Dibutyryl-cGMP sodium

Cat. No.: HY-130354

CAS No.: 51116-00-8

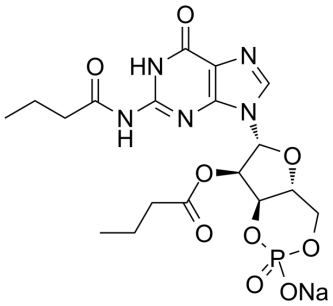
Molecular Formula: $C_{18}H_{23}N_5NaO_9P$

Molecular Weight: 507.37

Target: Potassium Channel

Pathway: Membrane Transporter/Ion Channel

Storage: -20°C, stored under nitrogen, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (197.09 mM; Need ultrasonic)
DMSO : 100 mg/mL (197.09 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9709 mL	9.8547 mL	19.7095 mL
	5 mM	0.3942 mL	1.9709 mL	3.9419 mL
	10 mM	0.1971 mL	0.9855 mL	1.9709 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (4.93 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.93 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.93 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Dibutyryl-cGMP sodium (Bt2cGMP sodium) is a cell-permeable cGMP analogue. Dibutyryl-cGMP sodium preferentially activates cGMP-dependent protein kinase (PKG). Dibutyryl-cGMP sodium inhibits the release of [^{3}H]-arachidonic acid from γ thrombin-stimulated human platelets. Dibutyryl-cGMP sodium induces peripheral antinociception via activation of ATP-sensitive K^+ channels[1][2][3].

IC_{50} & Target

cGMP-dependent protein kinase (PKG)[1];
ATP-sensitive K^+ channels[3]

In Vitro	Dibutyryl-cGMP is able to induce process elongation and branching in astrocytes resulting from a rapid, reversible and concentration-dependent redistribution of glial fibrillary acidic protein (GFAP) and actin filaments without significant change in protein levels[1]. When cells are co-incubated with Dibutyryl-cGMP (100 μM) stress fibre formation is prevented and cells acquired a stellate morphology in cerebellar astrocytes[1]. In cells treated with Dibutyryl-cGMP (100 μM, 2 h) the particulate fraction is nearly devoid of RhoA protein. Dibutyryl-cGMP prevents RhoA-membrane association[1]. Using the scratchwound model, the size of the wound is significantly smaller in cells treated with Dibutyryl-cGMP after the wound indicating that dbcGMP accelerates wound closure[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Dibutyryl-cGMP (50-200 μg/paw; subcutaneous injection; male Wistar rats) treatment antagonizes the hyperalgesic effect of PGE2 in a dose-dependent manner. Maximal antinociceptive effect of DbcGMP is at 1 h after administration and last for plus 2 h[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Wistar rats (180- 250 g) injection with Prostaglandin E2 (PGE2)[3]
Dosage:	50 μg/paw, 75 μg/paw, 100 μg/paw and 200 μg/paw
Administration:	Subcutaneous injection
Result:	Antagonized the hyperalgesic effect of PGE2 (2 μg/paw), in a dose-dependent manner.

CUSTOMER VALIDATION

- Cell Oncol. 2023 Mar 20.

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REFERENCES

[1]. Borán MS, et al. The cyclic GMP-protein kinase G pathway regulates cytoskeleton dynamics and motility in astrocytes. J Neurochem. 2007 Jul;102(1):216-30.

[2]. Sane DC, et al. Cyclic GMP analogs inhibit gamma thrombin-induced arachidonic acid release in human platelets. Biochem Biophys Res Commun. 1989 Dec 15;165(2):708-14.

[3]. Soares AC, et al. Dibutyryl-cyclic GMP induces peripheral antinociception via activation of ATP-sensitive K(+) channels in the rat PGE2-induced hyperalgesic paw. Br J Pharmacol. 2001 Sep;134(1):127-31.

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