Amifampridine phosphate

Cat. No.: HY-14946A

CAS No.: 446254-47-3

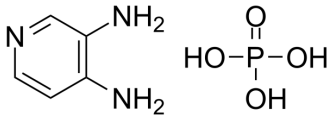
Molecular Formula: $C_5H_{10}N_3O_4P$

Molecular Weight: 207.12

Target: Potassium Channel

Pathway: Membrane Transporter/Ion Channel

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Amifampridine (3,4-Diaminopyridine) phosphate is an orally active, potent and cell permeable voltage-gated potassium (K_V) channel blocker (PCB). Amifampridine phosphate is efficacy in the reversal of BoNT/A (HY-P79153) intoxication. Amifampridine phosphate increases transmitter release from neuromuscular junctions (NMJs). Amifampridine phosphate can be used for Lambert-Eaton myasthenic syndrome (LEMS) research[1][2][3].

In Vitro

Amifampridine phosphate (1.5 μM) significantly reduces Kv3.3 and Kv3.4 currents by about 10% in HEK293T cells, has no effect on Cav2.1 or Cav1.2 current[3].

Amifampridine phosphate (0-100 μM) increases the duration of the presynaptic AP (action potential) waveform at mammalian and frog NMJs in a dose-dependent manner[3].

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

In Vivo

Amifampridine phosphate (Oral gavage; 10 mg/kg; once) can antagonize muscle paralysis following BoNT/A intoxication[2].

Amifampridine phosphate (2.5 mg/kg (IV); 10 mg/kg (PO); once) shows 1 hour plasma half-life and about 57% bioavailability (F) in mice[2].

Amifampridine phosphate has a short plasma half-life and can induce seizures when present at high concentrations, following penetration of the blood-brain barrier[2].

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

Animal Model:	CD-1 mouse (female,25 g, 6 weeks old)[2]
Dosage:	10 mg/kg
Administration:	Oral gavage, once, after BoNT/A administration (IP)
Result:	Revealed that neither LEMs alone (182 ± 43 min) nor the maximum safe orally deliverable dose of 3,4-DAP alone (225 ± 24 min) could significantly increase the time to death following toxin administration (216 ± 29 min). However, when the 10/50/40 3,4-DAP/LEM/shellac formulation was administered at 25 mg/kg the time to death was 302 ± 26 min - a 40% increase as compared to toxin alone.

Animal Model:	CD-1 mouse (30-35 g, 8 weeks old)[2]

Dosage:	2.5 mg/kg (IV); 10 mg/kg (PO)
Administration:	IV, orally, once (Pharmacokinetic Analysis)
Result:	Pharmacokinetic Parameters of Amifampridine in CD-1 mouse[1].

	IV (2.5 mg/kg)	PO (10 mg/kg)
$t_{1/2}$ (h)	1.04	1.28
AUC_{0-24} (μM·h)	4.29	9.72
F (%)	100	56.7

REFERENCES

[1]. Maarten J Titulaer, et al. Lambert-Eaton myasthenic syndrome: from clinical characteristics to therapeutic strategies. Lancet Neurol. 2011 Dec;10(12):1098-107.

[2]. T L Harris, et al. Lycopodium clavatum exine microcapsules enable safe oral delivery of 3,4-diaminopyridine for treatment of botulinum neurotoxin A intoxication. Chem Commun (Camb). 2016 Mar 18;52(22):4187-90.

[3]. Ojala KS, et al. A high-affinity, partial antagonist effect of 3,4-diaminopyridine mediates action potential broadening and enhancement of transmitter release at NMJs. J Biol Chem. 2021 Jan-Jun;296:100302.

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