4-P-PDOT

Cat. No.: HY-100609

CAS No.: 134865-74-0

Molecular Formula: $C_{19}H_{21}NO$

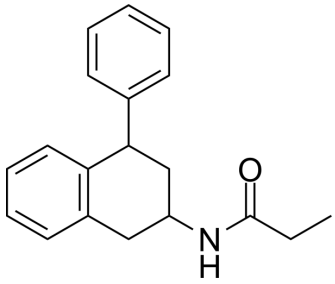
Molecular Weight: 279.38

Target: Melatonin Receptor

Pathway: GPCR/G Protein; Neuronal Signaling

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 41.67 mg/mL (149.15 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.5794 mL	17.8968 mL	35.7935 mL
	5 mM	0.7159 mL	3.5794 mL	7.1587 mL
	10 mM	0.3579 mL	1.7897 mL	3.5794 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 4.17 mg/mL (14.93 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 4.17 mg/mL (14.93 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

4-P-PDOT is a potent, selective and affinity Melatonin receptor (MT2) antagonist. 4-P-PDOT is >300-fold more selective for MT2 than MT1. 4-P-PDOT significantly counteracts Melatonin-mediated antioxidant effects (GSH/GSSG ratio, phospho-ERK, Nrf2 nuclear translocation, Nrf2 DNA-binding activity)[1][2][3][4].

IC_{50} & Target

MT2

In Vitro

In CHO-mt1 cells the amidotetraline 4-P-PDOT (10 mM) has no effect on forskolin-stimulated cyclic AMP levels, either alone, or in the presence of Melatonin. In contrast, in CHO-MT2 cells, 4-P-PDOT is an agonist, producing a concentration-dependent inhibition of forskolin stimulated cyclic AMP, with a pEC_{50} value of 8.72 and intrinsic activity of 0.86[1].

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

Cell Viability Assay[5]

Cell Line:	HT-29 and HeLa cells.
Concentration:	50 μM
Incubation Time:	30 min
Result:	Produced a negligible effect on cell viability induced by melatonin.

In Vivo

4-P-PDOT (0.5-1.0 mg/kg; intravenous injection; klotho mutant mice) treatment significantly reverses antioxidant effects mediated by Melatonin. And significantly reverses the changes in the levels of these GSH-related parameters. 4-P-PDOT treatment significantly reverses the memory function of Melatonin-treated klotho mutant mice. 4-P-PDOT also counteracts Melatonin-mediated attenuation in response to the decreases in phospho-ERK expression, Nrf2 nuclear translocation, Nrf2 DNA-binding activity, and GCL mRNA expression in the hippocampi of klotho mutant mice[2].

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

Animal Model:	Klotho mutant mice treatment with Melatonin[2]
Dosage:	0.5 mg/kg or 1.0 mg/kg
Administration:	Intravenous injection
Result:	Significantly reversed antioxidant effects mediated by Melatonin. Significantly reversed the changes in the levels of these GSH-related parameters. Significantly reversed the memory function of Melatonin-treated klotho mutant mice.

CUSTOMER VALIDATION

- Eur J Pharmacol. 2021 Oct 23;174589.
- Zool Res. 2022 Jul 18;43(4):537-551.
- bioRxiv. 2023 Apr 19.
- Oxid Med Cell Longev. 2021 Jun 23;2021:9981480.

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REFERENCES

[1]. Dubocovich ML. Melatonin receptors: are there multiple subtypes? Trends Pharmacol Sci. 1995 Feb;16(2):50-6.

[2]. hin EJ, et al. Melatonin attenuates memory impairment induced by Klotho gene deficiency via interactive signaling between MT2 receptor, ERK, and Nrf2-related antioxidant potential. Int J Neuropsychopharmacol. 2014 Dec 30;18(6). pii: pyu105.

[3]. Christopher Browning, et al. Pharmacological characterization of human recombinant melatonin mt1 and MT2 receptors. British Journal of Pharmacology (2000) 129, 877-886.

[4]. Dubocovich ML, et al. Melatonin receptor antagonists that differentiate between the human Mel1a and Mel1b recombinant subtypes are used to assess the pharmacological profile of the rabbit retina ML1 presynaptic heteroreceptor. Naunyn Schmiedebergs Arch Pharmacol. 1997 Mar;355(3):365-75.

[5]. Roberto Pariente, et al. Participation of MT3 melatonin receptors in the synergistic effect of melatonin on cytotoxic and apoptotic actions evoked by chemotherapeutics. Cancer Chemother Pharmacol. 2017 Nov;80(5):985-998.

Caution: Product has not been fully validated for medical applications. For research use only.

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