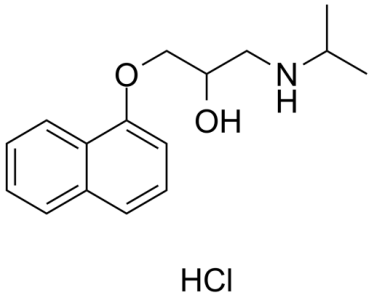
Data Sheet

Product Name:	Propranolol (hydrochloride)
Cat. No.:	CS-2680
CAS No.:	318-98-9
Molecular Formula:	C16H22ClNO2
Molecular Weight:	295.80
Target:	Adrenergic Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
Solubility:	DMSO : ≥ 150 mg/mL (507.10 mM); H2O : 33.33 mg/mL (112.68 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

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 to control hypertension, pheochromocytoma, myocardial infarction, cardiac arrhythmias, angina pectoris, and hypertrophic cardiomyopathy[3]. IC50 & Target: IC50: 12 nM (βAR)[1] **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].

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].

PROTOCOL (Extracted from published papers and Only for reference)

Animal Administration: [4]Male Wistar rats weighing 250–300 g are used in the study. Propranolol is dissolved with tap water, and given ad lib. The daily consumption of propranolol is estimated to be 40 mg/kg based on a mean intake of 35 mL/day of water for a 250 g rat. The treatment period of β-adrenoceptor antagonists is changed from 1 to 3 or 6 weeks and the effects are examined[4].

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.

CAIndexNames:

2-Propanol, 1-[(1-methylethyl)amino]-3-(1-naphthalenyloxy)-, hydrochloride (1:1)

SMILES:

OC(CNC(C)C)COC1=CC=CC2=CC=CC=C12.Cl

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

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