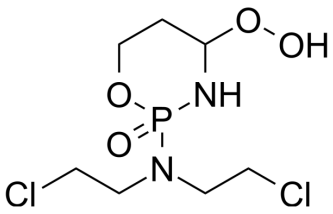
4-Hydroperoxy cyclophosphamide

Cat. No.:	HY-117433
CAS No.:	39800-16-3
Molecular Formula:	$C_7H_{15}Cl_2N_2O_4P$
Molecular Weight:	293.08
Target:	Drug Metabolite; Apoptosis; Reactive Oxygen Species; DNA Alkylator/Crosslinker
Pathway:	Metabolic Enzyme/Protease; Apoptosis; Immunology/Inflammation; NF-κB; Cell Cycle/DNA Damage
Storage:	-20°C, stored under nitrogen * In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (170.60 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4120 mL	17.0602 mL	34.1204 mL
	5 mM	0.6824 mL	3.4120 mL	6.8241 mL
	10 mM	0.3412 mL	1.7060 mL	3.4120 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 (8.53 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.53 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.53 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

4-Hydroperoxy cyclophosphamide is the active metabolite form of the precursor Cyclophosphamide. 4-Hydroperoxy cyclophosphamide cross-links DNA to induce T cell apoptosis independent of caspase receptor activation, and can activate the mitochondrial death pathway by producing reactive oxygen species (ROS). 4-Hydroperoxy cyclophosphamide can be used in the study of rheumatoid arthritis and autoimmune diseases[1][2].

In Vitro

4-Hydroperoxy cyclophosphamide (1 or 3 μg/mL, 24, 48, 72 h) Apoptosis of Caspase-independent T cells is mediated by oxidative stress-induced mitochondrial apoptosis factor AIF and nuclear relocalization of EndoG[1].
4-Hydroperoxy cyclophosphamide (1 μg/mL, 72, 96 h) Combines with methotrexate (HY-14519), RANKL expression was

inhibited in IL-6/ SIL-6R-induced fibroblast-like synoviocytes by inhibiting the JAK2/STAT3 and p38MAPK signaling pathways [2].

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

Western Blot Analysis[1]

Cell Line:	CTL
Concentration:	3 μg/mL
Incubation Time:	24, 48, 72 h
Result:	Increased the expression of p53 and Bax after 24 h.

Apoptosis Analysis[1]

Cell Line:	Jurkat, CTL
Concentration:	1 or 3 μg/mL
Incubation Time:	24 , 48 , 72h
Result:	Caspase-8 deficiency prevented CD95-mediated death induced by anti-CD95 monoclonal antibody (mAb) APO-1. Overexpression of Bcl-2 inhibited apoptosis. Induced cell death proceeded independently of caspase inhibition.

In Vivo

4-Hydroperoxy cyclophosphamide (200 mg/kg intraperitoneally injected) kills Caspase-independent T cells and B cells in mice[1].

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

Animal Model:	BALB/c mice[1]
Dosage:	200 mg/kg
Administration:	i.p.
Result:	Exhibited a marked depletion of immature double-positive CD4 þ CD8 þ thymocytes and mature single-positive $CD4^+$ and $CD8^+$ T cells.

CUSTOMER VALIDATION

- Eng Comput. 2023 Aug 7.
- Cell Death Discov. 2023 Nov 14;9(1):413.
- Int J Cancer. 2024 Jul 15;155(2):324-338.
- J Ethnopharmacol. 2024 Jun 4:118405.
- AfricArXiv Preprints. 2212.

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REFERENCES

[1]. Niu HQ, et al. Combination of 4-hydroperoxy cyclophosphamide and methotrexate inhibits IL-6/sIL-6R-induced RANKL expression in fibroblast-like synoviocytes via suppression of the JAK2/STAT3 and p38MAPK signaling pathway. Int Immunopharmacol. 2018 Aug;61:45-53.

[2]. Strauss G, et al. 4-hydroperoxy-cyclophosphamide mediates caspase-independent T-cell apoptosis involving oxidative stress-induced nuclear relocation of mitochondrial apoptogenic factors AIF and EndoG. Cell Death Differ. 2008 Feb;15(2):332-43.

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