CHPG sodium salt

Cat. No.: HY-101364A

CAS No.: 1303993-73-8

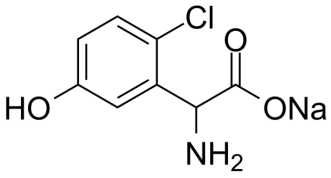
Molecular Formula: $C_8H_7ClNNaO_3$

Molecular Weight: 223.59

Target: mGluR; NF-κB; ERK; Akt

Pathway: GPCR/G Protein; Neuronal Signaling; NF-κB; MAPK/ERK Pathway; Stem Cell/Wnt; PI3K/Akt/mTOR

Storage: 4°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

DMSO : 100 mg/mL (447.25 mM; Need ultrasonic)
H_2O : 6.67 mg/mL (29.83 mM; ultrasonic and warming and heat to 80°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.4725 mL	22.3624 mL	44.7247 mL
	5 mM	0.8945 mL	4.4725 mL	8.9449 mL
	10 mM	0.4472 mL	2.2362 mL	4.4725 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 5 mg/mL (22.36 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (11.18 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (11.18 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (11.18 mM); Clear solution

BIOLOGICAL ACTIVITY

Description CHPG sodium salt is a selective mGluR5 agonist, and attenuates SO_2-induced oxidative stress and inflammation through TSG-6/NF-κB pathway in BV2 microglial cells[1]. CHPG sodium salt protects against traumatic brain injury (TBI) in vitro and in vivo by activation of the ERK and Akt signaling pathways.[2].

IC_{50} & Target	$mGlu_5$	NF-κB	ERK

In Vitro

CHPG sodium salt (10-500 μM; 24 hours) significantly increases the cell viability and decreases the LDH release after SO_2 derivatives treatment[1].
CHPG sodium salt (0.5 mM; 30 mins) protects BV2 cells against SO_2-induced apoptosis[1].
CHPG sodium salt (0.5 mM; 30 mins) treatment alone increases the expression of TSG-6 in both mRNA and protein levels[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	BV2 microglial cells
Concentration:	10, 50, 100 and 500 μM
Incubation Time:	24 hours
Result:	Increased the cell viability.

Apoptosis Analysis[1]

Cell Line:	BV2 microglial cells
Concentration:	0.5 mM
Incubation Time:	30 mins
Result:	Protected BV2 cells against SO_2-induced apoptosis.

Western Blot Analysis[1]

Cell Line:	BV2 microglial cells
Concentration:	0.5 mM
Incubation Time:	30 mins
Result:	Increased the expression of TSG-6 in both mRNA and protein levels.

In Vivo

CHPG sodium salt (injection; 250 nM; for 7 days) reduces significantly cerebral lesion volume[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Adult Sprague-Dawley male rats weighing 280-320 g[2]
Dosage:	250 nM
Administration:	Injection; for 7 days
Result:	Reduced significantly cerebral lesion volume.

REFERENCES

[1]. Qiu JL, et al. The selective mGluR5 agonist CHPG attenuates SO_2-induced oxidative stress and inflammation through TSG-6/NF-κB pathway in BV2 microglial cells. Neurochem Int. 2015 Jun-Jul;85-86:46-52.

[2]. Chen T, et al. The selective mGluR5 agonist CHPG protects against traumatic brain injury in vitro and in vivo via ERK and Akt pathway. Int J Mol Med. 2012 Apr;29(4):630-6.

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

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA