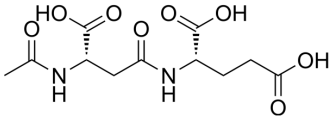
β-Spaglumic acid

Cat. No.:	HY-130553
CAS No.:	4910-46-7
Molecular Formula:	$C_{11}H_{16}N_2O_8$
Molecular Weight:	304.25
Target:	Aminopeptidase; mGluR
Pathway:	Metabolic Enzyme/Protease; GPCR/G Protein; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	β-Spaglumic acid (β-NAAG) is a competitive NAAG peptidase inhibitor (K_i=1 μM) that protects spinal cord neurons from excitotoxicity and hypoxic damage. β-Spaglumic acid is also a selective mGluR3 antagonist (mGluR3 receptor functions to regulate activity-dependent synaptic potentiation in the hippocampus). β-Spaglumic acid can be used in neuroprotection-related studies[1][2].
IC_{50} & Target	NAAG peptidase, mGluR3[1][2].
In Vitro	β-Spaglumic acid (63-1000 μM; 2 h) protects against NMDA-induced injury of spinal cord cells in a dose-dependent manner[1]. β-Spaglumic acid (0-1000 μM; 2 h) protects spinal cord cells against hypoxia[1]. β-Spaglumic acid (500 μM) significantly reduces intraneuronal free Ca^{2+} responses upon neuronal exposure to 25 μM NMDA[1]. β-Spaglumic acid (100 μM; 7 min) antagonizes mGluR3 in cerebellar granule cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Spinal cord cells (NMDA-induced) (from spinal cords removed from prenatal day 15 Sprague-Dawley rat fetuses)
Concentration:	63-1000 μM
Incubation Time:	2 h
Result:	Led to a significant attenuation of NMDA toxicity at a concentration of 63 μM and completely blocked NMDA toxicity with 500 and 1000 μM concentrations. Apparently minimized the basal loss of cell viability associated with experimental handling of the cells (e.g. serum removal, media changes).

Cell Viability Assay[1]

Cell Line:	Spinal cord cells (hypoxic-induced)
Concentration:	0-1000 μM

Incubation Time:	2 h
Result:	Provided 75% protection during hypoxia when at 8 μM and completely eliminated hypoxia-induced loss of viability (107.4-114.4% protection, respectively) when at 63-1000 μM.

Cell Viability Assay[2]

Cell Line:	Cerebellar granule cells (expressing group I-III mGluRs)
Concentration:	100 μM
Incubation Time:	7 min
Result:	Blocked NAAG inhibition of forskolin-stimulated cAMP formation via mGluR3.

REFERENCES

[1]. Yourick DL, et al. N-acetylaspartylglutamate and beta-NAAG protect against injury induced by NMDA and hypoxia in primary spinal cord cultures. Brain Res. 2003 Nov 21;991(1-2):56-64.

[2]. Wroblewska B, et al. β-NAAG rescues LTP from blockade by NAAG in rat dentate gyrus via the type 3 metabotropic glutamate receptor. J Neurophysiol. 2001 Mar;85(3):1097-106.

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