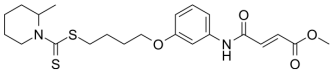
AChE-IN-24

Cat. No.:	HY-151152
CAS No.:	3033542-32-1
Molecular Formula:	$C_{22}H_{30}N_2O_4S_2$
Molecular Weight:	450.61
Target:	Cholinesterase (ChE)
Pathway:	Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	AChE-IN-24 is a potent AChE inhibitor and can penetrate the BBB. AChE-IN-24 has the mighty inhibitory activity to hAChE with an IC_{50} value of 0.053 μM. AChE-IN-24 can be used for the research of Alzheimer s disease (AD)[1].
IC_{50} & Target	AChE

In Vitro

AChE-IN-24 (compound 4c2) has good hAChE inhibitory activity with IC_{50}values of 0.053 μM but owns little inhibition to hBuChE[1].

AChE-IN-2 has inhibitory activity for electric eel acetylcholinesterase (eeAChE) and equine serum butyrylcholinesterase (eqBuChE) with IC_{50}values of 0.088 μM and 7.5μM, respectively[1].

AChE-IN-24 (0-0.2 μM) can cross the BBB comfortably by means of passive diffusion[1].

AChE-IN-2 (0-40 μM) triggers the translocation of Nrf2 to the nucleus, thereby expediting the binding of Nrf2 to the ARE for the transcription process[1].

AChE-IN-2 (7 μM) significantly induces the expression of antioxidant-related enzymes by activating Nrf2 in BV-2 cells[1].

AChE-IN-2 (1, 3, 7 μM) protects cells from H2O2-induced damage and inhibits ROS accumulation[1].

AChE-IN-2 (1, 3, 7 μM) attenuates inflammatory responses[1].

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

Cell Viability Assay[1]

Cell Line:	BV-2 microglial cells
Concentration:	0, 2.5, 5, 10, 20 and 40 μM
Incubation Time:	24 h
Result:	Not observed significant cytotoxicity.

Western Blot Analysis[1]

Cell Line:	BV-2 microglial cells
Concentration:	7 μM
Incubation Time:	0-15 h
Result:	Up-regulated the amount of total Nrf2 in a time-dependent manner, decreased gradually

	the cytosolic Nrf2 level with its continuous accumulation in the nucleus and increased the total cellular Nrf2 accumulation in concentration-dependently. Increased the protein expression levels of HO-1, NQO1, and GPX4 in a concentration-dependent manner with the biggest upregulation observed at 10 μM and significantly increased the protein levels of HO-1, NQO1, and GPX4 reaching the maximum at 9h, 6 h, and 3 h, respectively[1].

In Vivo

AChE-IN-24 (compound 4c2) (oral; 0, 625, 1250, and 2500 mg/kg) is well tolerated and no toxicity in KM mice[1].
AChE-IN-24 (7.5 mg/kg, 15 mg/kg and 30mg/kg; once) ameliorates cognitive deficit induced by Scopolamine, suggesting a practicable therapeutic effect on AD[1].

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

Animal Model:	KM mice[1]
Dosage:	0, 625, 1250, and 2500 mg/kg
Administration:	oral; 0, 625, 1250, and 2500 mg/kg
Result:	Not discovered abnormal behavior and acute toxicity were monitored for the first 4 h after administration, no acute neurological toxicities inclusive of tremor, convulsion, and death and no obvious signs of poisoning in the heart, liver, lungs, kidneys, and brain.

Animal Model:	The cognitive deficit mice model[1]
Dosage:	7.5 mg/kg, 15 mg/kg and 30mg/kg
Administration:	7.5 mg/kg, 15 mg/kg and 30mg/kg; once
Result:	Reversed the step-down latency and number of errors in a concentration-dependent manner.

REFERENCES

[1]. Jie Guo, et al. A multi-target directed ligands strategy for the treatment of Alzheimer's disease: Dimethyl fumarate plus Tranilast modified Dithiocarbate as AChE inhibitor and Nrf2 activator. Eur J Med Chem. 2022 Aug 11;242:114630.

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