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Lieferung & Zahlungsart

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Zuschläge

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- Trockeneiszuschlag
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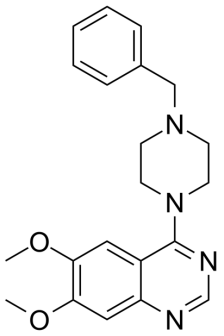
hAChE/hBACE-1-IN-4

Cat. No.:	HY-161512
CAS No.:	229476-71-5
Molecular Formula:	$C_{21}H_{24}N_4O_2$
Molecular Weight:	364.44
Target:	Cholinesterase (ChE); Amyloid-β
Pathway:	Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

hAChE/hBACE-1-IN-4 (compound AK-2) is a quinazoline derivative. hAChE/hBACE-1-IN-4 shows significant inhibitory activity against hAChE and hBACE-1 enzymes (hAChE, IC_{50}=0.283 μM; hBACE-1, IC_{50}=0.231 μM). hAChE/hBACE-1-IN-4 has the potential to inhibit Aβ aggregation. hAChE/hBACE-1-IN-4 has non-neurotoxicity , blood-brain barrier permeability and oral activity. hAChE/hBACE-1-IN-4 can be used in Alzheimer's disease research[1].

IC_{50} & Target

hAChE	hBCHE
0.283 μM (IC_{50})	0.231 μM (IC_{50})

In Vitro

hAChE/hBACE-1-IN-4 (10, 20, 40 and 80 μM; 24 h) has non-neurotoxic properties[1].

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

Cell Viability Assay[1]

Cell Line:	SH-SY5Y
Concentration:	10, 20, 40 and 80 μM
Incubation Time:	24 h
Result:	Reduced cell viability by 26% at maximum concentration (80 μM).

In Vivo

hAChE/hBACE-1-IN-4 (500 mg/kg; po, 14 days) possesses a substantial margin of safety and is safe to be utilized for further in-vivo investigations in Wistar rats[1].

hAChE/hBACE-1-IN-4 (20 mg/kg; po, 9 days) inhibits ACE-1 activity to restore cognitive deficits and acts as an anti-Aβ agent at its tested doses in Wistar rats[1].

hAChE/hBACE-1-IN-4 (10 and 20 mg/kg; ig, 4 days) possesses the ability to cross the BBB and reaches their specific site of action in the brain[1].

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

Animal Model:	Wistar rats [1]
Dosage:	500 mg/kg for 14 days
Administration:	Oral gavage (p.o.)

Result:	Observed the renal and hepatic functional parameters within the prescribed limits .Examined and found with normal tissue appearance such as kidneys, heart, liver, and brains.

Animal Model:	Wistar rats [1]
Dosage:	20mg/kg for 9 days
Administration:	Oral gavage (p.o.)
Result:	Decreased the ELT and increased total platform crossing as compared to the diseased model group .Increased the neuronal density and neuronal arrangement.

Animal Model:	Wistar rats[1]
Dosage:	10 and 20 mg/kg for 4 days
Administration:	i.g.
Result:	The concentrations of AK-2 in brain homogenates were observed to 0.633 and 0.977 μg/mL.

REFERENCES

[1]. Verma A, Waiker D K, Singh N, et al. Lead optimization based design, synthesis, and pharmacological evaluation of quinazoline derivatives as multi-targeting agents for Alzheimer's disease treatment[J]. European Journal of Medicinal Chemistry, 2024: 116450.

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