Sparfosic acid trisodium

Cat. No.: HY-112732B

CAS No.: 70962-66-2

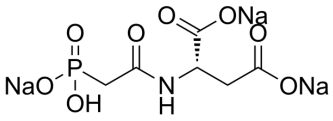
Molecular Formula: $C_6H_7NNa_3O_8P$

Molecular Weight: 321.06

Target: Apoptosis

Pathway: Apoptosis

Storage: -80°C, protect from light, stored under nitrogen



SOLVENT & SOLUBILITY

In Vitro

H_2O : 250 mg/mL (778.67 mM; Need ultrasonic)
DMSO : 180 mg/mL (560.64 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.1147 mL	15.5734 mL	31.1468 mL
5 mM	0.6229 mL	3.1147 mL	6.2294 mL
10 mM	0.3115 mL	1.5573 mL	3.1147 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (311.47 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 4.5 mg/mL (14.02 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 4.5 mg/mL (14.02 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 4.5 mg/mL (14.02 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Sparfosic acid trisodium is a DNA antimetabolite agent and a potent inhibitor of aspartate transcarbamoyl transferase. Aspartate transcarbamoyl transferase catalyzes the second step of de novo pyrimidine biosynthesis. Sparfosic acid trisodium synergistically enhances the cytotoxicity of a combination of 5-fluorouracil (5-FU) and interferon-alpha (IFN) against human colon cancer cell lines[1][2][3].

In Vitro

Sparfosic acid trisodium (N-(Phosphonacetyl)-L-aspartate, PALA) treatment causes apoptosis in the resistant Br1 cells[1].

?Sparfosic acid trisodium (PALA, 300 μM) shows progressive accumulation of cells in S phase and activation of an apoptotic pathway leading to cell death[1].

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

Cell Cycle Analysis[1]

Cell Line:	Br-l and L-2 cell lines established from metastasis in nude mouse injected with the human tumor cell line MDA-MB-435.
Concentration:	300 μM.
Incubation Time:	12, 24 and 48 h.
Result:	Cells were predominantly in S phase in both the cell lines, although slightly higher proportion of cells in S phase were noted in L-2 than Brl-3prl cells.

Western Blot Analysis[1]

Cell Line:	Br-l and L-2 cell lines.
Concentration:	300 μM.
Incubation Time:	4, 10 and 24 h.
Result:	There was moderate difference in the level of phosphorylated Rb proteins seen in the two cell types. Marked increase in the amount of cyclin A protein was detected in the L-2 cells undergoing apoptosis with the highest level detected at 10 h post-drug treatment. In contrast, there was no increase in the level of cyclin A seen in the Brl-3prl cells. Cyclin E protein was found elevated in the L-2 cells and Brl-3prl cells compared to their respective controls.

In Vivo

Sparfosic acid trisodium (490 mg/kg; i.p.; on days 1, 5, and 9; mice bearing B16 melanoma) shows the life-span is increased survives 77 to 86% longer than controls. Lewis lung carcinoma is highly sensitive to Sparfosic acid trisodium. Treatment on days 1, 5, and 9 following s.c. implantation of Lewis lung carcinoma is curative to 50% of the mice[4].

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

REFERENCES

[1]. Wang J, et al. Elevated cyclin A associated kinase activity promotes sensitivity of metastatic human cancer cells to DNA antimetabolite drug. Int J Oncol. 2015 Aug;47(2):782-90.

[2]. Angela D. Morris, et al. A New, Efficient, Two Step Procedure for the Preparation of the Antineoplastic Agent Sparfosic Acid

[3]. Johnson RK, et al. Antitumor activity of N-(phosphonacetyl)-L-aspartic acid, a transition-state inhibitor of aspartate transcarbamylase. Cancer Res. 1976;36(8):2720-2725.

[4]. Wadler S, et al. Phase II trial of N-(phosphonacetyl)-L-aspartate (PALA), 5-fluorouracil and recombinant interferon-alpha-2b in patients with advanced gastric carcinoma. Eur J Cancer. 1996;32A(7):1254-1256.

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