Prednisone acetate

Cat. No.:	HY-B1832
CAS No.:	125-10-0
Molecular Formula:	$C_{23}H_{28}O_6$
Molecular Weight:	
Target:	Glucocorticoid Receptor; Interleukin Related; Notch
Pathway:	Immunology/Inflammation; Vitamin D Related/Nuclear Receptor; Neuronal Signaling; O
Storage:	Powder -20°C 3 years 4°C 2 years
	In solvent -80°C 6 months -20°C 1 month

SOLVENT & SOLUBILITY

In Vitro		Solvent Mass Concentration	1 mg	5 mg	10 mg		
	Preparing Stock Solutions	1 mM	2.4971 mL	12.4856 mL	24.9713 mL		
		5 mM	0.4994 mL	2.4971 mL	4.9943 mL		
		10 mM	0.2497 mL	1.2486 mL	2.4971 mL		
	Please refer to the so	lubility information to select the app	propriate solvent.				
n Vivo		1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (6.24 mM); Clear solution					
		2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.08 mg/mL (5.19 mM); Clear solution					
		3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (5.19 mM); Clear solution					

BIOLOGICAL ACTIV			
BIOLOGICALMONT			
Description		ne 21-acetate), a glucocorticoid, d can enhance the immune resp	is an orally active Notch inhibitor. Prednisone acetate has $onse^{[1][2]}$.
IC ₅₀ & Target	IL-17	IL-10	IL-4
In Vivo	Prednisone acetate (5 mg/kg,	Intragastric gavage, once a day	for 4 weeks) induces hippocampal LTP impairment by causing

Product Data Sheet

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neuronal lesions in the dentate gyrus, which reduced glutamic acid (Glu) and NMDAR2A, and impaired spatial memory in Hippocampal LTP impairment model mice^[1].

Prednisone acetate (6 mg/kg, gavage, once a day for 15 days) reduces ocular and peripheral inflammatory responses and restores Th1/Th2 and Th17/Treg immune homeostasis through orchestrating the Notch signaling pathway in Experimental autoimmune uveitis (EAU) rats^[2].

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

Animal Model:	C57BL/6 mice ^[1]
Dosage:	5 mg/kg
Administration:	Intragastric gavage (i.g.)
Result:	Significantly failed to gain weight. Decreased the population spike (%) after high-frequency stimulation. Reduced the crossing times compared with the control group. Resulted in the impairment of memory, and caused a trend of more serious impairment. Reduced the levels of Glu and gamma-aminobutyric acid (GABA) in the hippocampus. Reduced the expressions of N-methyl-D-aspartate receptors in the hippocampus.

Animal Model:	Experimental autoimmune uveitis ^[2]
Dosage:	6 mg/kg
Administration:	gavage
Result:	Showed fewer manifestations in eyes from day 9 after immunization. Reduced the inflammation in EAU rats. Bound with the pocket of Notch signaling-related molecules with good affinity. Exhibited anti-inflammatory effects through inhibiting Notch signaling activation. Decreased Th1, Th17 and increased Th2, Treg frequencies in EAU.

REFERENCES

[1]. Wang Y, et al. Intragastric administration of prednisone acetate induced impairment of hippocampal long-term potentiation [J]. Brain Research, 2023, 1805: 148270.

[2]. Zhou M, Qu R, Yin X, et al. Prednisone acetate modulates Th1/Th2 and Th17/Treg cell homeostasis in experimental autoimmune uveitis via orchestrating the Notch signaling pathway [J]. International Immunopharmacology, 2023, 116: 109809.

[3]. Prednisolone acetate

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

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