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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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Oxidopamine hydrochloride

Cat. No.: HY-B1081

CAS No.: 28094-15-7

Molecular Formula: $C_8H_{12}ClNO_3$

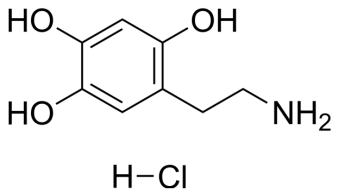
Molecular Weight: 205.64

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

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

Storage: 4°C, stored under nitrogen

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



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (486.29 mM; Need ultrasonic)
DMSO : 83.33 mg/mL (405.22 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.8629 mL	24.3143 mL	48.6287 mL
5 mM	0.9726 mL	4.8629 mL	9.7257 mL
10 mM	0.4863 mL	2.4314 mL	4.8629 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (486.29 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (10.11 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (10.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Oxidopamine (6-OHDA) hydrochloride is an antagonist of the neurotransmitter dopamine. Oxidopamine hydrochloride is a widely used neurotoxin and selectively destroys dopaminergic neurons. Oxidopamine hydrochloride promotes COX-2 activation, leading to PGE_2 synthesis and pro-inflammatory cytokine IL-1β secretion. Oxidopamine hydrochloride 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 hydrochloride (0-500 μM, 24 h) decreases the viability of both Neuro-2a cells and SH-SY5Y cells in a concentration-dependent manner[1].
Oxidopamine hydrochloride (75-150 μM, 0-24 h) induces COX-2 expression and nuclear translocation[1].
Oxidopamine hydrochloride (75-150 μM, 0-24 h) causes PGE_2 biosynthesis and pro-inflammatory cytokine IL-1β production [1].
Oxidopamine hydrochloride (0-150 μM, 12 h) induces apoptosis and mitochondrial membrane depolarization of pheochromocytoma PC12 cells[3].
Oxidopamine hydrochloride (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 hydrochloride (5 μg/2 μL, unilaterally injected into the right striatum) induces degeneration of dopaminergic neurons in substantia nigra of rats[2].

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

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]. 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.

[2]. 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.

[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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