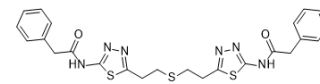


BPTES

Cat. No.:	HY-12683		
CAS No.:	314045-39-1		
Molecular Formula:	C ₂₄ H ₂₄ N ₆ O ₂ S 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	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (95.30 mM; Need ultrasonic)
 H₂O : ≥ 0.1 mg/mL (0.19 mM)
 * "≥" means soluble, but saturation unknown.

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 >> 90% corn oil
 Solubility: ≥ 2.08 mg/mL (3.96 mM); Clear solution

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 EK VX cell lines, and results in a growth reduction response not only in EK VX 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 μ L 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 μ L 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 mm³ 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 μ L nanoparticles per mouse) by intravenous injection, blank-NPs (100 μ L 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

- J Hepatol. 2020 May;72(5):909-923.
- Theranostics. 2020 May 16;10(14):6483-6499.
- Int Immunopharmacol. 2020 Jun 7;85:106664.
- Mol Hum Reprod. 2020 Jan 16. pii: gaaa004.

See more customer validations on www.MedChemExpress.com

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.

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