

Produktinformation



Forschungsprodukte & Biochemikalien
Zellkultur & Verbrauchsmaterial
Diagnostik & molekulare Diagnostik
Laborgeräte & Service

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Lieferung & Zahlungsart siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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Data Sheet (Cat.No.T1614)



Hydrocortisone

Chemical Propert	ies	
CAS No. :	50-23-7	
Formula:	C21H30O5	
Molecular Weight:	362.46	
Appearance:	no data available	
Storage:	keep away from direct sunlight Powder: -20°C for 3 years In solvent: -80°C for 1 year	

Description	Hydrocortisone (Cortisol) is a glucocorticoid hormone secreted by the adrenocortex. Hydrocortisone agonizes the glucocorticoid receptor, which promotes proteolytic metabolism, gluconeogenesis, capillary wall stabilization, and renal calcium excretion, as well as suppressing immune and inflammatory responses.
Targets(IC50)	Glucocorticoid Receptor,Endogenous Metabolite
In vitro	 METHODS: Human articular chondrocytes were cultured in medium containing Hydrocortisone (0.05-1.0 µg/mL), and the relevant indexes were measured by Flow Cytometry. RESULTS: The catabolic signaling pathway of chondrocytes treated with 0.05 µg/mL Hydrocortisone was reduced, and the ability to synthesize ECM macromolecules was enhanced. [1] METHODS: Porcine brain capillary endothelial cells PBCEC were cultured with medium containing Hydrocortisone (550 nM) for seven days, and the cytoskeleton was examined using fluorescence microscopy. RESULTS: Hydrocortisone-induced cytoskeletal rearrangement. [2]
In vivo	In ex vivo guinea pig hearts, Hydrocortisone reduces post-ischemic oxidative stress, perfusion pressure, and effusion formation. Within the human cerebral microvascular endothelial cell line hCMEC/D3, Hydrocortisone prevents endothelial barrier disruption induced by pro-inflammatory stimulation (administration of TNFα), partly by maintaining occludin levels. Hydrocortisone induces class-switch recombination from S to Sε in IL-4-treated B cells, supporting a model for sequential isotype transformation from IgM to IgE through IgG4. Following dendritic cell treatment, Hydrocortisone decreases the expression of MHC class II molecules, the dendritic-specific marker CD83, and costimulatory molecule CD86, while significantly reducing IL-12 secretion. Additionally, it inhibits the increase of IL-4 (induced by IFN-γ) but does not affect IL-5. Hydrocortisone lowers T cell proliferation in dendritic cells. It prevents TNF-α-induced severe degradation of glycocalyx, increases in coronary resistance, vascular leakage and permeability, hydroxyethyl starch, and causes degranulation of mast cells in ex viv
	guinea pig hearts. Hydrocortisone inhibits the shedding of multiligand glycosaminoglycans-1, heparan sulfate, and hyaluronic acid post-ischemia and suppresses the release of histamine from resident mast cells.

A DRUG SCREENING EXPERT

Cell Research	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 μ m pores,
	pore density 1.6×106?cm2) at densities of 2.5×104?cells cm2?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.

Solubility Information

Solubility	DMSO: 50 mg/mL (137.95 mM),
	Ethanol: 22 mg/mL (60.7 mM),
	(< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg	
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	
50 mM	0.0552 mL	0.2759 mL	0.5518 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

Wang J, et al. Physiological levels of hydrocortisone maintain an optimal chondrocyte extracellular matrix metabolism. Ann Rheum Dis. 2004 Jan;63(1):61-6.
br/>Lei H, Wang Z, Jiang D, et al. CRISPR screening identifies

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