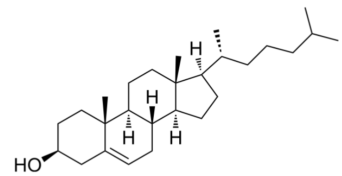
Cholesterol

Cat. No.:	HY-N0322
CAS No.:	57-88-5
Molecular Formula:	$C_{27}H_{46}O$
Molecular Weight:	386.65
Target:	Estrogen Receptor/ERR; Endogenous Metabolite; Bacterial; Liposome
Pathway:	Vitamin D Related/Nuclear Receptor; Metabolic Enzyme/Protease; Anti-infection
Storage:	Powder -20°C 3 years; 4°C 2 years; In solvent -80°C 6 months; -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

Ethanol : 20 mg/mL (51.73 mM; Need ultrasonic)
DMSO : < 1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble or slightly soluble)

Preparing Stock Solutions

Concentration / Solvent / Mass	1 mg	5 mg	10 mg
1 mM	2.5863 mL	12.9316 mL	25.8632 mL
5 mM	0.5173 mL	2.5863 mL	5.1726 mL
10 mM	0.2586 mL	1.2932 mL	2.5863 mL

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

In Vivo

1. Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1.43 mg/mL (3.70 mM); Clear solution
2. Add each solvent one by one: 10% EtOH >> 90% corn oil
   Solubility: ≥ 1.43 mg/mL (3.70 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cholesterol is the major sterol in mammals. It is making up 20-25% of structural component of the plasma membrane. Plasma membranes are highly permeable to water but relatively impermeable to ions and protons. Cholesterol plays an important role in determining the fluidity and permeability characteristics of the membrane as well as the function of both the transporters and signaling proteins[1][2]. Cholesterol is also an endogenous estrogen-related receptor α (ERRα) agonist[3]
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IC_{50} & Target

Microbial Metabolite	Human Endogenous Metabolite

In Vitro

GT1-7 hypothalamic cells subjected to Cholesterol depletion in vitro produced 20-31% reductions in cellular Cholesterol content. All Cholesterol-depleted neuron-derived cells, exhibit decreased phosphorylation/activation of IRS-1 and AKT following stimulation by insulin, insulin-like growth factor-1, or the neurotrophins (NGF and BDNF). Reduction in cellular Cholesterol also results in increased basal autophagy and impairment of induction of autophagy by glucose deprivation[1].

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

In Vivo

Cholesterol can be used in animal modeling to construct a rat hyperlipidemia model. The metabolic half-life of Cholesterol varies with the type of lipoprotein it binds and the tissue in which it is located, ranging from hours to years. Cholesterol can be used for hyperlipidemia, atherosclerosis and other related modeling purposes[4].The metabolic half-life of Cholesterol varies with the type of lipoprotein it binds to and the different tissues it is located in, ranging from a few hours to several years. Cholesterol can be used for modeling purposes related to hyperlipidemia and atherosclerosis[4].

1. Induction of Hyperlipidemia[5][6]

Background

Hyperlipidemia is a group of disorders characterized by elevated concentrations of circulating lipids, including cholesterol, cholesterol esters, phospholipids and triglycerides. If the intake of cholesterol is too much, and exceeds the body's metabolic capacity, it may lead to increased plasma cholesterol levels, causing hyperlipidemia.

Specific Mmodeling Methods

Rats: Wistar • male • 18-week-old (period: 8 weeks)

Administration: 2% cholesterol; diet • 8 weeks

Note

(1) Rats were raised in a room with a room temperature of 22±2°C and a 12-h light-dark cycle.(2) Serum cholesterol and triglyceride levels in Wistar rats are moderately increased by a high-cholesterol diet and do not develop substantial atherosclerosis. Wistar rats can be used in models of hyperlipidemia to study the direct effects of hyperlipidemia on the myocardium independent of atherosclerosis.

Modeling Record

Molecular changes: Significant increase in total cholesterol levels in blood samples (about 20%)

Correlated Product(s):

2. Induction of atherosclerosis[7][8]

Background

High levels of cholesterol in the blood, especially low-density lipoprotein cholesterol (LDL-C), can accumulate plaque on the walls of blood vessels, a process known as atherosclerosis. Over time, these plaques can block blood flow and cause serious health problems such as myocardial ischemia or myocardial infarction.

Specific Mmodeling Methods

Rabbits: Oryctolagus cuniculus • male • 4–6-month-old (period: 16 weeks)

Administration: 0.3% cholesterol and 3% soybean oil; diet • 16 weeks

Note

(1) The cholesterol-fed rabbit is a widely used model for experimental atherosclerosis research as cholesterol only cause atherosclerotic changes in the rabbit arterial intima, which was very similar to human atherosclerosis.

(2) As the absorption of dietary cholesterol requires fat, you must add oil into the diet. Otherwise, rabbits will use their internal fat, which makes them lean or sick. In addition, using soybean oil, which consists of unsaturated fatty acids, can prevent the levels of plasma cholesterol from becoming too high. Other vegetable oils, such as peanut oil or corn oil, can be used because they are all unsaturated fatty acids. Animal fat (saturated fatty acids) like tallow and lard is not recommended.

(3) 0.3–0.5% cholesterol diet is recommended for most experiments. Rabbits cannot tolerate a 1–2% cholesterol diet for a month as they develop severe liver dysfunction.

(4) Adult rabbits at 4 months or older can consume approximately ~150 g a day. You can either feed ab libitum or restricted (100–150 g/day/adult rabbit).

(5) Plasma lipids should be measured weekly, especially for the first 4 weeks, because you need to determine whether plasma levels of cholesterol are elevated in each animal. Non-responder rabbits can be excluded from the experiments if their plasma cholesterol levels do not increase after cholesterol diet feeding.

(6) Plasma lipoproteins can be measured at 8 and 16 weeks when the plasma levels of cholesterol are stable.

(7) The age of rabbits should be considered because young rabbits are more susceptible to aortic atherosclerosis than old rabbits even though they have similar plasma cholesterol levels. 4–6-month-old rabbits are usually used for cholesterol feeding experiments.

(8) Male and female rabbits are different in terms of response to a cholesterol diet and atherosclerosis. In our experience, female rabbits develop higher hypercholesterolemia and greater aortic lesions than their counterpart male rabbits. In general, male rabbits are recommended for experiments because estrogen may influence the results.

Modeling Record

Histological changes: atherosclerosis lesions can be seen on HE stained aortic arch and thoracic aorta segments

Correlated Product(s): Soybean oil (HY-108750)

CUSTOMER VALIDATION

- Nat Nanotechnol. 2021 Oct;16(10):1150-1160.
- Immunity. 2024 May 14;57(5):1087-1104.e7.
- Nat Commun. 2024 Jan 2;15(1):162.
- Adv Sci (Weinh). 2024 Jul 1:e2403640.
- Adv Sci (Weinh). 2023 Sep;10(27):e2206878.

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REFERENCES

[1]. Casaburi I, et al. Cholesterol as an Endogenous ERRα Agonist: A New Perspective to Cancer Treatment. Front Endocrinol (Lausanne). 2018 Sep 11;9:525.

[2]. Dietschy JM, et al. Thematic review series: brain Lipids. Cholesterol metabolism in the central nervous system during early development and in the mature animal. J Lipid Res. 2004 Aug;45(8):1375-97.

[3]. Fukui K, et al. Effect of Cholesterol Reduction on Receptor Signaling in Neurons. J Biol Chem. 2015 Sep 14.

Caution: Product has not been fully validated for medical applications. For research use only.

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