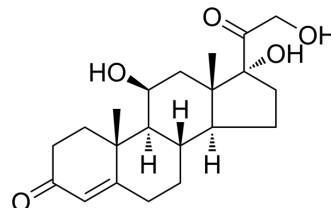


Hydrocortisone

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:	Immunology/Inflammation; Vitamin D Related/Nuclear Receptor; 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)
 H₂O : < 0.1 mg/mL (insoluble)
 * "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	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

- 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
- Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
Solubility: ≥ 2.5 mg/mL (6.90 mM); Clear solution
- Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (6.90 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.08 mg/mL (5.74 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.08 mg/mL (5.74 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

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

IC₅₀ & Target	Human Endogenous Metabolite
In Vitro	<p>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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

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 $1.6 \times 10^6 \text{ cm}^2$) 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 TNF α 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.
- Cancer Commun (Lond). 2021 Jul;41(7):576-595.
- Nat Chem Biol. 2022 Aug 18.
- Sci Adv. 2023 Oct 6;9(40):eadi6586.
- J Pharm Anal. 2024 Jan 4.

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REFERENCES

[1]. Förster C, et al. Differential effects of hydrocortisone and TNF α 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.

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

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