Hydrocortisone

Cat. No.: HY-N0583

CAS No.: 50-23-7

Molecular Formula: $C_{21}H_{30}O_5$

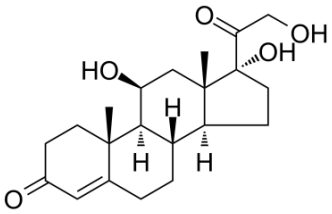
Molecular Weight: 362.46

Target: Glucocorticoid Receptor; Endogenous Metabolite

Pathway: GPCR/G Protein; Metabolic Enzyme/Protease

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 31 mg/mL (85.53 mM)

H_2O : 1.2 mg/mL (3.31 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.7589 mL	13.7946 mL	27.5893 mL
	5 mM	0.5518 mL	2.7589 mL	5.5179 mL
	10 mM	0.2759 mL	1.3795 mL	2.7589 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: ≥ 2.5 mg/mL (6.90 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.90 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.90 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Hydrocortisone (Cortisol) is a steroid hormone or glucocorticoid secreted by the adrenal cortex[1].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

Hydrocortisone (50 nM) shows a dose-dependent down-regulation of GR transcript in hCMEC/D3 cells. Hydrocortisone supplementation of the serum-reduced cell differentiation medium leads to a significant increase in TER across the hCMEC/D3 monolayer[1]. Hydrocortisone-treated Dendritic cells (DCs) show a decreased expression of MHC II molecules, the

costimulatory molecule CD86, and the DC-specific marker CD83, as well as a strongly reduced IL-12 secretion. Hydrocortisone-treated DCs inhibit production of IFN-γ but induce an increased release of IL-4 and no change in IL-5[2]. Hydrocortisone reduces postischemic oxidative stress, perfusion pressure, and transudate formation. Hydrocortisone inhibits postischemic shedding of syndecan-1, heparan sulfate, and hyaluronan as is release of histamine from resident mast cells[3].

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

PROTOCOL

Cell Assay [1]

Cells are plated on top of collagen IV-coated transwell chambers for six-well plates (24 mm diameter, membrane material: polyethylene terephthalate (PET), 0.4 μm pores, pore density 1.6×10^6 cm^2) at densities of 2.5×10^4 cells cm^2 per well. When they have reached confluence at day 5, the different experimental sets of cells are transferred to differentiation medium containing reduced amounts of FCS and treated with TNFα or hydrocortisone as indicated.

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

CUSTOMER VALIDATION

- Nature. 2021 Jan;589(7843):620-626.
- Exp Cell Res. 2020 Aug 1;393(1):112054.
- Cancer Manag Res. 2019 Jun 21;11:5557-5572.
- Fundação Oswaldo Cruz. Instituto Oswaldo Cruz. Laboratório de Transmissores de Hematozoários. 2020 Sep.

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REFERENCES

[1]. Förster C, et al. Differential effects of hydrocortisone and TNFalpha on tight junction proteins in an in vitro model of the human blood-brain barrier. J Physiol. 2008 Apr 1;586(7):1937-49.

[2]. Bellinghausen I, et al. Inhibition of human allergic T-cell responses by IL-10-treated dendritic cells: differences from hydrocortisone-treated dendritic cells. J Allergy Clin Immunol. 2001 Aug;108(2):242-9.

[3]. Chappell D, et al. Hydrocortisone preserves the vascular barrier by protecting the endothelial glycocalyx. Anesthesiology. 2007 Nov;107(5):776-84.

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