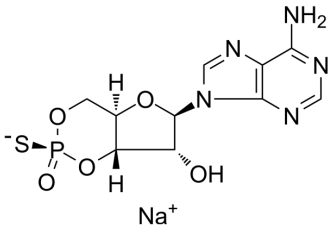
Sp-cAMPS sodium salt

Cat. No.:	HY-100530C
CAS No.:	142439-95-0
Molecular Formula:	$C_{10}H_{11}N_5NaO_5PS$
Molecular Weight:	367.25
Target:	PKA; Phosphodiesterase (PDE)
Pathway:	Stem Cell/Wnt; Metabolic Enzyme/Protease
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

In Vitro

DMSO : 100 mg/mL (272.29 mM; Need ultrasonic)

H_2O : 86.67 mg/mL (236.00 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 one by one: PBS
   Solubility: 50 mg/mL (136.15 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (6.81 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.81 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.81 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Sp-cAMPS sodium salt, a cAMP analog, is potent activator of cAMP-dependent PKA I and PKA II. Sp-cAMPS sodium salt is also a potent, competitive phosphodiesterase (PDE3A) inhibitor with a K_i of 47.6 μM. Sp-cAMPS sodium salt binds the PDE10 GAF domain with an EC_{50} of 40 μM[1][2][3].

IC_{50} & Target	PKA I	PKA II	PDE3A	PDE10 GAF domain

			47.6 μM (Ki)	50 μM (EC50)
In Vitro	Treatment of hepatocytes with Sp-cAMPS sodium salt, the stimulatory diastereomer of adenosine cyclic 3',5'-phosphorothioate, mimics the response seen with glucagon. The glucagon-stimulated increases in the level of Ca^{2+} can be mimicked by Sp-cAMPS sodium salt[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.			
In Vivo	In chronic alcohol consumption (CAC) mice, direct infusion of the Sp-cAMPS (1 μg/μL) sodium salt into the prefrontal cortex significantly improves or impairs, respectively, working memory performance in withdrawn and water animals[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.			

REFERENCES

[1]. Su H Hung, et al. A new nonhydrolyzable reactive cAMP analog, (Sp)-adenosine-3',5'-cyclic-S-(4-bromo-2,3-dioxobutyl)monophosphorothioate irreversibly inactivates human platelet cGMP-inhibited cAMP phosphodiesterase. Bioorg Chem. 2002 Feb;30(1):16-31.

[2]. L Y Wang, et al. Regulation of kainate receptors by cAMP-dependent protein kinase and phosphatases. Science. 1991 Sep 6;253(5024):1132-5.

[3]. Ronald Jäger, et al. Activation of PDE10 and PDE11 phosphodiesterases. J Biol Chem. 2012 Jan 6;287(2):1210-9.

[4]. P A Connelly,et al. A study of the mechanism of glucagon-induced protein phosphorylation in isolated rat hepatocytes using (Sp)-cAMPS and (Rp)-cAMPS, the stimulatory and inhibitory diastereomers of adenosine cyclic 3',5'-phosphorothioate. J Biol Chem. 1987 Mar 25;262(9):4324-32.

[5]. G Dominguez, et al. Rescuing prefrontal cAMP-CREB pathway reverses working memory deficits during withdrawal from prolonged alcohol exposure. Brain Struct Funct. 2016 Mar;221(2):865-77.

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