DMHCA

Cat. No.: HY-129098

CAS No.: 79066-03-8

Molecular Formula: $C_{26}H_{43}NO_2$

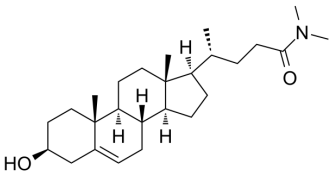
Molecular Weight: 401.63

Target: LXR

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

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 7.69 mg/mL (19.15 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4899 mL	12.4493 mL	24.8985 mL
	5 mM	0.4980 mL	2.4899 mL	4.9797 mL
	10 mM	0.2490 mL	1.2449 mL	2.4899 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: ≥ 0.77 mg/mL (1.92 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.77 mg/mL (1.92 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

DMHCA, a potent and selective LXR agonist, specifically activates the cholesterol efflux arm of the LXR pathway without stimulating triglyceride synthesis. DMHCA has anti-inflammatory effects and can be used for the research of cholesterol homeostasis diabetes[1].

IC_{50} & Target

IC50: LXR[1]

In Vitro

In ex vivo treatment of diabetic $CD34^+$ cells, DMHCA (10 μM; 16-18 hours) restores the fluidity of the membranes to baseline nondiabetic levels[1].
DMHCA restores the levels of BM-derived TNF-α and IL-3 to baseline and significantly reduces CCL-2 production by >50% in the BM supernatants and the systemic circulation compares to the control mice. DMHCA also increases the number of CACs

	in the BM and peripheral circulation and upregulates the egression of vascular reparative cells into the peripheral circulation [1]. In the PAGA analysis, DMHCA treatment enhances the overall production and robustness of erythrocyte progenitors. DMHCA enhances flux down the erythrocyte progenitor lineage. It also DMHCA increase in erythrocyte progenitor density in BM cells. DMHCA not only expands the proportion of erythrocyte progenitors but also increases the expression of the hemoglobin beta adult chain (HBB-BT)[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	DMHCA (oral administration; 6 months; chow 8mg/kg body weight /day) restores cholesterol homeostasis in diabetic retina. It significantly increases endogenous LXR ligands, desmosterol, and total oxysterols in the diabetic retina[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Cristiano P Vieira, et al. Selective LXR agonist DMHCA corrects retinal and bone marrow dysfunction in type 2 diabetes. JCI Insight. 2020 Jul 9;5(13):e137230.

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