8-Bromo-cGMP sodium

Cat. No.: HY-101379A

CAS No.: 51116-01-9

Molecular Formula: $C_{10}H_{10}BrN_5NaO_7P$

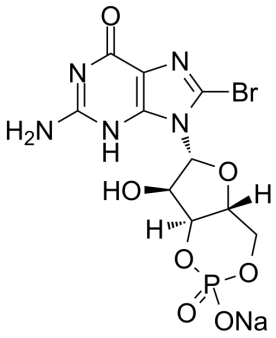
Molecular Weight: 446.08

Target: Calcium Channel

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: -20°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (224.18 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2418 mL	11.2088 mL	22.4175 mL
5 mM	0.4484 mL	2.2418 mL	4.4835 mL
10 mM	0.2242 mL	1.1209 mL	2.2418 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (224.18 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

8-Bromo-cGMP sodium, a membrane-permeable analogue of cGMP, is a PKG (protein kinase G) activator. 8-Bromo-cGMP sodium significantly inhibits Ca^{2+} macroscopic currents and impairs insulin release stimulated with high K^+[1]. 8-Bromo-cGMP sodium has antinociceptive effects and results in vasodilator responses[2].

In Vitro

8-Bromo-cGMP sodium (1-100 μM; 8 h) increases resistance of LLC-PK1 cells to CsA toxicity concentration-dependently[3].
8-Bromo-cGMP sodium (1-100 μM; 16 h) induces the synthesis of HO-1 protein in a concentration-dependent fashion[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[3]

Cell Line:	LLC-PK1 cells (ATCC CL 101)
Concentration:	1-100 μM
Incubation Time:	8 hours

Result:	Increased resistance of LLC-PK1 cells to Cyclosporin A (CsA) toxicity concentration-dependently and augmented cell viability by up to 65%.

Western Blot Analysis[3]

Cell Line:	LLC-PK1 cells (ATCC CL 101)
Concentration:	1-100 μM
Incubation Time:	16 hours
Result:	Induced the synthesis of HO-1 protein in a concentration-dependent fashion.

In Vivo

8-Bromo-cGMP sodium (0.3, 1, 3.0 nmol; intrathecal administration; 10 min before test) dose-dependently and significantly increases the tail-flick latency in Vincristine-treated mice to the level observed in vehicle-treated naive mice (male ICR mice, 4weeks of age and weighing 20 g). Vincristine (0.05 mg/kg 1 day after the pre-drug tail-flick latency, and then 0.125 mg/kg twice a week for 6 weeks) can induce painful neuropathy in mice[4].

8-Bromo-cGMP sodium (10 mg/kg; iv; single dose) results in vasodilator responses in eNOS-Tg mice and WT littermates in C57BL/6 background (19-35 g)[5].

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REFERENCES

[1]. Sarkar O , et al. Nitric oxide attenuates overexpression of Giα proteins in vascular smooth muscle cells from SHR: Role of ROS and ROS-mediated signaling. PLoS One. 2017 Jul 10;12(7):e0179301.

[2]. Tegeder I , et al. Dual effects of spinally delivered 8-bromo-cyclic guanosine mono-phosphate (8-bromo-cGMP) in formalin-induced nociception in rats. Neurosci Lett. 2002 Oct 31;332(2):146-50.

[3]. Tobias Polte, et al. Atrial natriuretic peptide reduces cyclosporin toxicity in renal cells: role of cGMP and heme oxygenase-1. Free Radic Biol Med. 2002 Jan 1;32(1):56-63.

[4]. Junzo Kamei, et al. Possible involvement of the spinal nitric oxide/cGMP pathway in vincristine-induced painful neuropathy in mice. Pain. 2005 Sep;117(1-2):112-20.

[5]. Elza D van Deel, et al. Vasomotor control in mice overexpressing human endothelial nitric oxide synthase. Am J Physiol Heart Circ Physiol. 2007 Aug;293(2):H1144-53.

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