Deoxycorticosterone acetate

Cat. No.: HY-B1472

CAS No.: 56-47-3

Molecular Formula: $C_{23}H_{32}O_4$

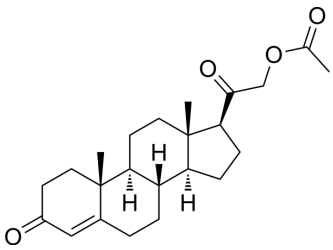
Molecular Weight: 372.5

Target: Mineralocorticoid Receptor; Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 16.67 mg/mL (44.75 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6846 mL	13.4228 mL	26.8456 mL
5 mM	0.5369 mL	2.6846 mL	5.3691 mL
10 mM	0.2685 mL	1.3423 mL	2.6846 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.58 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.58 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1.67 mg/mL (4.48 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Deoxycorticosterone acetate (DOCA) is an adrenocortin, acts as a precursor to aldosterone. Deoxycorticosterone acetate is a mineralocorticoid receptor agonist. Deoxycorticosterone acetate can cause severe renal injury, including inflammation, fibrosis, glomerular damage, and proteinuria[1][2].

IC_{50} & Target

Human Endogenous Metabolite

In Vivo

Deoxycorticosterone acetate (2.5 mg/day; s.c. implantation) with Tamoxifen (HY-13757A) (42 d; 2 mg/day) pre-treatment) induces hypertension by increasing blood pressure and cardiac hypertrophy in mice[1].

Induction of hypertension[3][4]

- Background

  Deoxycorticosterone acetate (DOCA) inhibits the renin-angiotensin system, resulting in low plasma renin activity, thereby mediating an increase in blood pressure

- Specific Mmodeling Methods

  Rat: Sprague-Dawley with a right nephrectomy • male • 250 to 300 g
  Administration: 100 mg slow-release DOCA pellet was inserted subcutaneously and drinking water is replaced by 1% saline; 21 days
  Rat: Sprague-Dawley with a right nephrectomy • male • 160 to 180 g
  Administration: 15 mg/kg • sc • and drinking water is replaced by 1% saline; twice weekly for 2 weeks

- Modeling Indicators

  Pathological changes: systolic blood pressure increase, superoxide (vascular O2??) increase

- Opposite Product(s): Atrasentan (HY-15403)A-192621 (HY-120295)

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

Animal Model:	MR (mineralocorticoid receptor) mutant mice ($MR^{Cdh5Cre}$) and MR wild-type mice ($MR^{wild-type}$) treated with unilateral nephrectomy (12-week-old)[1]
Dosage:	2.5 mg/d
Administration:	Subcutaneous implantation; 42 days; treated with 2 mg Tamoxifen (HY-13757A) (20 mg/mL in sunflower oil and 10% ethanol; i.p.; once daily on 5 consecutive days) at least 4 weeks before nephrectomy
Result:	Increased the blood pressure without differences between both genotypes ($MR^{Cdh5Cre}$ and $MR^{wild-type}$). Resulted glomerular injury and proteinuria, renal inflammation and fibrosis.

REFERENCES

[1]. Lu NZ, et al. International Union of Pharmacology. LXV. The pharmacology and classification of the nuclear receptor superfamily: glucocorticoid, mineralocorticoid, progesterone, and androgen receptors. Pharmacol Rev. 2006 Dec;58(4):782-97.

[2]. Lother A, et al. Deoxycorticosterone Acetate/Salt-Induced Cardiac But Not Renal Injury Is Mediated By Endothelial Mineralocorticoid Receptors Independently From Blood Pressure. Hypertension. 2016 Jan;67(1):130-8.

[3]. M J Somers, et al. Vascular Superoxide Production and Vasomotor Function in Hypertension Induced by Deoxycorticosterone Acetate–Salt. Circulation. 2000 Apr 11;101(14):1722-8.

[4]. Y Matsumura, et al. Different Contributions of Endothelin-A and Endothelin-B Receptors in the Pathogenesis of Deoxycorticosterone Acetate–Salt–Induced Hypertension in Rats. Hypertension. 1999 Feb;33(2):759-65.

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