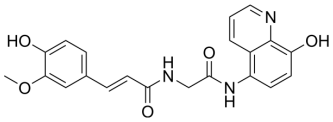
NLRP3-IN-33

Cat. No.:	HY-162402
Molecular Formula:	$C_{21}H_{19}N_3O_5$
Molecular Weight:	393.39
Target:	Reactive Oxygen Species; Cholinesterase (ChE)
Pathway:	Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

NLRP3-IN-33 (Compound 12o) is a blood-brain barrier permeable inhibitor of AChE and BChE, with IC_{50} values of 1.02 μM and 7.03 μM against hAChE and hBChE respectively. NLRP3-IN-33 possesses antioxidant, anti-inflammatory, and metal chelating activities, making it a potential candidate for research in Alzheimer's disease (AD)[1].

IC_{50} & Target

IC50: 1.02 Mm (hAChE)[1].
IC50: 7.03 μM (hBChE)[1].

In Vitro

NLRP3-IN-33 (12o) (1-30 μM; 24 h) exhibits no significant cytotoxicity in PC-12 cells[1].NLRP3-IN-33 possesses antioxidant activity and can inhibit the generation of free radicals, with an IC_{50} value of 6.19 μM. 12o (1-20 μM; 24 h) effectively alleviates H_2O_2 (600 μM; 24 h)-induced oxidative stress and exhibits neuroprotective effects in PC-1 cells[1].NLRP3-IN-33 (1-20 μM; 24 h) also inhibits the activation of the NLRP3 inflammasome in PC-1 cells and mitigates the damage caused by mitochondrial-induced reactive oxygen species (ROS) and mitochondrial membrane potential (MMP) triggered by LPS (1 μg/mL) and ATP (5 mM) in HMC-3 cells[1].

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

Western Blot Analysis[1]

Cell Line:	HMC-3
Concentration:	12.5 μM, 25 μM
Incubation Time:	24 h
Result:	Inhibited the NLRP3 inflammasome activation and caspase-1 release. Significantly decreased the expression of NF-κB and NLRP3 proteins.

In Vivo

LRP3-IN-33 (12o) (0.05-0.02 mg/mL) can more effectively reduce mitochondrial and cellular oxidative stress in Drosophila AD models at a lower dosage (0.05 mg/mL)[1].
NLRP3-IN-33 (5 mg/kg; i.p.; once daily for 22 consecutive days) is capable of improving memory and cognitive impairments in AD mouse models induced by scopolamine (HY-N0296) (1.4 mg/kg; i.p.; once daily for 5 consecutive days)[1].

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Animal Model:	Alzheimer's disease (AD) fractional model[1]

Dosage:	1 mg/kg, 5 mg/kg
Administration:	Intraperitoneal injection (i.p.); Once daily for 22 days. Received scopolamine (HY-N0296) (1.4 mg/kg; i.p.; once daily for 5 days) on the last 5 days of the experiment (day 18 to day 22).
Result:	Significantly shortened escape latency time as compared to the scopolamine-treated group.

REFERENCES

[1]. Singh G, et al. Design, Synthesis, and Biological Evaluation of Ferulic Acid Template-Based Novel Multifunctional Ligands Targeting NLRP3 Inflammasome for the Management of Alzheimer's Disease. ACS Chem Neurosci. 2024;15(7):1388-1414.

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