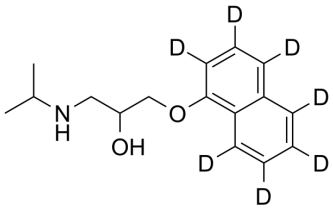
Propranolol-d_7 (ring-d_7)

Cat. No.:	HY-B0573S1
CAS No.:	344298-99-3
Molecular Formula:	$C_{16}H_{14}D_7NO_2$
Molecular Weight:	266.39
Target:	Adrenergic Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMF : ≥ 50 mg/mL (187.69 mM)
DMSO : 50 mg/mL (187.69 mM; Need ultrasonic)
DMSO : ≥ 30 mg/mL (112.62 mM)
Ethanol : ≥ 30 mg/mL (112.62 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7539 mL	18.7695 mL	37.5389 mL
5 mM	0.7508 mL	3.7539 mL	7.5078 mL
10 mM	0.3754 mL	1.8769 mL	3.7539 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1.25 mg/mL (4.69 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.25 mg/mL (4.69 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.25 mg/mL (4.69 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Propranolol-d_7 (ring-d_7) is the deuterium labeled Propranolol hydrochloride. Propranolol hydrochloride is a nonselective β-adrenergic receptor (βAR) antagonist, has high affinity for the β1AR and β2AR with Ki values of 1.8 nM and 0.8 nM, respectively[1]. Propranolol hydrochloride inhibits [3H]-DHA binding to rat brain membrane preparation with an IC50 of 12

	nM[2]. Propranolol hydrochloride is used for study of hypertension, pheochromocytoma, myocardial infarction, cardiac arrhythmias, angina pectoris, and hypertrophic cardiomyopathy[3].
In Vitro	Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019;53(2):211-216.

[2]. 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

[3]. 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.

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

[5]. 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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