Galanthamine hydrobromide

Cat. No.: HY-A0009

CAS No.: 1953-04-4

Molecular Formula: $C_{17}H_{22}BrNO_3$

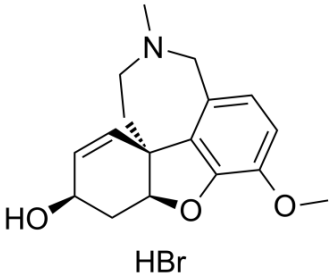
Molecular Weight: 368.27

Target: AChE; nAChR

Pathway: Neuronal Signaling; Membrane Transporter/Ion Channel

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : 25 mg/mL (67.88 mM; Need ultrasonic)

DMSO : ≥ 3.7 mg/mL (10.05 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Mass / Solvent / Concentration	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.

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 $\alpha_3\beta_4$, $\alpha_4\beta_2$, $\alpha_6\beta_4$ nicotinic receptors (nAChRs). Galanthamine hydrobromide is developed for the research of Alzheimer's disease (AD)[1][2][3].

IC_{50} & Target

IC50: 0.35 μM (AChE)[3]

In Vitro

Galanthamine hydrobromide is selective for AChE rather than butyrylcholinesterase[1].

Galanthamine hydrobromide is 53-fold selectivity for AChE over butyrylcholinesterase[2].

Galanthamine hydrobromide (25-1000 μM) inhibited both Aβ 1-40 (50 μM) and Aβ 1-42 (50 μM) aggregation[4].

Galanthamine hydrobromide (25-1000 μM) protects against Aβ(1-40) and Aβ(1-42) toxicity in SH-SY5Y cells[4].

Galanthamine hydrobromide also dramatically reduced Aβ(1-40)-induced cellular apoptosis[4].

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

In Vivo

Galanthamine hydrobromide (1.25-2.5 mg/kg; i.p.) reduces cognitive deficits in APP23 mice[5].
Galanthamine hydrobromide (10 mg/kg; i.g.) displays short elimination half-life of approximately 2 h in wild-type mice[6].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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 μg/ml), $t_{1/2\beta}$ (1.6 h), AUC_{0-24h} (0.67 μg • h/ml).

CUSTOMER VALIDATION

- Free Radic Biol Med. 2019 Dec;145:20-32.
- Biochem Pharmacol. 2020 Oct;180:114139.
- Asian Pac J Trop Med. 2018, 8(10):500-512.

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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.

[6]. Johan Monbaliu, et al. Pharmacokinetics of galantamine, a cholinesterase inhibitor, in several animal species. Arzneimittelforschung. 2003;53(7):486-95.

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

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