Cyclophosphamide

Cat. No.: HY-17420

CAS No.: 50-18-0

Molecular Formula: $C_7H_{15}Cl_2N_2O_2P$

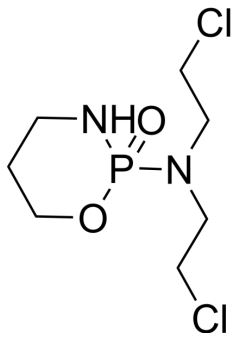
Molecular Weight: 261.09

Target: DNA Alkylator/Crosslinker

Pathway: Cell Cycle/DNA Damage

Storage: 4°C, protect from light

* The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 38 mg/mL (145.54 mM)

H_2O : 33.33 mg/mL (127.66 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.8301 mL	19.1505 mL	38.3010 mL
	5 mM	0.7660 mL	3.8301 mL	7.6602 mL
	10 mM	0.3830 mL	1.9150 mL	3.8301 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 25 mg/mL (95.75 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (9.58 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.58 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.58 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cyclophosphamide is a synthetic alkylating agent chemically related to the nitrogen mustards with antineoplastic activity, a immunosuppressant.

IC_{50} & Target

DNA Alkylator[1]

In Vitro

Cyclophosphamide induces outer membrane blebbing, leads to DNA fragmentation, as revealed by TUNEL staining of free 3'-OH DNA ends, and induces cleavage of the caspase 3 and caspase 7 substrate PARP in 9L/P450 cells. Bcl-2 expression fully blocks the activation of both initiator caspases as well as the effector caspase 3 in cells treated with activated Cyclophosphamide. Bcl-2 inhibits the cytotoxic effects but not the cytostatic effects of activated Cyclophosphamide[1]. Cyclophosphamide inhibits the AChE reversibly with an IC_{50} of 511 μM[2]. Carbon tetrachloride does not affect the direct cytotoxicity of cyclophosphamide or 4-hydroxycyclophosphamide to cells in culture[3].

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

In Vivo

Cyclophosphamide can be used in animal modeling to establish bone marrow suppression model.

Cyclophosphamide (injected i.p.;2mg/mouse in 0.1 mL PBS, in C3H mice bearing SW1 tumors) increases the percentage of cells that stained for CD3, CD4 or CD8 in both spleens and tumors[4].

Cyclophosphamide (CP) is a prodrug that is enzymatically converted to the cytotoxic 4-hydroxycyclophosphamide (4OHCP) by hepatic enzymes, the cytochrome P450. Cyclophosphamide has vast interspecies and intraspecies variations in kinetics. Dog microsomes were 55-fold more efficient than human microsomes, 2.8-fold more efficient than cat microsomes, and 1.2-fold more efficient than mouse microsomes at catalyzing CP bioactivation[5].

1. Induction of Ovarian Failure Model[6]

- Background

  Cyclophosphamide (Cy) induces ovarian insufficiency (POI) via causes primordial follicle activation.

- Specific Mmodeling Methods

  Mice: Balb/C • female • 5-week-old

  Administration: 150 mg/kg • ip • single dose.

- Modeling Indicators

  Decreased number of primary follicles in the ovary

2. Induction of Myelosuppression and Immunosuppression[7]

- Background

  Cyclophosphamide induces myelosuppressi.on via interferes with the proliferation and differentiation of bone marrow (BM) cells.

- Specific Mmodeling Methods

Mice: Swiss • male • 6-week-old

Administration: 150 mg/kg • ip • single dose.

- Modeling Indicators

  Induced important changes in BM tissue structure, reduces the myeloid/erythroid ratio, and decreases the number of blood leukocytes.

- Opposite Product(s): Ginsenoside Rg1 (HY-N0045)

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

Animal Model:	Six to eight-week old female C3H/HeN mice bearing SW1 tumors[4]
Dosage:	2 mg/mouse
Administration:	Injected i.p.; 2mg/mouse in 0.1 mL PBS; 4 days
Result:	Increased the percentage of cells that stained for CD3, CD4 or CD8 in both spleens and tumors.

CUSTOMER VALIDATION

- Nat Commun. 2023 Apr 13;14(1):2109.
- Nat Commun. 2021 Jan 4;12(1):20.
- J Clin Invest. 2024 Mar 7:e172716.
- Cell Death Dis. 2020 Nov 12;11(11):976.
- Life Sci. 2020 Aug 1;254:117590.

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REFERENCES

[1]. Dominique A Ramirez, et al. Kinetics of Cyclophosphamide Metabolism in Humans, Dogs, Cats, and Mice and Relationship to Cytotoxic Activity and Pharmacokinetics. Drug Metab Dispos. 2019, 47, 3.

[2]. H. Roness, et al. Pharmacological administration of recombinant human AMH rescues ovarian reserve and preserves fertility in a mouse model of chemotherapy, without interfering with anti-tumoural effects. J Assist Reprod Genet. 2019, 36, 9.

[3]. Susana Salva, et al. Probiotic Lactobacillus strains protect against myelosuppression and immunosuppression in cyclophosphamide-treated mice. Int Immunopharmacol. 2014, 22, 1.

[4]. Schwartz PS, et al. Cyclophosphamide induces caspase 9-dependent apoptosis in 9L tumor cells. Mol Pharmacol. 2001 Dec;60(6):1268-1279.

[5]. al-Jafari AA, et al. Inhibition of human acetylcholinesterase by cyclophosphamide. Toxicology. 1995 Jan 19;96(1):1-6.

[6]. Harris RN, et al. Carbon tetrachloride-induced increase in the antitumor activity of cyclophosphamide in mice: a pharmacokineticstudy. Cancer Chemother Pharmacol. 1984;12(3):167-72.

[7]. Liu P, et al. Administration of cyclophosphamide changes the immune profile of tumor-bearing mice. J Immunother. 2010 Jan;33(1):53-9.

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