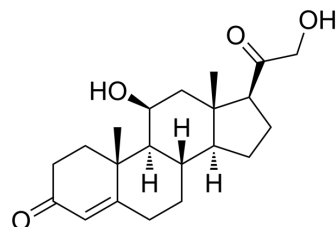


Corticosterone

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:	<table border="0"> <tr> <td>Powder</td> <td>-20°C</td> <td>3 years</td> </tr> <tr> <td></td> <td>4°C</td> <td>2 years</td> </tr> <tr> <td>In solvent</td> <td>-80°C</td> <td>1 year</td> </tr> <tr> <td></td> <td>-20°C</td> <td>6 months</td> </tr> </table>	Powder	-20°C	3 years		4°C	2 years	In solvent	-80°C	1 year		-20°C	6 months
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)
 H₂O : < 0.1 mg/mL (ultrasonic) (insoluble)

Preparing Stock Solutions	Solvent Concentration	Mass		
		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

- Add each solvent one by one: 5% Cremophor EL >> 95% (20% HP-β-CD in Saline)
Solubility: 5 mg/mL (14.43 mM); Clear solution; Need ultrasonic
- Add each solvent one by one: 20% HP-β-CD in saline
Solubility: 4 mg/mL (11.55 mM); Suspended solution; Need ultrasonic
- 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
- 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
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (7.22 mM); Clear solution
- 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
- 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: \geq 1.43 mg/mL (4.13 mM); Clear solution
9. Add each solvent one by one: 10% EtOH >> 90% corn oil
Solubility: \geq 1.43 mg/mL (4.13 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	<p>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]}.</p>																
IC₅₀ & Target	Human Endogenous Metabolite																
In Vitro	<p>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.</p> <p>Cell Viability Assay^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>HEK293 cells</td> </tr> <tr> <td>Concentration:</td> <td>100 nM</td> </tr> <tr> <td>Incubation Time:</td> <td>30 min</td> </tr> <tr> <td>Result:</td> <td> <p>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-S^{45A}GDI, and GST-S^{121A}GDI, but not GST-S^{213A}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.</p> </td> </tr> </table> <p>Cell Viability Assay^[2]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>BMDC cells</td> </tr> <tr> <td>Concentration:</td> <td>1 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>48 h</td> </tr> <tr> <td>Result:</td> <td> <p>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.</p> </td> </tr> </table>	Cell Line:	HEK293 cells	Concentration:	100 nM	Incubation Time:	30 min	Result:	<p>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-S^{45A}GDI, and GST-S^{121A}GDI, but not GST-S^{213A}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.</p>	Cell Line:	BMDC cells	Concentration:	1 μ M	Incubation Time:	48 h	Result:	<p>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.</p>
Cell Line:	HEK293 cells																
Concentration:	100 nM																
Incubation Time:	30 min																
Result:	<p>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-S^{45A}GDI, and GST-S^{121A}GDI, but not GST-S^{213A}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.</p>																
Cell Line:	BMDC cells																
Concentration:	1 μ M																
Incubation Time:	48 h																
Result:	<p>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.</p>																
In Vivo	<p>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].</p>																

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.

See more customer validations on www.MedChemExpress.com

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.

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

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