYSE30 and KYSE150 esophageal cancer cells) treatment suppresses cell proliferation in a dose-dependent manner[1].

BCH (30 mM; 24 and 48 hours; KYSE30 and KYSE150 cells) treatment significantly increases cell population in the G0/G1 phase in both KYSE30 and KYSE150 cells, indicating that BCH induces cell cycle arrest at G1 phase[1].

BCH (30 mM; 0-24 hours; KYSE30 and KYSE150 cells) treatment decreases phosphorylation of 4E-BP1 and p70S6K at 30 minutes and the decrease is continued for 24 hours. The amount of mTOR, 4E-BP1, and p70S6K proteins is slightly decreased[1].

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

Cell Proliferation Assay[1]

Cell Line:	KYSE30 and KYSE150 esophageal cancer cells
Concentration:	1 mM, 3 mM, 5 mM, 10 mM, 20 mM, 30 mM, 40 mM, 50 mM, or 100 mM
Incubation Time:	3 days
Result:	Cell proliferation was suppressed in a dose-dependent manner.

Cell Cycle Analysis[1]

Cell Line:	KYSE30 and KYSE150 cells
Concentration:	30 mM
Incubation Time:	24 and 48 hours
Result:	Cell cycle arrest.

Western Blot Analysis[1]

Cell Line:	KYSE30 and KYSE150 cells
Concentration:	30 mM
Incubation Time:	0 hour, 0.5 hour, 1 hour, 3 hours, 6 hours, 24 hours
Result:	Phosphorylation of 4E-BP1 and p70S6K was decreased. The amount of mTOR, 4E-BP1, and p70S6K proteins was slightly decreased.

In Vivo

BCH (200 mg/kg; intravenous injection; daily; for 14 days; male BALB/c nude mice) treatment significantly delays tumor growth and decreases glucose metabolism, indicating that LAT1 inhibition potentially suppresses esophageal cancer growth in vivo[1].

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

Animal Model:	Male BALB/c nude mice (5-week-old) with KYSE150 cells[1]
Dosage:	200 mg/kg
Administration:	Intravenous injection; daily; for 14 days
Result:	Significantly delayed tumor growth and decreased glucose metabolism.

CUSTOMER VALIDATION

- Nat Immunol. 2023 Dec;24(12):2042-2052.
- Nat Immunol. 2023 Oct;24(10):1685-1697.
- Front Immunol. 2022 May 19;13:880262.

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REFERENCES

[1]. Ohshima Y, et al. Efficacy of system l amino acid transporter 1 inhibition as a therapeutic target in esophageal squamous cell carcinoma. Cancer Sci. 2016 Oct;107(10):1499-1505.

[2]. Singh N, et al. Discovery of Potent Inhibitors for the Large Neutral Amino Acid Transporter 1 (LAT1) by Structure-Based Methods. Int J Mol Sci. 2018 Dec 21;20(1).

[3]. Wang Q, et al. L-type amino acid transport and cancer: targeting the mTORC1 pathway to inhibit neoplasia. Am J Cancer Res. 2015 Mar 15;5(4):1281-94. eCollection 2015.

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