Dexamethasone

Cat. No.: HY-14648

CAS No.: 50-02-2

Molecular Formula: $C_{22}H_{29}FO_5$

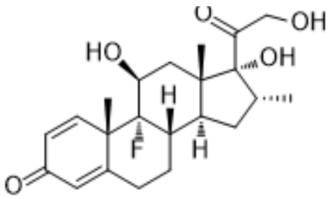
Molecular Weight: 392.46

Target: Glucocorticoid Receptor; Autophagy; Mitophagy; Complement System; Bacterial; Antibiotic; SARS-CoV; ADC Cytotoxin

Pathway: Immunology/Inflammation; Vitamin D Related/Nuclear Receptor; Autophagy; Anti-infection; Antibody-drug Conjugate/ADC Related

Storage: 4°C, protect from light

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (637.01 mM; ultrasonic and warming and heat to 60°C)
Ethanol : 8.33 mg/mL (21.23 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5480 mL	12.7402 mL	25.4803 mL
	5 mM	0.5096 mL	2.5480 mL	5.0961 mL
	10 mM	0.2548 mL	1.2740 mL	2.5480 mL

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

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 18.18 mg/mL (46.32 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.37 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (5.30 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.30 mM); Clear solution
5. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.30 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Dexamethasone (Hexadecadrol) is a glucocorticoid receptor agonist, apoptosis inducer, and common disease inducer in experimental animals, constructing models of muscle atrophy, hypertension, and depression. Dexamethasone can inhibit

	the production of inflammatory miRNA-155 exosomes in macrophages and significantly reduce the expression of inflammatory factors in neutrophils and monocytes. Dexamethasone also has potential for use in COVID-19 research[1][2][3][4].
IC_{50} & Target	Glucocorticoid receptor[1]
In Vitro	Dexamethasone (Hexadecadrol) regulates several transcription factors, including activator protein-1, nuclear factor-AT, and nuclear factor-kB, leading to the activation and repression of key genes involved in the inflammatory response[1]. Dexamethasone potently inhibits granulocyte-macrophage colony stimulating factor (GM-CSF) release from A549 cells with EC_{50} of 2.2 nM. Dexamethasone (EC_{50}=36 nM) induces transcription of the β_2-receptor is found to correlate with glucocorticoid receptor (GR) DNA binding and occurred at 10-100 fold higher concentrations than the inhibition of GM-CSF release. Dexamethasone (IC_{50}=0.5 nM) inhibits a 3×κB (NF-κB, IκBα, and I-κBβ), which is associated with inhibition of GM-CSF release[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Based on blood and multi-tissue concentration-time profiles of Dexamethasone (DEX), no significant sex differences were found in its tissue distribution. Blood cell to plasma partitioning (0.664) and plasma free fraction (0.175) were moderate, with widespread distribution in the liver (Kp=6.76). Possibly due to P-glycoprotein-mediated efflux, the concentration of DEX in the brain is very low compared to the expected high permeability[5]. Dexamethasone (DEX) can be used in animal modeling to construct models of muscle atrophy, hypertension and depression.

1. Induction of muscle atrophy[6][7]

- Background

  Glucocorticoids are important mediators of skeletal muscle protein degradation and upregulation of the ubiquitin-proteasome pathway. Dexamethasone induces tibialis anterior muscle protein degradation by binding to the glucocorticoid receptor, resulting in muscle atrophy.

- Specific Mmodeling Methods

  Mice: C57BL/6 • male • 6-week-old
  Administration: 5 mg/kg • ip • once daily for 2 weeks

- Modeling Indicators

  Molecular changes: Increased indicators: C_2C_{12} ubiquitin ligase, MuRF1, Atrogin-1, Cbl-b, p-Foxo1, p-Foxo3a. Resulted C_2C_{12} myotube protein degradation, and Glucocorticoid receptor translocation to the nucleus

  Phenotypic observation: Decreased indicators: The weight of the anterior muscles, gastrocnemius, quadriceps and soleus muscles. The ratio of skeletal muscle to body weight decreases.

- Correlated Product(s): Betamethasone (HY-13570)

Opposite Product(s): Glabridin (HY-N0393)

2. Induction of hypertension[8][9]

- Background

  The underlying mechanisms that induce hypertension (HT) are unknown.

- Specific Modeling Methods

  Rat: Sprague-Dawley • Male • 200-300 g
  Administration: 20 μg • sc • once daily from days 5 to 16 • control rats: saline with 0.1 mL/100 g/day from days 1 to 16 (po) or 0.2 mL/rat/day (sc).

  Dog: 10.1-19.1 kg • average=13.7 kg
  Administration: 0.5 mg/kg • po • once daily for 10 days

- Modeling Indicators

  b>Hemodynamics：MAP, systolic blood pressure, diastolic blood pressure, TPR levels increased in central hemodynamics. Total peripheral resistance, blood pressure, atrial natriuretic peptide, and the pressor response to norepinephrine, are significantly increased in Systemic and renal hemodynamics.

  Behavior: The dog showed obvious natriuresis and diuresis.

- Opposite Product(s): Saralasin (HY-P0205); Prazosin (HY-B0193)

3. Induction of depressive behavior[10][11]

- Background

  Astrocytes are a key feature of major depressive disorder (MD), and reduced expression by glucocorticoids results in reduced astrocyte numbers. Long-term treatment with Dexamethasone can cause a series of depression-like symptoms in rodents.

- Specific Modeling Methods

  Rat: Sprague-Dawley • Male • 200-250 g
  Administration: 1 mg/kg • po • once daily every other day for 5 months

Note

(1) Prepare a 3% (w/v) treatment solution and filter through a 0.45 μm cellulose acetate filter.

(2) Rats should not be exposed to any other water source for 4 days before starting treatment.

(3) On the 4th day, change the treatment solution to water and feed for 3 days to allow the mice to recover temporarily. Weigh and conduct behavioral experiments on day 7.

Mice: C57BL/6 • Male • 9-10 weeks old • 23-25 g

Administration: 4 mg/kg • ip • once dailly for 21 days

- Modeling Indicators

Metabolism changes: Serum cortisol levels in rats ↑.

Behavior: Immobility time during the forced swim test (FST) &uarr. Preference for sucrose &darr in the sucrose preference test.

- Opposite Product(s): Amitriptyline (HY-B0527)

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

PROTOCOL

Animal Administration [3][4]

Mice[3]

Female C57Bl/6JBom mice (age 10-12 weeks) are used in all experiments. Dexamethasone is administered as a single injection of 1 or 10 mg/kg. Dexamethasone is dissolved in saline and 400 μL are injected intraperitoneally, either 1 h before or 1 h after LPS exposure. In one experiment, N-acetylcysteine (NAC) (100 and 500 mg/kg) is injected successively every 4•5 h, starting 1 h before challenge (five injections in total). A control group of LPS-exposed animals are injected intraperitoneally with solvent alone (saline). Intratracheal administration is performed by instillation of 100 μL NAC (50, 100 or 500 mg/kg) or Dexamethasone (10 mg/kg) into the lungs of mice.

Rats[4]

Male Sprague-Dawley rats are used.Dexamethasone-treated rats are injected intraperitoneally once daily with Dexamethasone (1.5 mg/kg body weight) for 5 days and are allowed to feed ad libitum. The Dexamethasone dose (1.5 mg/kg/day) and the duration of treatment (5 days) are specifically chosen as this treatment induced a reproducible and marked catabolic state. Control rats received no treatment and are fed ad libitum. In order to take into account the decrease in food intake induced by Dexamethasone treatment, a third group of pair-fed rats are used. These rats are provided with the same amount of food as Dexamethasone-injected rats and are treated with a daily isovolumic intraperitoneal injection of NaCl (0.9%) for 5 days. After the final injection of Dexamethasone or NaCl, the animals are fasted overnight prior to being killed by decapitation.

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

CUSTOMER VALIDATION

- Cell. 2023 Jun 22;186(13):2823-2838.e20.

- Nat Genet. 2024 Mar 7.
- ACS Nano. 2023 Oct 27.
- Chem Eng J. 2023 Aug 6, 145212.
- Adv Sci (Weinh). 2022 Sep;9(26):e2202505.

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[2]. Troncoso R, et al. Dexamethasone-induced autophagy mediates muscle atrophy through mitochondrial clearance. Cell Cycle. 2014;13(14):2281-95.

[3]. Ong SL, et al. Hemodynamics of dexamethasone-induced hypertension in the rat. Hypertens Res. 2009 Oct;32(10):889-94.

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