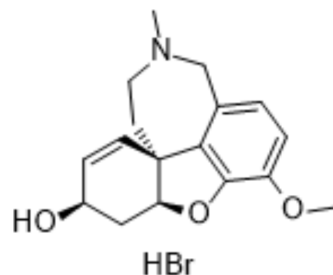


Galanthamine hydrobromide

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	H ₂ O : 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	<div>Mass Solvent Concentration</div>	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	1. Add each solvent one by one: PBS Solubility: 10 mg/mL (27.15 mM); Clear solution; Need ultrasonic				
	2. 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				
	3. 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				
	4. 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 IC ₅₀ of 0.35 μM. Galanthamine hydrobromide is a potent allosteric potentiating ligand (APL) of human α ₃ β ₄ , α ₄ β ₂ , α ₆ β ₄ nicotinic receptors (nAChRs). Galanthamine hydrobromide is developed for the research of Alzheimer's disease (AD) ^{[1][2][3]} .
IC ₅₀ & 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> <tr> <td>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> <tr> <td>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).
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CUSTOMER VALIDATION

- Nat Commun. 2023 Apr 17;14(1):2182.
- Free Radic Biol Med. 2019 Dec;145:20-32.
- Antioxidants (Basel). 2022, 11(7), 1228.
- Antioxidants (Basel). 2022 Feb 14;11(2):385.
- Biochem Pharmacol. 2020 Oct;180:114139.

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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.

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