Dexamethasone phosphate disodium

Cat. No.: HY-B1829A

CAS No.: 2392-39-4

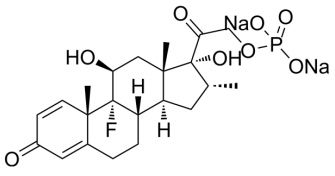
Molecular Formula: $C_{22}H_{28}FNa_2O_8P$

Molecular Weight: 516.4

Target: Glucocorticoid Receptor

Pathway: Immunology/Inflammation; Vitamin D Related/Nuclear Receptor

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 1 year; -20°C, 6 months (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 100 mg/mL (193.65 mM)
DMSO : 1 mg/mL (1.94 mM; ultrasonic and warming and heat to 80°C)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9365 mL	9.6824 mL	19.3648 mL
	5 mM	0.3873 mL	1.9365 mL	3.8730 mL
	10 mM	0.1936 mL	0.9682 mL	1.9365 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 100 mg/mL (193.65 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Dexamethasone phosphate (Dexamethasone 21-phosphate) disodium is an orally active Glucocorticoid receptor agonist[1].

IC_{50} & Target

Glucocorticoid receptor[1]

In Vitro

Dexamethasone phosphate disodium 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 phosphate disodium potently inhibits granulocyte-macrophage colony stimulating factor (GM-CSF) release from A549 cells with EC_{50} of 2.2 nM. Dexamethasone phosphate disodium (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 phosphate disodium (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	Treatment with Dexamethasone phosphate disodium at a dose of 2×5 mg/kg efficiently inhibits lipopolysaccharide (LPS)-induced inflammation. In our experimental system, treatment with a single dose of Dexamethasone phosphate disodium 10 mg/kg (i.p.) significantly decreases recruitment of granulocytes as well as spontaneous production of oxygen radicals compared with animals expose to LPS and injected with solvent alone (saline). The effects are statistically significant when administered both 1 h before and 1 h after inhalation of LPS. The number of granulocytes in BALF decreased to levels comparable to healthy animals (given an aerosol of water)[3]. Rats treated with Dexamethasone phosphate disodium consume less food and weighed less than control rats. Treated rats also weigh less than pair-fed animals though their food intake is similar. Five days of Dexamethasone phosphate disodium injection result in a significant increase in both the liver mass (+42%) and the liver to body weight ratio (+65%). The wet weight of gastrocnemius muscle decreases 20% after 5 days of treatment, but it remains unaffected relative to body weight (g/100 g body weight), indicating that muscle weight loss paralleled body weight loss[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [3][4]	Rats: 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. Control rats receive 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[4]. Mice: 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 anaesthetized with 15 mg/kg Rapinovet (i.v.)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2021 Dec 9;12(1):7162.
- MedComm. 2023 Jun 5;4(3):e293.

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REFERENCES

[1]. LaLone CA, et al. Effects of a glucocorticoid receptor agonist, Dexamethasone, on fathead minnow reproduction, growth, and development. Environ Toxicol Chem. 2012 Mar;31(3):611-22.

[2]. Adcock IM, et al. Ligand-induced differentiation of glucocorticoid receptor (GR) trans-repression and transactivation: preferential targetting of NF-kappaB and lack of I-kappaB involvement. Br J Pharmacol. 1999 Jun;127(4):1003-11.

[3]. Rocksén D, et al. Differential anti-inflammatory and anti-oxidative effects of Dexamethasone and N-acetylcysteine in endotoxin-induced lung inflammation. Clin Exp Immunol. 2000 Nov;122(2):249-56.

[4]. Roussel D, et al. Dexamethasone treatment specifically increases the basal proton conductance of rat liver mitochondria. FEBS Lett. 2003 Apr 24;541(1-3):75-9.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA