Cambinol

Cat. No.: HY-100732

CAS No.: 14513-15-6

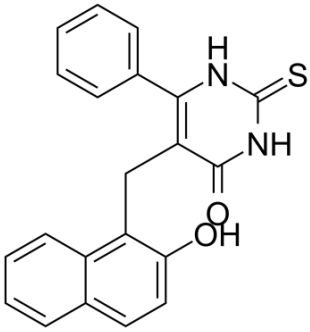
Molecular Formula: $C_{21}H_{16}N_2O_2S$

Molecular Weight: 360.43

Target: Sirtuin; Apoptosis; Phospholipase

Pathway: Cell Cycle/DNA Damage; Epigenetics; Apoptosis; Metabolic Enzyme/Protease

Storage: -20°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (138.72 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.7745 mL	13.8723 mL	27.7446 mL
	5 mM	0.5549 mL	2.7745 mL	5.5489 mL
	10 mM	0.2774 mL	1.3872 mL	2.7745 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: ≥ 2.08 mg/mL (5.77 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.77 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cambinol is a SIRT1 and SIRT2 inhibitor with IC_{50} values of 56 μM and 59 μM, respectively. Cambinol is a potent brain penetrant neutral sphingomyelinase (N-SMase) inhibitor (exosome inhibitor)[1][2].

IC_{50} & Target

SIRT1	SIRT2
56 μM (IC_{50})	59 μM (IC_{50})

In Vitro

Cambinol inhibits NAD-dependent deacetylase activity of human SIRT1 and SIRT2. Inhibition of SIRT1 activity with cambinol during genotoxic stress leads to hyperacetylation of key stress response proteins and promotes cell cycle arrest. Treatment of BCL6-expressing Burkitt lymphoma cells with cambinol as a single agent induces apoptosis, which is accompanied by hyperacetylation of BCL6 and p53. Cambinol has only weak inhibitory activity against SIRT5 (42% inhibition at 300 μM) and no activity against SIRT3[1].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Cambinol is well tolerated in mice (100 mg/kg) and inhibits growth of Burkitt lymphoma xenografts. No significant weight loss occurs in cambinol-treated animals relative to controls. Inhibitors of NAD-dependent deacetylases may constitute novel anticancer agents[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	.The reporter construct with or without varying amounts of GAL4-BCL6 expression plasmid are introduced into NCI-H460 cells using calcium phosphate method. A plasmid containing cytomegalovirus (CMV)-driven β-galactosidase reporter (50 ng) is cotransfected to control for transfection efficiency. Sixteen hours after transfection, cells are treated with 100 μM cambinol of DMSO (control) for 24 hours and the luciferase and β-galactosidase activity is measured[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Mice: Cambinolat the dose of 100 mg/kg, or vehicle are administered i.v. through tail vein injection or i.p. daily from day 5 to 19 (five injections per week). The dose of 100 mg/kg cambinol is the highest dose that could be administered as a single i.v. injection due to limited solubility of the drug. Tumor size is measured thrice a week using caliper and the tumor volumes are calculated[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Am J Transl Res. 2019 Jul 15;11(7):3992-4009.

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REFERENCES

[1]. Heltweg B, et al. Antitumor activity of a small-molecule inhibitor of human silent information regulator 2 enzymes. Cancer Res. 2006 Apr 15;66(8):4368-77.

[2]. Huarui Zhang, et al. Advances in the discovery of exosome inhibitors in cancer. J Enzyme Inhib Med Chem. 2020 Dec;35(1):1322-1330.

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