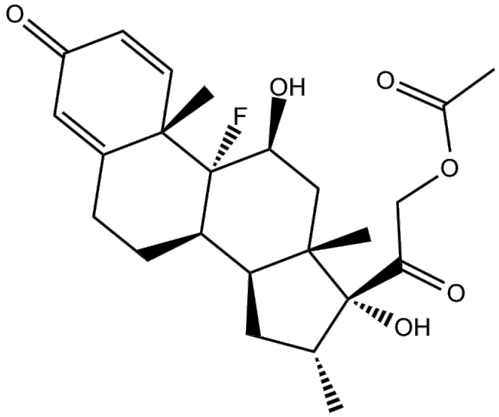
Product Data Sheet

Product Name: Dexamethasone acetate
Cat. No.: GC11237

Chemical Properties

Cas. No.	1177-87-3		
Chemical Name	[2-[(8S,9R,10S,11S,13S,14S,16R,17R)-9-fluoro-11,17-dihydroxy-10,13,16-trimethyl-3-oxo-6,7,8,11,12,14,15,16-octahydrocyclopenta[a]phenanthren-17-yl]-2-oxoethyl] acetate		
SMILES	CC1CC2C3CCC4=CC(=O)C=CC4(C3(C(CC2(C1(C(=O)COC(=O)C)O)C)O)F)C		
Formula	C24H31FO6	M.Wt	434.5
Solubility	≥ 13.35mg/mL in DMSO	Storage	4°C, protect from light
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

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Animal experiment: 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 anaesthetized with 15 mg/kg Rapinovet (i.v.). 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.

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

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.

Background

Dexamethasone acetate is a glucocorticoid receptor agonist.

Dexamethasone 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 EC50 of 2.2 nM. Dexamethasone (EC50=36 nM) induces transcription of the β2-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 (IC50=0.5 nM) inhibits a 3×κB (NF-κB, IκBα, and I-κBβ), which is associated with inhibition of GM-CSF release[2].

It has previously been reported that treatment with Dexamethasone 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 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 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 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].

Reference:

[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
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