BPTES

Cat. No.: HY-12683

CAS No.: 314045-39-1

Molecular Formula: $C_{24}H_{24}N_6O_2S_3$

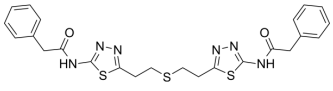
Molecular Weight: 524.68

Target: Glutaminase

Pathway: 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 : 37.5 mg/mL (71.47 mM; ultrasonic and warming and heat to 60°C)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9059 mL	9.5296 mL	19.0592 mL
5 mM	0.3812 mL	1.9059 mL	3.8118 mL
10 mM	0.1906 mL	0.9530 mL	1.9059 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.5 mg/mL (4.76 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.96 mM); Clear solution
3. Add each solvent one by one: 2% DMSO >> 40% PEG300 >> 5% Tween-80 >> 53% saline
   Solubility: 2 mg/mL (3.81 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

BPTES is an allosteric and selective glutaminase inhibitor with an IC_{50} of 0.16 μM.

IC_{50} & Target

Glutaminase[1]

In Vitro

BPTES (10 μM) exhibits inhibition of PDAC cell proliferation[1]. BPTES preferentially slows growth of mutant IDH1 cells without inducing apoptosis. BPTES (10 μM) reduces glutaminase activity in both WT and mutant IDH1 expressing cells,

	diminishes glutamate and α-KG levels, and increases glycolytic intermediates while leaving total 2-HG levels unaffected[2]. BPTES (10 μM) shows a clear synergistic anti-cancer effect with 10 μM of 5-FU in A549 and EKVX cell lines, and results in a growth reduction response not only in EKVX and A549 but also in most of the NSCLC cell lines[3]. BPTES (10 μM) effectively reduces the levels of the metabolites of the TCA cycle, with no changes in the levels of metabolites in glycolysis and the pentose phosphate pathway. BPTES treatment reduces about 30% ATP production under normoxia, and an additional 10% reduction of ATP production is observed under hypoxia in EKVX[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	BPTES-NPs (BPTES nanoparticles, 1.2 mg BPTES in 100 μL nanoparticles, i.v.) significantly attenuates tumor growth in the patient-derived pancreatic orthotopic tumor model[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Cells are plated at a density of 500 cells/well in a 96-well black clear bottom plate. At 24 hrs, media is changed to the appropriate media (DMEM with 4.5 g/L, 1.5 g/L or 0.1 g/L glucose, 10% FBS, and 4 mM glutamine). 48 hours after plating, compounds or DMSO are added. Media and alamarBlue is added to a volume of 200 μL in each well. Fluorescence is measured at 48 hrs or 72 hrs (EGCG) using a Victor3 plate-reader. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Four-week-old female Foxn1nuathymic nude mice are used for the assay. Freshly resected pancreatic tumor samples obtained from patients at the time of surgery are propagated from mouse to mouse as a live tumor bank. Once a tumor volume of 50 mm^3 is reached (4 wk postimplantation), mice are treated with 12.5 mg/kg BPTES by intraperitoneal injection, 200 mg/kg CB-839 twice per d by oral gavage, 54 mg/kg BPTES-NPs (1.2 mg BPTES in 100 μL nanoparticles per mouse) by intravenous injection, blank-NPs (100 μL per mouse) by intravenous injection, 25 mg/kg LY 188011 intraperitoneally, or a combination of BPTES-NPs with LY 188011. BPTES-NPs are injected once every 3 d for a total of six injections over 16 d. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Metab. 2022 Sep 7;S1550-4131(22)00359-X.
- J Hepatol. 2020 May;72(5):909-923.
- Nano Today. 2023 Dec, 53, 102009.
- Theranostics. 2020 May 16;10(14):6483-6499.
- Acta Biomater. 2023 May 28;S1742-7061(23)00303-3.

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REFERENCES

[1]. Elgogary A, et al. Combination therapy with BPTES nanoparticles and targets the metabolic heterogeneity of pancreatic cancer. Proc Natl Acad Sci U S A. 2016 Sep 6;113(36):E5328-36.

[2]. Meghan J. Seltzer, et al. Inhibition of glutaminase preferentially slows growth of glioma cells with mutant IDH1. Cancer Res. 2010 Nov 15; 70(22): 8981-8987.

[3]. Lee JS, et al. Glutaminase 1 inhibition reduces thymidine synthesis in NSCLC. Biochem Biophys Res Commun. 2016 Aug 26;477(3):374-82

[4]. Lee JS, et al. Dual targeting of glutaminase 1 and thymidylate synthase elicits death synergistically in NSCLC. Cell Death Dis. 2016 Dec 8;7(12):e2511.

Caution: Product has not been fully validated for medical applications. For research use only.

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