MSOP

Cat. No.: HY-101226

CAS No.: 66515-29-5

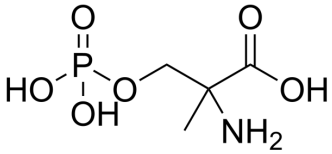
Molecular Formula: $C_4H_{10}NO_6P$

Molecular Weight: 199.1

Target: mGluR

Pathway: GPCR/G Protein; Neuronal Signaling

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

H_2O : 62.5 mg/mL (313.91 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	5.0226 mL	25.1130 mL	50.2260 mL
	5 mM	1.0045 mL	5.0226 mL	10.0452 mL
	10 mM	0.5023 mL	2.5113 mL	5.0226 mL

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

BIOLOGICAL ACTIVITY

Description

MSOP is a selective group III metabotropic glutamate receptor antagonist with apparent K_D of 51 μM for the L-AP4-sensitive presynaptic mGluR.

IC_{50} & Target

K_D: 51 μM (L-AP4-sensitive presynaptic mGluR)[1].

In Vitro

In the presence of 200 μM MSOP, a rightward parallel shift of the dose-response curve to L-AP4 is observed, with an apparent K_D calculated as 51±6 μM (n=3). MSOP is shown to be selective for the L-APC sensitive presynaptic mGluR, the apparent K_D for the interaction of MSOP with the (1S, 3S)-ACPD sensitive receptor calculated as greater than 700 μM (n=3)[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

It is found that TBOA-induced antinociceptive effects are significantly blocked by intrathecal co-administration of MSOP (second phase of formalin model: $F_{3,16}$=30.96, P<0.001; CFA model: $F_{3,16}$=30.77, P<0.001). As expected, intrathecal TBOA (10 μg) reduces the number of formalin-induced flinches and shakes by 47% of the value in the saline-treated group in the second phase (P<0.001) and blocked the CFA-induced decrease in ipsilateral paw withdrawal latency by 60% of the value in the saline-treated group (P=0.01). The number of formalin-induced flinches in the second phase in the group treated with MSOP and TBOA is increased by 56% (P=0.04) of the value in the TBOA-treated group. CFA-induced paw withdrawal latency

in the group treated with MSOP and TBOA is decreased by 86% (P=0.03) of the value in the TBOA-treated group[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [2]

Rats[2]
Male Sprague-Dawley rats (250-300 g) are housed individually in cages on a standard 12 h-12 h light-dark cycle. Water and food are available as libitum until rats are transported to the labotatory approximately 1 h before the experiments. A glutamate transporter activator, three glutamate transporter inhibitors, TBOA, DL-THA, dihydrokainate, and a selective group III mGluR antagonist MSOP are used. All drugs are dissolved in 0.9% physiological saline. To examine the role of group III mGluRs in the antinociceptive effect produced by intrathecal TBOA in the formalin model, the rats are intrathecally injected with saline (10 μL; n=5), MSOP (10 μg/10 μL; n=5), TBOA (10 μg/10 μL; n=5), or MSOP plus TBOA (n=5). Ten minutes later, 2% formalin (100 μL) is injected into the plantar side of a hind paw and formalin-induced pain behaviors are assessed.To examine the role of group III mGluRs in the antinociceptive effect produced by intrathecal TBOA in the complete Freund's adjuvant (CFA) model, the rats are intrathecally injected with saline (10 μL; n=5), MSOP (10 μg/10 μL; n=5), TBOA (10 μg/10 μL; n=5), or MSOP plus TBOA (n=5) at 6 h post-CFA and then measured paw withdrawal latencies[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Thomas NK, et al. alpha-Methyl derivatives of serine-O-phosphate as novel, selective competitive metabotropic glutamate receptor antagonists. Neuropharmacology. 1996 Jun;35(6):637-42.

[2]. Myron Yaster, et al. Effect of inhibition of spinal cord glutamate transporters on inflammatory pain induced by formalin and complete Freund's adjuvant. Anesthesiology. 2011 Feb; 114(2): 412–423.

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