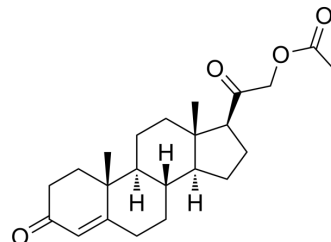


## Deoxycorticosterone acetate

<b>Cat. No.:</b>	HY-B1472		
<b>CAS No.:</b>	56-47-3		
<b>Molecular Formula:</b>	C <sub>23</sub> H <sub>32</sub> O <sub>4</sub>		
<b>Molecular Weight:</b>	372.5		
<b>Target:</b>	Mineralocorticoid Receptor; Endogenous Metabolite		
<b>Pathway:</b>	Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor		
<b>Storage:</b>	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<sub>2</sub>O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions	Solvent Concentration	Mass		
		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

- 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
- Add each solvent one by one: 10% DMSO >> 90% corn oil  
 Solubility: ≥ 2.08 mg/mL (5.58 mM); Clear solution
- 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<sup>[1][2]</sup>.

#### IC<sub>50</sub> & 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<sup>[1]</sup>.

#### Induction of hypertension<sup>[3][4]</sup>

- Background

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

- Specific Modeling 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 O<sub>2</sub><sup>••</sup>) 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 <sup>Cdh5Cre</sup> ) and MR wild-type mice (MR <sup>wild-type</sup> ) treated with unilateral nephrectomy (12-week-old) <sup>[1]</sup>
Dosage:	2.5 mg/d
Administration:	Subcutaneous implantation; 42 days; treated with 2 mg <a href="#">Tamoxifen</a> (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 <sup>Cdh5Cre</sup> and MR <sup>wild-type</sup> ). 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.

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

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: [tech@MedChemExpress.com](mailto:tech@MedChemExpress.com)

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA