Rp-cAMPS sodium salt

Cat. No.:	HY-100530D	
CAS No.:	142439-94-9	
Molecular Formula:	$C_{10}H_{11}N_{5}NaO_{5}PS$	NH ₂
Molecular Weight:	367.25	
Target:	РКА	
Pathway:	Stem Cell/Wnt	Na ⁺ 0 ⁻ H [™] OH
Storage:	-20°C, sealed storage, away from moisture and light	
	* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture	
	and light)	

SOLVENT & SOLUBILITY

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In Vitro H	H ₂ O : 250 mg/mL (680.74 mM; Need ultrasonic)					
	Preparing Stock Solutions	Solvent Mass Concentration	1 mg	5 mg	10 mg	
		1 mM	2.7229 mL	13.6147 mL	27.2294 mL	
		5 mM	0.5446 mL	2.7229 mL	5.4459 mL	
		10 mM	0.2723 mL	1.3615 mL	2.7229 mL	
	Please refer to the solubility information to select the appropriate solvent.					
In Vivo	1. Add each solvent o Solubility: 100 mg	one by one: PBS /mL (272.29 mM); Clear solution; Ne	eed ultrasonic			

BIOLOGICAL ACTIVITY				
Description	Rp-cAMPS sodium salt, a cAMP analog, is a potent, competitive cAMP-induced activation of cAMP-dependent PKA I and II (K _i s of 12.5 μM and 4.5 μM, respectively) antagonist. Rp-cAMPS sodium salt is resistant to hydrolysis by phosphodiesterases ^{[1][2]} ^{[3][4][5][6]} .			
IC ₅₀ & Target	Ki: 6.05 μM (PKA I) and 9.75 μM (PKA II) $^{[1]}$			
In Vitro	A membrane-permeable competitive cAMP antagonist (Rp-cAMPS) that blocks PKA activation by binding to the regulatory subunits without dissociating the kinase holoenzyme also inhibits synaptic plasticity but has no effect on normal synaptic transmission ^[2] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.			
In Vivo	Rp-cAMPS (10 μM, 15 min) decreases the monosynaptic EPSCs evoked at the PB-CeLC and BLA-CeLC synapses in slices from arthritic rats but not in control neurons from normal animals. The inhibitory effect of Rp-cAMPS is significant compared to			

Product Data Sheet



predrug (ACSF) control values obtained in the same neurons^[2].

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

CUSTOMER VALIDATION

• Theranostics. 2021 Mar 24;11(12):5650-5674.

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REFERENCES

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[2]. Fu Y, et al. PKA and ERK, but not PKC, in the amygdala contribute to pain-related synaptic plasticity and behavior. Mol Pain. 2008 Jul 16;4:26.

[3]. Kuriyama S, et al. Isoproterenol inhibits rod outer segment phagocytosis by both cAMP-dependent and independent pathways. Invest Ophthalmol Vis Sci. 1995 Mar;36(3):730-6.

[4]. Dostmann WR, et al. Probing the cyclic nucleotide binding sites of cAMP-dependent protein kinases I and II with analogs of adenosine 3',5'-cyclic phosphorothioates. J Biol Chem. 1990 Jun 25;265(18):10484-91.

[5]. Van Haastert PJ, et al. Competitive cAMP antagonists for cAMP-receptor proteins. J Biol Chem. 1984 Aug 25;259(16):10020-4.

[6]. R J de Wit, et al. Inhibitory action of certain cyclophosphate derivatives of cAMP on cAMP-dependent protein kinases. Eur J Biochem. 1984 Jul 16;142(2):255-60.

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