

Product Data Sheet

Product Name: CHPG
Cat. No.: GC12532

Chemical Properties

Cas. No. 170846-74-9

Chemical Name (S)-2-amino-2-(2-chloro-5-hydroxyphenyl)acetic acid

SMILES C1C(C=C1)=C(C=C1O)[C@@H](C(O)=O)N

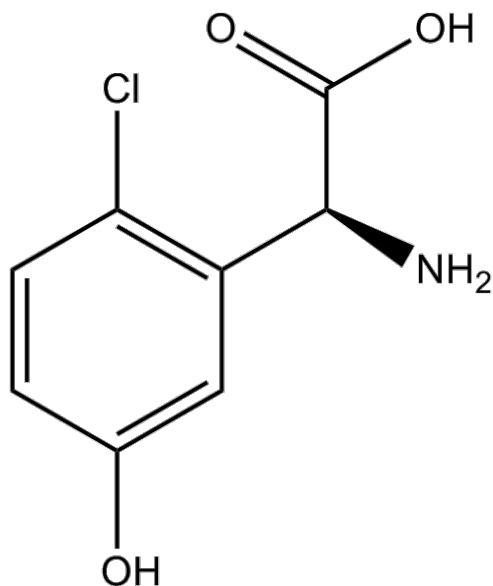
Formula $C_8H_8ClNO_3$ M.Wt 201.6

Solubility 1 M NaOH: Soluble Storage $-20^{\circ}C$

General tips For obtaining a higher solubility, please warm the tube at $37^{\circ}C$ and shake it in the ultrasonic bath for a while. Stock solution can be stored below $-20^{\circ}C$ for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

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

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Cell lines	BV2 microglial cells
Preparation Method	BV2 cells were pretreated with CHPG (0.5mM) for 30min before 100μM SO ₂ treatment. Cell viability was measured by WST-1 assay and cytotoxicity was measured by LDH assay. Apoptotic cell death was detected by TUNEL staining, and apoptotic rate was calculated.
Reaction Conditions	0.5mM; 30min
Applications	CHPG alone has no obvious cytotoxicity in BV2 cells. CHPG significantly increased the cell viability and decreased the LDH release after SO ₂ derivatives treatment. CHPG protected BV2 cells against SO ₂ -induced apoptosis.

Animal experiment [2]:

Animal models	Sprague-Dawley male rats
Preparation Method	Rats were randomly divided into the following four groups, the vehicle group, the CHPG group, the TBI group and the TBI+CHPG group. Vehicle (1% DMSO in saline), CHPG (250nM) was injected in a volume of 5μl into right lateral ventricle (anteroposterior, 0.8mm; lateral, 1.5mm; depth, 3.5mm from bregma) 30min before traumatic brain injury (TBI).
Dosage form	250nM, 5μl; injected into right lateral ventricle

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Applications

There were no obvious TUNEL-positive cells in the vehicle group or the CHPG group. However, 24h after TBI, the number of TUNEL-positive cells significantly increased (117 ± 5 for the TBI group). Pre-treatment with 250nM of CHPG attenuated this increase to 63 ± 7 for the TBI+CHPG group. Cerebral lesion volume of the TBI+CHPG group was significantly smaller than that of the TBI group.

References:

- [1] Qiu, Jun-Ling et al. "The selective mGluR5 agonist CHPG attenuates SO₂-induced oxidative stress and inflammation through TSG-6/NF- κ B pathway in BV2 microglial cells." *Neurochemistry international* vol. 85-86 (2015): 46-52.
- [2] Chen, Tao et al. "The selective mGluR5 agonist CHPG protects against traumatic brain injury in vitro and in vivo via ERK and Akt pathway." *International journal of molecular medicine* vol. 29,4 (2012): 630-6.

Background

CHPG is a selective metabotropic glutamate receptor 5 (mGlu5) agonist^[1]. Activation of mGlu5 suppresses oxidative stress and inflammation induced by lipopolysaccharide (LPS) or SO₂ derivatives in microglia, thereby inhibiting neuronal apoptosis^[1].

In vitro, CHPG (0.5mM; 30min) pretreatment showed no obvious cytotoxicity in BV2 cells, and significantly increased cell viability while reducing LDH release and apoptosis following SO₂ derivatives treatment^[1]. CHPG (0.5mM; 30min) attenuated SO₂-induced oxidative stress, the expression of inflammatory cytokines, like IL-1 β , IL-6 and TNF- α , in BV2 microglial cells^[1]. CHPG (1mM; 30min) protected cultured cortical neurons against traumatic brain injury (TBI) and reduced the neuronal apoptotic rate^[2].

In vivo, CHPG (250nM, 50 μ l; injected into the right lateral ventricle; 30min pretreatment) enhanced the activation of ERK and Akt, attenuated neuronal apoptosis, and reduced cerebral lesion volume in TBI rats^[2].

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[1] Qiu, Jun-Ling et al. "The selective mGluR5 agonist CHPG attenuates SO₂-induced oxidative stress and inflammation through TSG-6/NF- κ B pathway in BV2 microglial cells." *Neurochemistry international* vol. 85-86 (2015): 46-52.

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