Nicainoprol

Cat. No.: HY-100572

CAS No.: 76252-06-7

Molecular Formula: $C_{21}H_{27}N_3O_3$

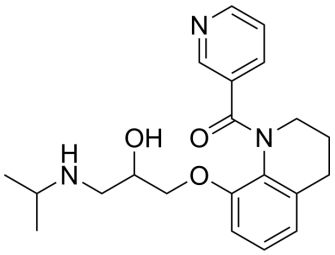
Molecular Weight: 369.46

Target: Sodium Channel

Pathway: Membrane Transporter/Ion Channel

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 : ≥ 29 mg/mL (78.49 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7067 mL	13.5333 mL	27.0665 mL
5 mM	0.5413 mL	2.7067 mL	5.4133 mL
10 mM	0.2707 mL	1.3533 mL	2.7067 mL

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

BIOLOGICAL ACTIVITY

Description

Nicainoprol is a fast-sodium-channel blocking agent, which is a potent antiarrhythmic agent.

IC_{50} & Target

Sodium Channel[1]

In Vitro

The antiarrhythmic agent Nicainoprol, a fast-sodium-channel blocking drug, also protected isolated rat hearts against reperfusion arrhythmias, but is without beneficial effects on cardiac hemodynamics and biochemical parameters, in contrast to the ACE inhibitor[1].

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

In Vivo

The effect of the novel antiarrhythmic agent Nicainoprol on coronary occlusion and reperfusion arrhythmia is investigated in isolated working rat hearts and in anesthetized rats. In isolated working rat hearts Nicainoprol (10 μM, 5 μM and 100 μM) induces concentration-related protection against reperfusion arrhythmia without changing the cardiodynamics, with the exception of a decrease in heart rate at the highest concentration. Enzyme levels (lactate dehydrogenase and creatine kinase) in the coronary venous effluent, and cardiac tissue concentrations of glycogen, lactate, ATP and creatine phosphate

are not affected by Nicainoprol. Given to anesthetized rats, Nicainoprol (5 and 10 mg/kg i.v.) reduces dose dependently in the early post occlusion (0-30 min) period, the percentage of animals with premature ventricular complexes (PVCs) and ventricular tachycardia while completely preventing the occurrence of ventricular fibrillation. In the reperfusion period no animal treated with 5 mg/kg and 12% of the rats treated with 10 mg/kg showed PVCs (the only form of arrhythmia observed in this period) versus 60% of the control rats. Both doses of Nicainoprol induces a decrease in heart rate, blood pressure and myocardial oxygen consumption. The ratio of infarct mass to ventricular mass is significantly reduced by 20% at a dose of 5 mg/kg and by 28% at the dose of 10 mg/kg. Nicainoprol could be useful in the prevention and treatment of arrhythmias associated with acute myocardial infarction[2].

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

REFERENCES

[1]. Linz W, et al. Cardiac arrhythmias are ameliorated by local inhibition of angiotensin formation and bradykinin degradation with the converting-enzyme inhibitor ramipril. Cardiovasc Drugs Ther. 1989 Dec;3(6):873-82.

[2]. Martorana PA, et al. Effects of nicainoprol on reperfusion arrhythmia in the isolated working rat heart and on ischemia and reperfusion arrhythmia and myocardial infarct size in the anesthetized rat. Eur J Pharmacol. 1987 Nov 17;143(3):391-401.

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