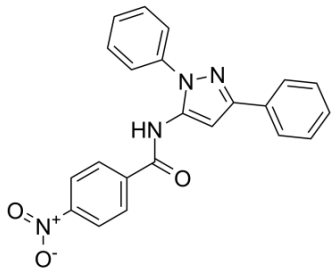
VU-29

Cat. No.:	HY-107508
CAS No.:	890764-36-0
Molecular Formula:	$C_{22}H_{16}N_4O_3$
Molecular Weight:	384.39
Target:	mGluR
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (130.08 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6015 mL	13.0076 mL	26.0152 mL
	5 mM	0.5203 mL	2.6015 mL	5.2030 mL
	10 mM	0.2602 mL	1.3008 mL	2.6015 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: 5 mg/mL (13.01 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

VU-29 is a positive allosteric modulator of metabotropic glutamate 5 (mGlu5) receptor (EC_{50}=9 nM and K_i=244 nM for rmGluR5). VU-29 is selective for mGluR5 relative to other mGluR subtypes (EC_{50}: rmGluR1/rmGluR2=557 nM/1.5 μM; hmGluR4=154 nM)[1][2].

IC_{50} & Target

rat mGluR5 9 nM (EC50)	rat mGluR1 557 nM (EC50)	rat mGluR2 1.5 μM (EC50)	hmGluR4 154 nM (EC50)
rat mGluR5 224 nM (Ki)			

In Vivo

VU-29 (500 nM) potentiates DHPG induced increases in phosphoinositide (PI) hydrolysis in rat hippocampal slices. VU-29

potentiates threshold TBS-induced long term potentiation (LTP) in rat hippocampal CA1 region. VU-29 (1 μM) potentiates chemically induced mGluR-long term depression (LTD) in area CA1 of the rat hippocampus. VU-29 (1 μM) potentiates stimulus-induced NMDA receptor-independent LTD[1].

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

REFERENCES

[1]. Ayala JE, et al. mGluR5 positive allosteric modulators facilitate both hippocampal LTP and LTD and enhance spatial learning. Neuropsychopharmacology. 2009;34(9):2057-2071.

[2]. Chen Y, et al. Interaction of novel positive allosteric modulators of metabotropic glutamate receptor 5 with the negative allosteric antagonist site is required for potentiation of receptor responses. Mol Pharmacol. 2007;71(5):1389-1398.

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