# Data Sheet (Cat.No.T1571)



### **Estriol**

# **Chemical Properties**

CAS No.: 50-27-1

Formula: C18H24O3

Molecular Weight: 288.38

Appearance: no data available

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description					
Description	Estriol (Oestriol) is a hydroxylated metabolite of ESTRADIOL or ESTRONE that has a hydroxyl group at C3, 16-alpha, and 17-beta position. Estriol is a major urinary estrogen. During PREGNANCY, a large amount of estriol is produced by the PLACENTA.				
Targets(IC50)	Estrogen Receptor/ERR,Estrogen/progestogen Receptor,Endogenous Metabolite,MRF				
In vitro	Intraperitoneal injection of 20 mg/kg of Estriol in rats increased the sensitivity of Kupffe cells to lipopolysaccharides (LPS) through a mechanism that depends on the elevation of portal blood endotoxin, which is caused by an increase in rat intestinal permeability leading to a rise in CD14. When half of the rats were given an intraperitoneal preinjection of Estriol 24 hours before administering a sub-lethal dose of LPS (5 mg/kg), 50% of these rats died within 24 hours. In mPTEN +/- mice, Estriol treatment resulted in 187.54% increase in the relative ratio of uterine wet weight to body weight, while in wild-type mice, the same treatment led to a 176.88% increase in the ratio.				
n vivo	Research indicates that estrogens (estrone, estradiol, and estriol) inhibit the formation of low-level Aβ oligomers associated with Alzheimer's disease, with estriol demonstrating the strongest in vitro activity. Cell-free transcription assays reveal that the anti-estrogenic activity exhibited by estriol is due to its interference with the positive cooperation binding and dimerization induced by estradiol, the binding of hER complexes with ERE, and the reduction in estradiol-dependent transcription, highlighting a dose-dependent effect. MTT assays utilizing G-1 show that in SkBr3 cells the proliferative action induced by 100 nM G-1 is nullified in the presence of 1 μM estricating as an antagonist in the GPR30-dependent pathway.				
Cell Research	For the 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay, cel are cultured in plastic 96-well plates under 200 µL of growth medium and at an initial density of 10,000 cells per well. Cells are washed once they have attached and further incubated in medium containing 2.5% charcoal-stripped FBS with the indicated treatment. The medium is changed every 2 days (with treatment). Where applicable, 20 ng of the indicated plasmids are transfected every 2 days before treatments using Fugene6 Reagent, as recommended by the manufacturer. Following 6 days of incubation, the assay mixture (10µL per well) containing 1 mg/mL 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) is added to each well an				

incubated at 37 ?C for 4 h in a 5% CO2 atmosphere. The yellow tetrazolium MTT, reduced by metabolically active cells, results in intracellular purple formazan, which is released after overnight incubation with 200  $\mu$ L 1% sodium dodecyl sulfate in 0.01N HCL and

quantified spectrophotometrically by reading absorbance at 570 nM using an enzymelinked immunosorbent assay plate reader.(Only for Reference)

# **Solubility Information**

C - 1l- :1:4	DMCO. CO was freel (200 OC well) Construction in management and all	
Solubility	DMSO: 60 mg/mL (208.06 mM), Sonication is recommended.	
	Ethanol: 10 mg/mL (34.68 mM), Sonication is recommended.	
	H2O: < 1 mg/mL (insoluble or slightly soluble),	
	(< 1 mg/ml refers to the product slightly soluble or insoluble)	

## **Preparing Stock Solutions**

	1mg	5mg	10mg
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
50 mM	0.0694 mL	0.3468 mL	0.6935 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

#### Reference

Lappano R, et al. Mol Cell Endocrinol. 2010, 320(1-2), 162-170.

Melamed M, et al. Endocrinol. 1997, 11(12), 1868-1878.

Morinaga A, et al. Exp Neurol. 2011, 228(2), 298-302.

Begum M, et al. Lab Invest. 2006,86(3), 286-296.

Hewitt SC, et al. Environ Health Perspect. 2011, 119(1), 63-70.

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Page 2 of 2 www.targetmol.com