Estriol

Cat. No.: HY-B0412

CAS No.: 50-27-1

Molecular Formula: $C_{18}H_{24}O_3$

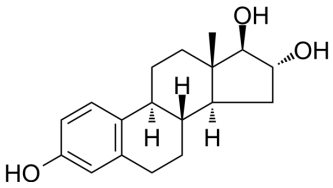
Molecular Weight: 288.38

Target: Endogenous Metabolite; Estrogen Receptor/ERR

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

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (866.91 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 80°C) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.4676 mL	17.3382 mL	34.6765 mL
5 mM	0.6935 mL	3.4676 mL	6.9353 mL
10 mM	0.3468 mL	1.7338 mL	3.4676 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (7.21 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (7.21 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (7.21 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Estriol (Oestriol), an orally active estrogen, is a ERα and ERβ agonist. Estriol is a potent GPR30 antagonist in estrogen receptor-negative breast cancer cells. Estriol can ameliorate disease severity through immunomodulatory mechanisms that decrease tissue inflammation. Estriol has powerful proconvulsant effects[1][2][3].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

Estriol (Oestriol) binds to both ER and GPR30, however it can exhibit ER agonism or GPR30 antagonism depending on the receptor expression profile in the different cancer cell contexts[1].

Estriol (1-80 μM; for 7 days) reduces cell number of 17β-estradiol stimulated HCC1806 cells very clearly down to 16% at 80 μM[2].

Estriol (100 μM; pretreated for 30 minutes) completely prevented activation of cyclin D1 expression by 17β-estradiol[2].

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

Cell Viability Assay[2]

Cell Line:	GPR30 positive (HCC1806) and a GPR30 negative TNBC cell line (MDA-MB-231)
Concentration:	1, 10, 20, 40, 60, 80 μM
Incubation Time:	For 7 days
Result:	Reduced cell number of 17β-estradiol stimulated HCC1806 cells very clearly down to 16±12% ($p < 0.01$) at 80 μM whereas in MDA-MB-231 cells was reduced to only 61±10% at the highest applied concentration.

Cell Cycle Analysis[2]

Cell Line:	HCC1806 cells
Concentration:	100 μM
Incubation Time:	Pretreated for 30 minutes
Result:	Completely prevented activation of cyclin D1 expression by 17β-estradiol (10 nM; 10 min or 20 minutes).

In Vivo

Estriol (Oestriol ; 0.005, 0.01 mg/kg; IP; daily; for 5 weeks) enhances the percentage incidence of seizures[3].

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

Animal Model:	Swiss Albino mice weighing between 25 and 35 g[3]
Dosage:	0.005, 0.01 mg/kg
Administration:	IP; daily; for 5 weeks
Result:	Reduced the time for induction of kindling from 5 weeks to 3 and 2 weeks for male and female mice respectively and enhanced the percentage incidence of seizures.

CUSTOMER VALIDATION

- Nat Chem Biol. 2022 Aug 18.
- Biosens Bioelectron. 12 July 2022, 114548.
- Proc Natl Acad Sci U S A. 2022 Apr 12;119(15):e2117004119.

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REFERENCES

[1]. Rosamaria Lappano, et al. Estriol acts as a GPR30 antagonist in estrogen receptor-negative breast cancer cells. Mol Cell Endocrinol. 2010 May 14;320(1-2):162-70.

[2]. Rainer Girgert, et al. Inhibition of GPR30 by estriol prevents growth stimulation of triple-negative breast cancer cells by 17β-estradiol. BMC Cancer. 2014 Dec 11:14:935.

[3]. Aakifa Ahmad, et al. Proconvulsant effects of estriol, the third estrogen, in the mouse PTZ-kindling model. Neurol Sci. 2014 Oct;35(10):1561-6.

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