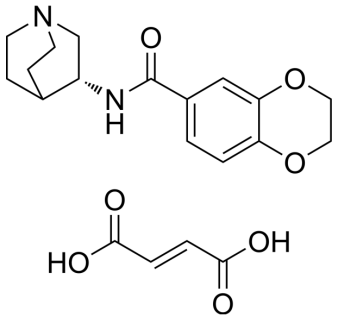
PHA 568487

Cat. No.:	HY-107666
CAS No.:	527680-57-5
Molecular Formula:	$C_{20}H_{24}N_2O_7$
Molecular Weight:	404.41
Target:	nAChR
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (618.18 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4727 mL	12.3637 mL	24.7274 mL
	5 mM	0.4945 mL	2.4727 mL	4.9455 mL
	10 mM	0.2473 mL	1.2364 mL	2.4727 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: 2.08 mg/mL (5.14 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.14 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.14 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

PHA 568487 a selective agonist of alpha-7 nicotinic acetylcholine receptor (α-7 nAchR)[1][2].PHA 568487 reduces neuroinflammation and oxidative stress[2]. PHA-568487 has rapid brain penetration[3].

In Vitro

PHA 568487 increases anti-oxidant gene expression and decreases oxidative stress and phosphorylation of NF-κb p65. Methyllycaconitine (MLA) has the opposite effects[2].
PHA increases anti-oxidant genes and NADPH oxidase expression associated with decreased phosphorylation of NF-kB p65 in microglia/macrophages[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

PHA 568487 attenuates neuronal injury and behavioral dysfunction in mice with ischemic stroke only and ischemic stroke plus tibia fracture[2].

PHA 568487 (1.25 mg/kg; i.p.; treated daily)-treated ischemic rats shows a significant reduction of the cerebral infarct volumes and an improvement of the neurologic outcome[4].

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

Animal Model:	C57BL/6J male mice (10-12 weeks old)[2]
Dosage:	PHA 568487 (PHA; 0.4 and 0.8 mg/kg); Methyllycaconitine (MLA; 4 and 6 mg/kg)
Administration:	Injected intraperitoneally once on day 1, or twice on days 1 and 2, after pMCAO
Result:	Injection of PHA (0.8 mg/kg) and MLA (6 mg/kg) on days 1 and 2 after pMCAO yielded the best effect on infarct volume and behavior tests.

Animal Model:	Adult male Sprague-Dawley rats (297 6±8.3 g)[4]
Dosage:	1.25 mg/kg
Administration:	I.p.;0.1 mL; treated daily
Result:	Showed a significant reduction of the cerebral infarct volumes and an improvement of the neurologic outcome.

REFERENCES

[1]. F Barclay Shilliday, et al. Multiple species metabolism of PHA-568487, a selective alpha 7 nicotinic acetylcholine receptor agonist. Drug Metab Lett. 2010 Aug;4(3):162-72.

[2]. Zhenying Han, et al. Alpha-7 nicotinic acetylcholine receptor agonist treatment reduces neuroinflammation, oxidative stress, and brain injury in mice with ischemic stroke and bone fracture. J Neurochem. 2014 Nov;131(4):498-508.

[3]. Dingquan Zou, et al.Activation of Alpha-7 Nicotinic Acetylcholine Receptor Reduces Brain Edema in Mice with Ischemic Stroke and Bone Fracture. Mol Neurobiol. 2017 Dec;54(10):8278-8286.

[4]. Lorena Colás, et al. In vivo imaging of A7 nicotinic receptors as a novel method to monitor neuroinflammation after cerebral ischemia. Glia. 2018 Aug;66(8):1611-1624.

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