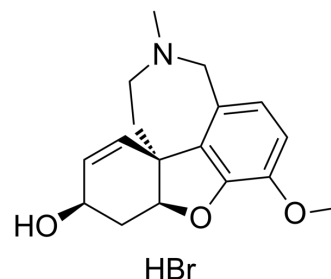


Galanthamine hydrobromide

Cat. No.:	HY-A0009
CAS No.:	1953-04-4
Molecular Formula:	C ₁₇ H ₂₂ BrNO ₃
Molecular Weight:	368.27
Target:	Cholinesterase (ChE); nAChR
Pathway:	Neuronal Signaling; Membrane Transporter/Ion Channel
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

H₂O : 16.67 mg/mL (45.27 mM; ultrasonic and warming and heat to 80°C)
DMSO : 12.5 mg/mL (33.94 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	2.7154 mL	13.5770 mL	27.1540 mL
	5 mM	0.5431 mL	2.7154 mL	5.4308 mL
	10 mM	0.2715 mL	1.3577 mL	2.7154 mL

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

In Vivo

- Add each solvent one by one: PBS
Solubility: 10 mg/mL (27.15 mM); Clear solution; Need ultrasonic
- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 1.25 mg/mL (3.39 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 1.25 mg/mL (3.39 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 1.25 mg/mL (3.39 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Galanthamine hydrobromide (Galantamine hydrobromide) is a selective, reversible, competitive, alkaloid AChE inhibitor, with an IC₅₀ of 0.35 μM. Galanthamine hydrobromide is a potent allosteric potentiating ligand (APL) of human α₃β₄, α₄β₂, α₆β₄ nicotinic receptors (nAChRs). Galanthamine hydrobromide is developed for the research of Alzheimer's disease (AD)^{[1][2][3]}.

IC₅₀ & Target

AChE

In Vitro	<p>Galanthamine hydrobromide is 53-fold selectivity for AChE over butyrylcholinesterase^[2].</p> <p>Galanthamine hydrobromide (25-1000 μM) inhibits both Aβ 1-40 (50 μM) and Aβ 1-42 (50 μM) aggregation^[4].</p> <p>Galanthamine hydrobromide (25-1000 μM) protects against Aβ(1-40) and Aβ(1-42) toxicity in SH-SY5Y cells^[4].</p> <p>Galanthamine hydrobromide also dramatically reduces Aβ(1-40)-induced cellular apoptosis^[4].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>																
In Vivo	<p>Galanthamine hydrobromide (1.25-2.5 mg/kg; i.p.) reduces cognitive deficits in APP23 mice^[5].</p> <p>Galanthamine hydrobromide (10 mg/kg; i.g.) displays short elimination half-life of approximately 2 h in wild-type mice^[6].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 30%;">Animal Model:</td> <td>APP23 mice^[5]</td> </tr> <tr> <td>Dosage:</td> <td>1.25 mg/kg, 2.5 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>Intraperitoneal injection, daily, 1 week</td> </tr> <tr> <td>Result:</td> <td>Effectively remedied the spatial learning deficit.</td> </tr> </table> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 30%;">Animal Model:</td> <td>Female 57B1/6J wild type^[6]</td> </tr> <tr> <td>Dosage:</td> <td>10 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>Oral gavage (Pharmacokinetic Analysis)</td> </tr> <tr> <td>Result:</td> <td>C_{max} (0.31 μg/mL), t_{1/2β} (1.6 h), AUC_{0-24h} (0.67 μg • h/mL).</td> </tr> </table>	Animal Model:	APP23 mice ^[5]	Dosage:	1.25 mg/kg, 2.5 mg/kg	Administration:	Intraperitoneal injection, daily, 1 week	Result:	Effectively remedied the spatial learning deficit.	Animal Model:	Female 57B1/6J wild type ^[6]	Dosage:	10 mg/kg	Administration:	Oral gavage (Pharmacokinetic Analysis)	Result:	C _{max} (0.31 μ g/mL), t _{1/2β} (1.6 h), AUC _{0-24h} (0.67 μ g • h/mL).
Animal Model:	APP23 mice ^[5]																
Dosage:	1.25 mg/kg, 2.5 mg/kg																
Administration:	Intraperitoneal injection, daily, 1 week																
Result:	Effectively remedied the spatial learning deficit.																
Animal Model:	Female 57B1/6J wild type ^[6]																
Dosage:	10 mg/kg																
Administration:	Oral gavage (Pharmacokinetic Analysis)																
Result:	C _{max} (0.31 μ g/mL), t _{1/2β} (1.6 h), AUC _{0-24h} (0.67 μ g • h/mL).																

CUSTOMER VALIDATION

- Nat Commun. 2023 Apr 17;14(1):2182.
- PLoS Biol. 2024 June 27.
- Acta Pharmacol Sin. 2024 Mar 4.
- Free Radic Biol Med. 2019 Dec;145:20-32.
- Antioxidants (Basel). 2022, 11(7), 1228.

See more customer validations on www.MedChemExpress.com

REFERENCES

- [1]. L J Scott, et al. Galantamine: a review of its use in Alzheimer's disease. *Drugs*. 2000 Nov;60(5):1095-122.
- [2]. Marek Samochocki, et al. Galantamine is an allosterically potentiating ligand of neuronal nicotinic but not of muscarinic acetylcholine receptors. *Pharmacol Exp Ther*. 2003 Jun;305(3):1024-36.
- [3]. Acharya Balkrishna, et al. Anti-Acetylcholinesterase Activities of Mono-Herbal Extracts and Exhibited Synergistic Effects of the Phytoconstituents: A Biochemical and Computational Study. *Molecules*. 2019 Nov; 24(22): 4175.
- [4]. Balpreet Matharu, et al. Galantamine inhibits beta-amyloid aggregation and cytotoxicity. *J Neurol Sci*. 2009 May 15;280(1-2):49-58.
- [5]. Debby Van Dam, et al. Symptomatic effect of donepezil, rivastigmine, galantamine and memantine on cognitive deficits in the APP23 model. *Psychopharmacology (Berl)*. 2005 Jun;180(1):177-90.

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA