Corticosterone

Cat. No.: HY-B1618

CAS No.: 50-22-6

Molecular Formula: $C_{21}H_{30}O_4$

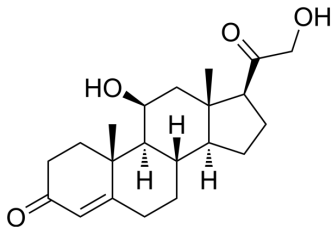
Molecular Weight: 346.46

Target: Glucocorticoid Receptor; Endogenous Metabolite; iGluR

Pathway: Immunology/Inflammation; Vitamin D Related/Nuclear Receptor; Metabolic Enzyme/Protease; Membrane Transporter/Ion Channel; Neuronal Signaling

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 : 100 mg/mL (288.63 mM; Need ultrasonic)

Ethanol : 14.29 mg/mL (41.25 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.8863 mL	14.4317 mL	28.8634 mL
5 mM	0.5773 mL	2.8863 mL	5.7727 mL
10 mM	0.2886 mL	1.4432 mL	2.8863 mL

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

In Vivo

1. Add each solvent one by one: 5% Cremophor EL >> 95% (20% HP-β-CD)
   Solubility: 5 mg/mL (14.43 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 20% HP-β-CD in saline
   Solubility: 4 mg/mL (11.55 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.22 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.22 mM); Clear solution
5. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.22 mM); Clear solution
6. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 2 mg/mL (5.77 mM); Suspended solution; Need ultrasonic and warming and heat to 60°C
7. Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1.43 mg/mL (4.13 mM); Clear solution

8. Add each solvent one by one: 10% EtOH >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 1.43 mg/mL (4.13 mM); Clear solution

9. Add each solvent one by one: 10% EtOH >> 90% corn oil
Solubility: ≥ 1.43 mg/mL (4.13 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Corticosterone (17-Deoxycortisol) is an orally active and adrenal cortex-produced glucocorticoid, which plays an important role in regulating neuronal functions of the limbic system (including hippocampus, prefrontal cortex, and amygdala). Corticosterone increases the Rab-mediated AMPAR membrane traffic via SGK-induced phosphorylation of GDI. Corticosterone also interferes with the maturation of dendritic cells and shows a good immunosuppressive effect[1][2][3][4].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

Corticosterone (100 nM; 30 min) via SGK phosphorylation of GDI at Ser-213, increases the formation of GDI-Rab4 complex, facilitating the functional cycle of Rab4 and Rab4-mediated recycling of AMPARs to the synaptic membrane[1].
Corticosterone (CORT) (1 μM; 48 h) shows good immunosuppressive properties (functionally compromises maturation of BMDC), which impairs LPS-induced up-regulation of maturation-associated markers (MHC class II, B7.2, B7.1 and CD40)[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	HEK293 cells
Concentration:	100 nM
Incubation Time:	30 min
Result:	Caused a significant enhancement of mEPSC amplitude (mEPSC represents the postsynaptic response to release of individual vesicles of glutamate). Increased the transmission of glutamatergic, and increased synaptic AMPAR currents via a Rab4-dependent mechanism. Profoundly increased surface GluR1 cluster density, cluster size and cluster fluorescence intensity. Significantly increased the amount of Rab4 that binded to WT-GDI, S45A-GDI, or S121A-GDI but not S213A-GDI. Induced the phosphorylation of GST-^{WT}GDI, GST-^{S45A}GDI, and GST-^{S121A}GDI, but not GST-^{S213A}GDI, and this effect was blocked in cells transfected with SGK1 small interfering RNA. Increased AMPAR surface expression via a mechanism dependent on GDI phosphorylation.

Cell Viability Assay[2]

Cell Line:	BMDC cells
Concentration:	1 μM
Incubation Time:	48 h
Result:	Completely blocked the expression of MHC class II and B7.2 that induced by LPS, and maximally impaired BMDC cells maturation at 12 h. Reduced B7.1 by 50%, and slightly down-regulated CD40.

In Vivo

Corticosterone results in a marked reduction in the ability of BMDC cells to prime naive $CD8^+$ T cells in vivo[2].
Corticosterone (0.03 or 1 mg/kg; s.c.; single) downregulates expression of BDNF mRNA in dentate gyrus and CA1 of rats[3].

Corticosterone (2.6 mg/kg; in animal feedings; 8 days) restores ethanol intake and preference to approximately normal preoperative levels in adrenalectomy (ADX) rats[4].

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

Animal Model:	Adult male Wistar rats (150-170 g; adrenalectomized)[3].
Dosage:	0.03 or 1 mg/kg
Administration:	Subcutaneous injection; single.
Result:	Decreased expression of BDNF mRNA in dentate gyrus, with 25% and 50% lower for dosages of 0.03 and 1 mg/kg, respectively (3 h after administration). Reduced approximately 40% BDNF mRNA level as compared to the t=0 h control group (3 h after administration), but the level increased by 100% when 12 h after administration (compared to t=3 h and t=6 h group).

Animal Model:	Male Wistar rats (3-week-old; adrenalectomized)[4].
Dosage:	2.6 mg/kg
Administration:	In animal feedings; 8 days.
Result:	Restored ethanol intake and preference of adrenalectomy (ADX) rats to approximately normal preoperative levels and to the levels observed in the sham-operated group (SH) rats.

CUSTOMER VALIDATION

- Cell. 2023 Dec 7;186(25):5500-5516.e21.
- Cell Discov. 2023 Aug 29;9(1):90.
- Nat Neurosci. 2021 Dec 9.
- Nat Chem Biol. 2022 Aug 18.
- Sci Adv. 2022 Nov 11;8(45):eadd7063.

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REFERENCES

[1]. Elftman MD, et al. Corticosterone impairs dendritic cell maturation and function. Immunology. 2007 Oct;122(2):279-90.

[2]. Schaaf MJ, et al. Downregulation of BDNF mRNA and protein in the rat hippocampus by corticosterone. Brain Res. 1998 Nov 30;813(1):112-20.

[3]. Fahlke C, et al. Involvement of corticosterone in the modulation of ethanol consumption in the rat. Alcohol. 1994 May-Jun;11(3):195-202.

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