DL-AP3

Cat. No.: HY-100786

CAS No.: 5652-28-8

Molecular Formula: $C_3H_8NO_5P$

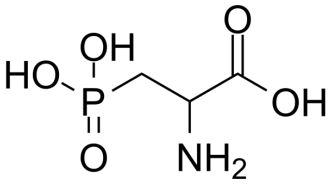
Molecular Weight: 169.07

Target: Endogenous Metabolite; mGluR; Phosphatase

Pathway: Metabolic Enzyme/Protease; GPCR/G Protein; 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 : 5 mg/mL (29.57 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	5.9147 mL	29.5735 mL	59.1471 mL
	5 mM	1.1829 mL	5.9147 mL	11.8294 mL
	10 mM	0.5915 mL	2.9574 mL	5.9147 mL

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

BIOLOGICAL ACTIVITY

Description

DL-AP3 is a competitive mGluR1 and mGluR5 antagonist. DL-AP3 is also an inhibitor of phosphoserine phosphatase. DL-AP3 has neuroprotective effect[1][2][3].

IC_{50} & Target	mGluR 1	mGluR 5

In Vitro

DL-AP3 (10 μM, 6 h) alleviates oxygen-glucose deprivation (OGD)-induced injury (cell viability) in primary neurons[1].

DL-AP3 (10 μM, 6 h) recovers the decreased levels of p-Akt1 and the increase of cytochrome C induced by OGD in primary neurons[1].

DL-AP3 (1-100 μM) inhibits activity of rat brain phosphoserine phosphatase, with an IC_{50} of 187 μM and K_i of 77 μM[2].

DL-AP3 (10 μM, 10 min) together with SKF81297 (5 μM) induces a significant Long-term potentiation (LTP) in the slices of Fmr1 KO mice[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Primary neurons with oxygen-glucose deprivation (OGD) treatment

Concentration:	10 μM
Incubation Time:	24 h or 72 h
Result:	Attenuated the inhibitory effect of OGD on neuronal viability.

Western Blot Analysis[1]

Cell Line:	Primary neurons with oxygen-glucose deprivation (OGD) treatment
Concentration:	10 μM
Incubation Time:	6 h
Result:	Increased the decreased levels of p-Akt1, and decreased the increase of cytochrome C.

In Vivo

DL-AP3 (4 mg/kg, i.p., for 5 weeks) with SKF81297 (1 mg/kg, i.p.) reduces the hyperactivity phenotype in Fmr1 KO mice[3].
DL-AP3 (4.0-12.0 mg/animal, i.c.v. infusion, 100 μL) blocks development of visceral pain symptoms and neuroendocrinological changes in the blood plasma of sheep[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Fmr1 KO mice[3]
Dosage:	4 mg/kg with SKF81297 (1 mg/kg)
Administration:	i.p., for 5 weeks.
Result:	Reduced the distance traveled by Fmr1 KO mice (open-field test). Reduced the swim latency on the final day in the Fmr1 KO mice (Morris Water Maze test).

Animal Model:	Sheep with visceral pain evoked by colonic distension (CD)[4]
Dosage:	4.0-12.0 mg/animal
Administration:	i.c.v. infusion, 100 μL
Result:	Decreased intensity from appearance of clinical signs of visceral pain caused by CD test. Diminished the increase of plasma cortisol, E, NE and DA concentrations caused by visceral pain provoked by CD episode.

REFERENCES

[1]. Cui D, et al. DL-2-amino-3-phosphonopropionic acid protects primary neurons from oxygen-glucose deprivation induced injury. Bosn J Basic Med Sci. 2017 Feb 21;17(1):12-16.

[2]. Hawkinson JE, et al. The metabotropic glutamate receptor antagonist L-2-amino-3-phosphonopropionic acid inhibits phosphoserine phosphatase. Eur J Pharmacol. 1996 Jun 27;307(2):219-25.

[3]. Xu ZH, et al. Group I mGluR antagonist rescues the deficit of D1-induced LTP in a mouse model of fragile X syndrome. Mol Neurodegener. 2012 May 28;7:24.

[4]. B.F. Kania, et al. Supraspinal basis of analgesic and clinical effects of the metabotropic glutamate mGluR1 antagonist during colonic distension in sheep. Small Ruminant Research. 2014. 117 (1).

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

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