Pronethalol

Cat. No.: HY-B1238

CAS No.: 54-80-8

Molecular Formula: $C_{15}H_{19}NO$

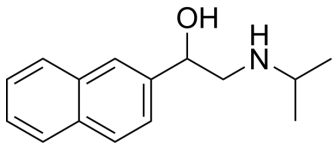
Molecular Weight: 229.32

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

DMSO : 50 mg/mL (218.04 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.3607 mL	21.8036 mL	43.6072 mL
5 mM	0.8721 mL	4.3607 mL	8.7214 mL
10 mM	0.4361 mL	2.1804 mL	4.3607 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: ≥ 2.5 mg/mL (10.90 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.90 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.90 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pronethalol ((±)-Pronethalo) is a non-selective β-adrenergic antagonist. Pronethalol is a potent inhibitor of Sox2 expression. Pronethalol protects against and to reverse Digitalis-induced ventricular arrhythmias and limits the cerebral arteriovenous malformation (AVMs)[1][2].

IC_{50} & Target

β adrenergic receptor

In Vitro

Pronethalol (2, 10, 20 μM) represses EGFP expression in a dose- and time-dependent manner in ReNcell VM cells. Pronethalol

	(10 μM; 2 days) reduces Sox2 expression to less than 10% after 2 days of treatment[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Pronethalol (0.15 mg/g; daily; for 14 days) stabilizes endothelial differentiation, lumen formation and improves cerebral arteriovenous malformation (AVMs) in $Mgp^{-/-}$ mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Int J Biol Macromol. 2023 May 19;242(Pt 2):124870.

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REFERENCES

[1]. Jiayi Yao, et al. Elevated endothelial Sox2 causes lumen disruption and cerebral arteriovenous malformations. J Clin Invest. 2019 Jun 24;129(8):3121-3133.

[2]. Aroesty JM, et al. The effects of a beta-adrenergic blocking agent, pronethalol, on digitalis-induced ventricular arrhythmias. Am Heart J. 1966 Apr;71(4):503-508.

[3]. Aroesty JM, et al. The effects of a beta-adrenergic blocking agent, pronethalol, on digitalis-induced ventricular arrhythmias. Am Heart J. 1966 Apr;71(4):503-8.

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