Oxidopamine hydrobromide

Cat. No.: HY-B1081A

CAS No.: 636-00-0

Molecular Formula: $C_8H_{12}BrNO_3$

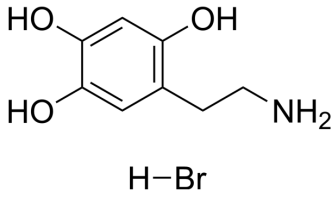
Molecular Weight: 250.09

Target: Dopamine Receptor; Autophagy; Mitophagy; COX; PGE synthase; Interleukin Related; p38 MAPK; Apoptosis; Caspase

Pathway: GPCR/G Protein; Neuronal Signaling; Autophagy; Immunology/Inflammation; MAPK/ERK Pathway; Apoptosis

Storage: 4°C, stored under nitrogen

* The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (199.93 mM; ultrasonic and warming and heat to 60°C)

H_2O : 20 mg/mL (79.97 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.9986 mL	19.9928 mL	39.9856 mL
	5 mM	0.7997 mL	3.9986 mL	7.9971 mL
	10 mM	0.3999 mL	1.9993 mL	3.9986 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 50 mg/mL (199.93 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (10.00 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (8.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Oxidopamine (6-OHDA) hydrobromide is an antagonist of the neurotransmitter dopamine. Oxidopamine hydrobromide is a widely used neurotoxin and selectively destroys dopaminergic neurons. Oxidopamine hydrobromide promotes COX-2 activation, leading to PGE_2 synthesis and pro-inflammatory cytokine IL-1β secretion. Oxidopamine hydrobromide can be used for the research of Parkinson's disease (PD), attention-deficit hyperactivity disorder (ADHD), and Lesch-Nyhan syndrome[1][2][3][4].

IC_{50} & Target	COX-2	IL-1β	Caspase-3	Caspase-8
	Caspase-9			

In Vitro

Oxidopamine hydrobromide (0-500 μM, 24 h) decreases the viability of both Neuro-2a cells and SH-SY5Y cells in a concentration-dependent manner[1].
Oxidopamine hydrobromide (75-150 μM, 0-24 h) induces COX-2 expression and nuclear translocation[1].
Oxidopamine hydrobromide (75-150 μM, 0-24 h) causes PGE_2 biosynthesis and pro-inflammatory cytokine IL-1β production [1].
Oxidopamine hydrobromide (0-150 μM, 12 h) induces apoptosis and mitochondrial membrane depolarization of pheochromocytoma PC12 cells[3].
Oxidopamine hydrobromide (75 μM, 0-12 h) induces p38 phosphorylation[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Neuro-2a cells and SH-SY5Y cells
Concentration:	0-500 μM
Incubation Time:	24 or 48 h
Result:	Induced neurotoxicity, caused cytotoxicity in both Neuro-2a cells and SH-SY5Y cells in a concentration dependent manner. EC_{50}=111 μM for 24 h incubation and 109 μM for 48 h incubation in the Neuro-2a cells; EC_{50}=118 μM for 24 h incubation and 107 μM for 48 h incubation in the SH-SY5Y cells.

RT-PCR[1]

Cell Line:	Neuro-2a cells and SH-SY5Y cells
Concentration:	75 or 150 μM
Incubation Time:	0, 6 or 24 h
Result:	Quickly and robustly induced COX-2 in a time-dependent manner. Induced COX-2 activation characterized by expression induction and nuclear translocation. Substantially increased PGE_2 in the culture medium by nearly 5-fold in Neuro-2a cells (at 75 μM) and 3-fold in SH-SY5Y cells (at 150 μM). Significantly upregulated the pro-inflammatory cytokine interleukin-1β (IL-1β) within Neuro-2a cells and SH-SY5Y cells.

Apoptosis Analysis[3]

Cell Line:	PC12 cells
Concentration:	0, 25, 50, 75, and 150 μM
Incubation Time:	0, 2, 4, 6, 12, and 20 h
Result:	Induced apoptosis of PC12 cells. Increased the activities of caspase-3, -8 and -9 in PC12 cells in a time- and concentration-dependent manner. Increased these caspase activities at 2-4 h and reached a maximum at 12 h. Decreased cells with high mitochondrial membrane potential (JC-1 aggregate) in a time- and concentration-dependent manner.

Western Blot Analysis[3]

Cell Line:	PC12 cells

Concentration:	75 μM
Incubation Time:	0, 3, 5, 6, 8, 10, and 12 h
Result:	Increased the level of p-p38 in a time-dependent manner.

In Vivo

Oxidopamine hydrobromide can be used in animal modeling to construct Parkinson's syndrome models. Oxidopamine hydrobromide (5 μg/2 μL, unilaterally injected into the right striatum) induces degeneration of dopaminergic neurons in substantia nigra of rats[2].

Induction ofParkinson's disease model[5][6]

- Background

  The chemical structure ofoxidopamine hydrochloride is similar to dopamine (DA), enabling it to competewith DA for uptake sites and be subsequently taken into cells. Once inside thecells, oxidopamine hydrochloride can be oxidized and decomposed, generatingreactive oxygen species, which further produce oxygen free radicals through MAO(monoamine oxidase) or directly cause mitochondrial dysfunction, leading to thedeath of dopaminergic neurons.

- Specific Mmodeling Methods

  Rats: Sprague-Dawley (SD) • Male • 200-250g •
  Administration: 5μg/2μL/site • stereotaxicallyinjected in the fight striatum • single dose.

  Note

  (1) Lesions were made by the unilateral injection of Oxidopamine hydrochloride (5 μg in 2 μl/site) into the right striatum at the two coordinates: ⊠ AP, ?0.7; L, ?3.0; DV, ?5.5 and 4.5 mm from Bregma. ⊠ AP, ?0.2; L, ?2.6; DV, ?5.5 and 4.5 mm from Bregma. The two coordinates were injected Oxidopamine hydrochloride 10 μg in 4 μl/2 sites.(2) Oxidopamine hydrochloride was prepared freshly in dark to avoid autooxidation, and was administered using a 5 μl microinjector at a rate of 0.5 μl/min. The syringe was left in place for 5 min before slowly retracting it to allow for toxin diffusion and prevent the toxin reflux.(3) On the 56th day after the injury, the animals were decapitated under deep halothane anesthesia. Their brains were quickly removed from the skull, rinsed with chilled saline, and tissue samples containing the caudate-putamen head were dissected from both the lesioned and unlesioned striata on ice.(4) The animals were housed in an environment with a 12-hour light/dark cycle, with the temperature maintained at 22-23°C. They were allowed free access to food and tap water.

- Modeling Indicators

Behavioral monitoring: Rats exhibit rotation with a rotation count greater than 210 r/30 min.Molecular changes: Elevated levels of COX-2, TNF-α mRNA, and COX-2 protein.Histopathological changes: Chromatin condensation into clumps around the nucleus, along with evident mitochondrial swelling and vacuolation. Induced nigrostriatal nerve terminal lesions. Decreased striatal dopamine levels and reduced number of tyrosine hydroxylase immunoreactive cells in the ipsilateral substantia nigra, accompanied by long-term significant atrophy of remaining dopaminergic neurons.

Opposite Product(s): Resveratrol (HY-16561)

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CUSTOMER VALIDATION

- Immunity. 2024 Feb 13;57(2):364-378.e9.
- Cell Metab. 2022 Nov 11;S1550-4131(22)00490-9.
- Cell Stem Cell. 2023 Jun 1;30(6):832-850.e6.
- CNS Neurosci Ther. 2023 Aug 11.
- CNS Neurosci Ther. 2023 Apr 26.

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REFERENCES

[1]. Jin F, et al. Neuroprotective effect of resveratrol on 6-OHDA-induced Parkinson's disease in rats. Eur J Pharmacol. 2008 Dec 14;600(1-3):78-82.

[2]. Kang X, et al. Cyclooxygenase-2 contributes to oxidopamine-mediated neuronal inflammation and injury via the prostaglandin E2 receptor EP2 subtype. Sci Rep. 2017 Aug 25;7(1):9459.

[3]. Fujita H et al. Cell-permeable cAMP analog suppresses 6-hydroxydopamine-induced apoptosis in PC12 cells through the activation of the Akt pathway. Brain Res. 2006 Oct 3;1113(1):10-23.

[4]. Soto-Otero R et al. Autoxidation and neurotoxicity of 6-hydroxydopamine in the presence of some antioxidants: potential implication in relation to the pathogenesis of Parkinson's disease. J Neurochem. 2000 Apr;74(4):1605-12.

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