C8-Ceramide

Cat. No.: HY-108391

CAS No.: 74713-59-0

Molecular Formula: $C_{26}H_{51}NO_3$

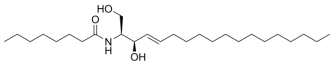
Molecular Weight: 425.69

Target: Apoptosis; PKC; Autophagy

Pathway: Apoptosis; Epigenetics; TGF-beta/Smad; Autophagy

Storage: -20°C, stored under nitrogen

* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (234.91 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3491 mL	11.7456 mL	23.4913 mL
5 mM	0.4698 mL	2.3491 mL	4.6983 mL
10 mM	0.2349 mL	1.1746 mL	2.3491 mL

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

BIOLOGICAL ACTIVITY

Description

C8-Ceramide (N-Octanoyl-D-erythro-sphingosine) is a cell-permeable analog of naturally occurring ceramides. C8-Ceramide has anti-proliferation properties and acts as a potent chemotherapeutic agent. C8-Ceramide stimulates dendritic cells to promote T cell responses upon virus infections. C8-Ceramide induces slight activation of protein kinase (PKC) in vitro[1][2][3][4].

IC_{50} & Target			
	PKC	apoptosis	autophagy

In Vitro

C8-ceramide (3 μM; 48 hours) irreversibly reduces tumor-cell proliferation and induces morphological changes[1].

C8-ceramide can induce necrosis-like cell death, but does not induce caspase-dependent cleavage of PARP (biochemical marker of apoptosis) in human cervical tumor cells[1].

C8-ceramide may increase the endogenous ROS level (10-30 μM; 24 hours) by regulating the switch of SOD1 and SOD2, causing the anti-proliferation (10-50 μM; 24 hours), and consequently triggering the apoptosis (10-50 μM; 48 hours) of NSCLC H1299 cells[2].

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

Cell Viability Assay[1]

Cell Line:	CALO cells, INBL cells, HeLa cells
Concentration:	3 μM
Incubation Time:	48 hours
Result:	Markedly reduced the tumor cell number.

Cell Proliferation Assay[2]

Cell Line:	H1299 cells
Concentration:	10 μM, 20 μM, 30 μM, 40 μM, 50 μM
Incubation Time:	24 hours
Result:	Decreased the rate of cellular proliferation in a dose-dependent manner, with an IC_{50} of 22.9 μM.

Cell Cycle Analysis[2]

Cell Line:	H1299 cells
Concentration:	10 μM, 20 μM, 30 μM, 40 μM, 50 μM
Incubation Time:	24 hours
Result:	Caused the G1 arrest.

Apoptosis Analysis[2]

Cell Line:	H1299 cells
Concentration:	10 μM, 20 μM, 30 μM
Incubation Time:	24 hours, 48 hours
Result:	Increased the level of cleaved caspase-3.

In Vivo

C8-ceramide (0.1 mg/kg; intranasal administration) induces more robust $CD8^+$ and $CD4^+$ T cell responses to viral infections in virus infected mice[3].

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

Animal Model:	C57BL/6 mice, with lymphocytic choriomeningitis virus infected[3]
Dosage:	0.1 mg/kg
Administration:	Intranasal administration
Result:	Increased the $CD8^+$ T cell response to influenza in the lungs.

REFERENCES

[1]. Rebeca López-Marure , et al. Ceramide promotes the death of human cervical tumor cells in the absence of biochemical and morphological markers of apoptosis. Biochem Biophys Res Commun. 2002 May 10;293(3):1028-36.

[2]. Curtis J. Pritzl, et al. A ceramide analogue stimulates dendritic cells to promote T cell responses upon virus infections. J Immunol. 2015 May 1; 194(9): 4339-4349.

[3]. Yuli C. Chang, et al. Exogenous C8-Ceramide Induces Apoptosis by Overproduction of ROS and the Switch of Superoxide Dismutases SOD1 to SOD2 in Human Lung Cancer Cells. Int J Mol Sci. 2018 Oct; 19(10): 3010.

[4]. H W Huang, et al. Ceramides modulate protein kinase C activity and perturb the structure of Phosphatidylcholine/Phosphatidylserine bilayers. Biophys J. 1999 Sep; 77(3): 1489-1497.

[5]. Lan Weiss, et al. Ceramide contributes to pathogenesis and may be targeted for therapy in VCP inclusion body myopathy. Hum Mol Genet. 2021 Jan 7;ddaa248.

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