Vitamin D3

Cat. No.: HY-15398

CAS No.: 67-97-0

Molecular Formula: $C_{27}H_{44}O$

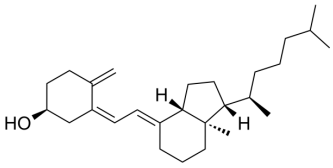
Molecular Weight: 384.64

Target: VD/VDR; Endogenous Metabolite; Bacterial

Pathway: Vitamin D Related/Nuclear Receptor; Metabolic Enzyme/Protease; Anti-infection

Storage: 4°C, protect from light, stored under nitrogen

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 10 mg/mL (26.00 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5998 mL	12.9992 mL	25.9983 mL
5 mM	0.5200 mL	2.5998 mL	5.1997 mL
10 mM	0.2600 mL	1.2999 mL	2.5998 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (5.41 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1 mg/mL (2.60 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1 mg/mL (2.60 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Vitamin D3 (Cholecalciferol; Colecalciferol) is a naturally occuring form of vitamin D. Vitamin D3 induces cell differentiation and prevents proliferation of cancer cells.

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

Vitamin D3 is an inactive vitamin D molecule in vivo. Vitamin D3 undergoes two hydroxylation processes to activate it. Vitamin D3 is first hydroxylated in the liver to form the circulating prohormone 25-hydroxy vitamin D3 [25(OH)D3] by the enzyme 25-hydroxylase (CYP27A1) and probably also by other enzymes (e.g., CYP2R1)[1].
The second hydroxylation occurs in the kidneys via the enzyme 1-alpha-hydroxylase, yielding 1,25- dihydroxycholecalciferol

(calcitriol), which is the biologically active form of vitamin D[1].
Vitamin D3 (2-10 μM; 24-48 hours) exhibits anti-proliferative effects in a dose- and time-dependent manner. Maximal reduction of viability post-treatment of 62% (IK), 52% (RL-95-2), and 55% (Hec-1A) occurs by 72 h of treatment with 10 μM Vitamin D3. but 24-hour exposure lacks significant reduction in viable cells[2].
Cholecalciferol (10 μM; 24-48 hours) shows marked increases in nuclear VDR staining and produces local VDR activation in IK cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	EC cell lines from EEC, Ishikawa 3-H-12(IK), RL-95/2, and HEC-1-A cells
Concentration:	2-10 μM
Incubation Time:	24-72 hours
Result:	Reduced viability in response to VD3 in a dose- and time-dependent manner. Indicated that the conversion of VD3 to 25(OH)D is an essential step for the reduced cell viability effect.

Cell Viability Assay[2]

Cell Line:	EC cell lines from EEC, Ishikawa 3-H-12(IK) cells
Concentration:	10 μM
Incubation Time:	24-48 hours
Result:	Improved nuclear VDR content in IK cells.

In Vivo

Cholecalciferol (oral gavage; 5 mg/kg; 7 days) potentiates the CCl4 toxicity only in the liver, as indicated by plasma levels of ALT and AST, biochemical markers of hepatic damage. It significantly increases renal calcium levels in mice, but renal calcium content does not differ significantly between mice[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male ddY mice on CCl4 toxicity[3]
Dosage:	5 mg/kg
Administration:	Oral gavage; 5 mg/kg; 7 days
Result:	Potentiated CCl4-induced hepatotoxicity and enhanced mouse mortality, without increasing renal toxicity and generation of liver fibrosis.

CUSTOMER VALIDATION

- Int J Oral Sci. 2022 Aug 1;14(1):39.
- Nat Chem Biol. 2022 Aug 18.
- Theranostics. 2024 Jan 1;14(1):436-450.
- Small Methods. 16 December 2021.
- Proc Natl Acad Sci U S A. 2022 Apr 12;119(15):e2117004119.

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REFERENCES

[1]. Nazik Al-Hashimi, et al. Cholecalciferol

[2]. Laura Bergadà, et al. Role of local bioactivation of vitamin D by CYP27A1 and CYP2R1 in the control of cell growth in normal endometrium and endometrial carcinoma. Lab Invest. 2014 Jun;94(6):608-22

[3]. Hiroki Yoshioka, et al. Vitamin D3-induced hypercalcemia increases carbon tetrachloride-induced hepatotoxicity through elevated oxidative stress in mice. PLoS One. 2017 Apr 27;12(4):e0176524.

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