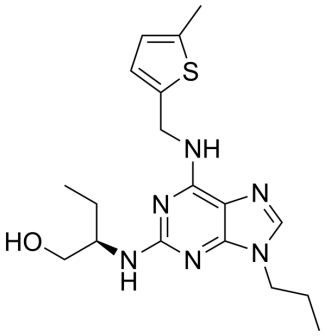
GV-58

Cat. No.:	HY-12498
CAS No.:	1402821-41-3
Molecular Formula:	$C_{18}H_{26}N_6OS$
Molecular Weight:	374.5
Target:	Calcium Channel
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 75 mg/mL (200.27 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6702 mL	13.3511 mL	26.7023 mL
5 mM	0.5340 mL	2.6702 mL	5.3405 mL
10 mM	0.2670 mL	1.3351 mL	2.6702 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: ≥ 2.5 mg/mL (6.68 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.68 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

GV-58 is a novel N- and P/Q-type calcium (Ca^{2+}) channel agonist with EC_{50}s of 7.21 and 8.81 μM, respectively. GV-58 slows the deactivation of channels, resulting in a large increase in presynaptic Ca^{2+} entry during activity. GV-58 can be used in lambert-eaton myasthenic syndrome (LEMS) research[1][2][3].

IC_{50} & Target

N-type calcium channel	P/Q-type calcium channel
7.21 μM (EC50)	8.81 μM (EC50)

In Vitro

GV-58 (50 μM; 30 min) restores function in LEMS passive transfer neuromuscular junction[1].

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

Cell Viability Assay[1]

Cell Line:	Upper arm muscle isolated from LEMS mice
Concentration:	50 μM
Incubation Time:	30 min
Result:	Increased the mEPP frequency from 3.27 s^{-1} in vehicle controls to 10.45 s^{-1}. Showed a slight facilitation followed by depression to ⊠94% at the final EPP in the train.

REFERENCES

[1]. Tarr TB, et al. Evaluation of a novel calcium channel agonist for therapeutic potential in Lambert-Eaton myasthenic syndrome. J Neurosci. 2013 Jun 19;33(25):10559-67.

[2]. Tarr TB, et al. Complete reversal of Lambert-Eaton myasthenic syndrome synaptic impairment by the combined use of a K+ channel blocker and a Ca2+ channel agonist. J Physiol. 2014 Aug 15;592(16):3687-96.

[3]. Meriney SD, et al. Lambert-Eaton myasthenic syndrome: mouse passive-transfer model illuminates disease pathology and facilitates testing therapeutic leads. Ann N Y Acad Sci. 2018 Jan;1412(1):73-81.

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