Propranolol hydrochloride

Cat. No.: HY-B0573

CAS No.: 318-98-9

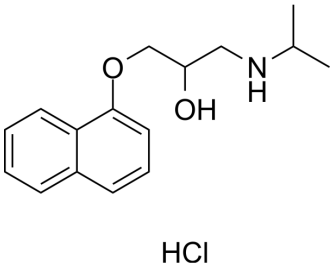
Molecular Formula: $C_{16}H_{22}ClNO_2$

Molecular Weight: 295.8

Target: Adrenergic Receptor; Bacterial

Pathway: GPCR/G Protein; Neuronal Signaling; Anti-infection

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 150 mg/mL (507.10 mM)
H_2O : 33.33 mg/mL (112.68 mM; Need ultrasonic)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Concentration / Solvent / Mass	1 mg	5 mg	10 mg
	1 mM	3.3807 mL	16.9033 mL	33.8066 mL
	5 mM	0.6761 mL	3.3807 mL	6.7613 mL
	10 mM	0.3381 mL	1.6903 mL	3.3807 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 25 mg/mL (84.52 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Propranolol hydrochloride is a nonselective β-adrenergic receptor (βAR) antagonist, has high affinity for the β1AR and β2AR with K_i values of 1.8 nM and 0.8 nM, respectively[1]. Propranolol hydrochloride inhibits [^{3}H]-DHA binding to rat brain membrane preparation with an IC_{50} of 12 nM[2]. Propranolol hydrochloride is used for study of hypertension, pheochromocytoma, myocardial infarction, cardiac arrhythmias, angina pectoris, and hypertrophic cardiomyopathy[3].

IC_{50} & Target

β adrenergic receptor

In Vitro

Propranolol hydrochloride (10^{-7} M-10^{-3} M; 24 and 48 hours) increases the total ERK1/2 levels in a dose-dependent manner, and ERK1/2 activation is observed specifically at 10^{-5} M in HemSCs[4].
Propranolol hydrochloride (10^{-9} M-10^{-3} M; 24 and 48 hours) significant decreases cell proliferation at 10^{-4} M propranolol after 24 hours and 10^{-9} M propranolol after 48 hours in HemSCs[4].
Propranolol hydrochloride (50 μM-200 μM; 24 hours) increases Annexin V positivity and caspase-3 activation, rapidly induces

HemSC apoptosis[4].

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

Western Blot Analysis[4]

Cell Line:	HemSC cells
Concentration:	10^{-7} M-10^{-3} M
Incubation Time:	24 and 48 hours
Result:	Increased the total ERK1/2 levels in a dose-dependent manner.

Cell Viability Assay[4]

Cell Line:	HemSC cells
Concentration:	10^{-9} M-10^{-3} M
Incubation Time:	24 and 48 hours
Result:	Suppressed HemSC Proliferation.

Apoptosis Analysis[4]

Cell Line:	HemSC cells
Concentration:	50 μM, 100 μM, or 200 μM
Incubation Time:	24 hours
Result:	Induced HemSC cell death occurred via an apoptotic pathway.

In Vivo

Propranolol hydrochloride (orally administration; 40 mg/kg; daily) significantly reduces the vessel diameter relative to the vehicle-treated implants, and increases the number of cells that expressed phosphorylated ERK1/2 within the IH Matrigel implant[4].

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

Animal Model:	A xenograft mouse model of IH (infantile hemangiomas) with HemSC cells[4]
Dosage:	40 mg/kg
Administration:	Orally administration; 40 mg/kg; daily
Result:	Improved vessel development in the IH mouse model that correlated with MAPK pathway activation.

CUSTOMER VALIDATION

- Nat Commun. 2023 May 2;14(1):2523.
- Chemosphere. 2019 Jun;225:378-387.
- Sci Signal. 2020 Nov 24;13(659):eaax0273.
- Am J Pathol. 2023 Apr 21;S0002-9440(23)00132-3.
- iScience. 2023 Jul 19.

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REFERENCES

[1]. Galandrin S, et al. Distinct signaling profiles of beta1 and beta2 adrenergic receptor ligands toward adenylyl cyclase and mitogen-activated protein kinase reveals the pluridimensionality of efficacy. Mol Pharmacol. 2006 Nov;70(5):1575-84. Epub 2006 Aug 1

[2]. Briley M, et al. Evidence against beta-adrenoceptor blocking activity of diltiazem, a drug with calcium antagonist properties. Br J Pharmacol. 1980 Aug;69(4):669-73.

[3]. Al-Majed AA, et al. Propranolol. Profiles Drug Subst Excip Relat Methodol. 2017;42:287-338.

[4]. Munabi NC, et al. Propranolol Targets Hemangioma Stem Cells via cAMP and Mitogen-Activated Protein Kinase Regulation. Stem Cells Transl Med. 2016 Jan;5(1):45-55.

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