Carglumic Acid

Cat. No.: HY-B0711

CAS No.: 1188-38-1

Molecular Formula: $C_6H_{10}N_2O_5$

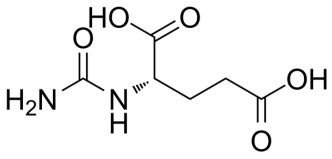
Molecular Weight: 190.15

Target: Others

Pathway: Others

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (525.90 mM; Need ultrasonic)

H_2O : 10 mg/mL (52.59 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	5.2590 mL	26.2950 mL	52.5901 mL
	5 mM	1.0518 mL	5.2590 mL	10.5180 mL
	10 mM	0.5259 mL	2.6295 mL	5.2590 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 37.5 mg/mL (197.21 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (13.15 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (13.15 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (13.15 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Carglumic acid (N-Carbamyl-L-glutamic acid), a functional analogue of N-acetylglutamate (NAG) and a carbamoyl phosphate synthetase 1 (CPS1) activator, is used to treat acute and chronic hyperammonemia associated with NAG synthase (NAGS) deficiency.

IC_{50} & Target	CPS1[1]
In Vitro	Carglumic acid suppresses cell viability in the pancreatic ductal adenocarcinoma cell lines, triple-negative breast cancer cell lines, hepatoma cell lines, and human non-small cell lung carcinoma cell lines in a dose-dependent manner. The 50% inhibitory concentration (IC_{50}) of Carglumic acid against those cell lines is between 5 and 7.5 mM. The results show that Carglumic acid does not induce complete cell cycle arrest. Instead, there are more sub-G1 cells among Carglumic acid-treated AsPC1 and MDA-MB-231 cells than among untreated cells. In AsPC1 and HPDE-E6E7 cells, the IC_{50}s of Carglumic acid are 5 mM and over 10 mM, respectively . In MDA-MB-231 and MCF-12A cells, the IC_{50}s of Carglumic acid are 5 mM and 6 mM, respectively[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	The results show that Carglumic acid, but not the vehicle control, markedly inhibits tumor growth. In the orthotopic pancreatic cancer model, tumor growth inhibition by Carglumic acid on day 21 is 80% ($P<0.01$). In the orthotopic triple-negative breast cancer model, tumor growth inhibition by Carglumic acid on day 20 is 82% ($P<0.01$). These results indicate that Carglumic acid suppresses tumor growth in pancreatic cancer and triple-negative breast cancer. On day 20, mean tumor growth inhibition in orally and intravenously treated mice is 55% and 93%, respectively, relative to untreated mice ($P<0.01$)[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	Caspase activity is measured by using a fluorimetric caspase-3 assay kit. In brief, cells that are treated with Carglumic Acid or that are left untreated are lysed in a lysis buffer, and 50 μg of protein lysate is incubated with Ac-DEVD-AMC substrate in the assay buffer for 1 h. The resultant fluorescence signals are read by using a fluorometer (excitation 360 nm, emission 460 nm), and the results are tabulated as fold changes relative to the untreated control cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [1]	Cell viability is evaluated by MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) assay. In brief, various cancer cell lines are seeded (1×10^4 cells/well) in a 96-well plate and treated with different doses of Carglumic Acid. After 48 h, 50 μL of MTT solution per well (stock solution concentration 5 mg/mL) is added to each well, and the cells are incubated for 2 h more, followed by addition of 100 μL of dimethyl sulfoxide to each well. Absorbance at 570 nm is measured immediately using a multiwell scanner[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	For orthotopic cancer models, AsPC1/luc human pancreatic cancer cells (1×10^6) are injected into the pancreas of nude mice or MDA-MB-231 human triple-negative breast cancer cells (3×10^6) are injected into the mammary fat pad of nude mice. Carglumic acid is administered to mice 5 days after tumor inoculation in the pancreatic cancer model and 7 days after tumor inoculation in the triple-negative breast cancer model. Tumor-bearing mice receive a Carglumic acid dose of 120 mg/kg orally every day for 10 days, 60 mg/kg orally three times per week for 2 weeks, or 60 mg/kg intravenously three times per week for 2 weeks. Tumor volume is determined by measuring luciferase signals using the in vivo imaging system in the pancreatic cancer model[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Chen CT, et al. Carglumic acid promotes apoptosis and suppresses cancer cell proliferation in vitro and in vivo. Am J Cancer Res. 2015 Nov 15;5(12):3560-9.

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