Eldecalcitol

Cat. No.: HY-A0020

CAS No.: 104121-92-8

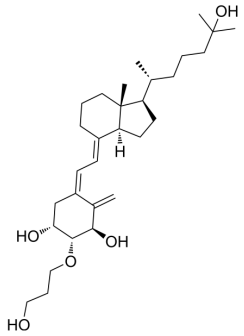
Molecular Formula: $C_{30}H_{50}O_5$

Molecular Weight: 490.72

Target: VD/VDR; Apoptosis

Pathway: Vitamin D Related/Nuclear Receptor; Apoptosis

Storage: 4°C, protect from light, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light, stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

Methanol : 8.33 mg/mL (16.98 mM; Need ultrasonic)
DMSO : 3.33 mg/mL (6.79 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0378 mL	10.1891 mL	20.3782 mL
5 mM	0.4076 mL	2.0378 mL	4.0756 mL
10 mM	0.2038 mL	1.0189 mL	2.0378 mL

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

BIOLOGICAL ACTIVITY

Description

Eldecalcitol (ED-71) is an orally active vitamin D3 analogue, inhibits bone resorption and increases bone mineral density. Eldecalcitol (ED-71) displays anti-tumor effect and inhibits cell proliferation, migration and induces apoptosis by suppressing GPx-1[1][2][3][4].

In Vitro

Eldecalcitol (0-50 nM; 24 h) displays no cytotoxicity, and (0.5-50 nM; 24 h) reduces cell death induced by LPS (5 μg/mL)[2].
Eldecalcitol (5 nM; 24 h) inhibits the LPS-induced pyroptosis by activating Nrf2 and its effector molecule HO-1[2].
Eldecalcitol (0.5-50 nM; 24 h) exhibits anti-pyroptotic ability, and decreases NLRP3, caspase-1, and IL-1β expression dose-dependently[3].
Eldecalcitol (0.04-40 nM; 0-48 h) inhibits the proliferation and migration of SCC-15 and CAL-27 cells[3].
Eldecalcitol (0.4 nM; 48 h) arrests cell cycle at G0/G1 phase and induces apoptosis by suppressing the expression of GPx-1 (glutathione peroxidase) in OSCC cells[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	Human gingival fibroblasts (HGFs)
Concentration:	0, 0.5, 5, and 50 nM
Incubation Time:	24 hours
Result:	Decreased TLR4, NLRP3, caspase-1 p20, ASC, and GSDMD-N level in a dose-dependent manner compared with the group treated with LPS. Reduced the release of IL-1β and IL-18 induced by LPS to normal levels.

Cell Proliferation Assay[3]

Cell Line:	SCC-15 and CAL-27 cells
Concentration:	0, 0.04, 0.4, 4, and 40 nM
Incubation Time:	6, 8, 12, 24, 48 hours
Result:	Inhibited the cell viability of the OSCC cells to reach 50% at 24 h with 0.4 nM.

Cell Proliferation Assay[3]

Cell Line:	OSCC cells
Concentration:	0.4 nM
Incubation Time:	48 hours
Result:	Increased the proportion of cells at the late phases of apoptosis from 7.1% to 16.1%. Upregulated Bax and caspase-3, downregulated Bcl-2. Significantly triggered apoptosis in SCC- 15 and CAL- 27 cells.

In Vivo

Eldecalcitol (0.5 μg/kg; p.o.; twice a week for 4 weeks) displays anti-cancer effect by GPX-1 (glutathione peroxidase) inhibition[3].

Eldecalcitol (10, 30, or 90 ng/kg; p.o.; 5-times per week for 12 weeks), as a more potent vitamin D3 analog, stimulates focal bone formation (minimodeling) and suppresses bone resorption more strongly than does calcitriol[4].

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

Animal Model:	Xenograft tumor model in mice (male athymic nude BALB/c mice)[3]
Dosage:	0.5 μg/kg
Administration:	Oral gavage; twice a week for 4 weeks
Result:	Reduced the growth rate of tumors, and downregulated the expression levels of PCNA and MMP- 2 and upregulated the expression of Bax in the tumors. Resulted in decrease of proliferation, the inhibition of migration, and the promotion of apoptosis.

Animal Model:	Ovariectomized (OVX) rat model[4]
Dosage:	10, 30, or 90 ng/kg
Administration:	Oral gavage; 5-times per week for 12 weeks

Result:	Increased the lumbar and femoral BMD in a dose dependent manner. Stimulated focal bone formation that started without prior bone resorption, a process known as bone minimodeling.

REFERENCES

[1]. Matsumoto T.Osteoporosis Treatment by a New Active Vitamin D3 Compound, Eldecalcitol, in Japan.Curr Osteoporos Rep. 2012 Aug 24.

[2]. Huang C, et al. Eldecalcitol Inhibits LPS-Induced NLRP3 Inflammasome-Dependent Pyroptosis in Human Gingival Fibroblasts by Activating the Nrf2/HO-1 Signaling Pathway. Drug Des Devel Ther. 2020 Nov 13;14:4901-4913.

[3]. Lu Y, et al. Eldecalcitol inhibits the progression of oral cancer by suppressing the expression of GPx-1. Oral Dis. 2021 Aug 24.

[4]. Saito H, et al. Eldecalcitol and calcitriol stimulates 'bone minimodeling,' focal bone formation without prior bone resorption, in rat trabecular bone. J Steroid Biochem Mol Biol. 2013 Jul;136:178-82.

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