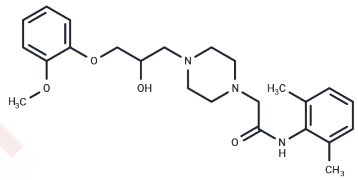
Data Sheet (Cat.No.T6633)

TargetMol

Ranolazine

Chemical Properties



CAS No. : 95635-55-5

Formula: C24H33N3O4

Molecular Weight: 427.54

Appearance: no data available

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	Ranolazine (RS 43285-003) is a calcium uptake inhibitor via the sodium/calcium channel, used to treat chronic angina. It affects the sodium-dependent calcium channels during myocardial ischemia in rabbits by altering the intracellular sodium level.
Targets(IC50)	Calcium Channel,Sodium Channel
In vitro	In the absence and presence of IK-blocking drugs, when late INa is increased, Ranolazine inhibits the late component of INa and attenuates prolongation of action potential duration. Ranolazine (10 mM) reduces by 89% of the 13.6-fold increase in variability of APD caused by 10 nM ATX-II. Ranolazine is found to bind more tightly to the inactivated state than the resting state of the sodium channel underlying I(NaL), with apparent dissociation constants K(dr)=7.47 mM and K(di)=1.71 mM, respectively. Ranolazine（5 mM and 10 mM） reversibly shortens the duration of TCs and abolishes the after contraction.
In vivo	In dog left ventricular myocytes, Ranolazine significantly and reversibly stimulate the action potential duration (APD) of shortened myocytes at 0.5 or 0.25 Hz in a concentration-dependent manner. In rat hearts, Ranolazine (10 mM) significantly increased 1.5-fold to 3-fold under glucose oxidation conditions. Ranolazine (10 mM) also increased glucose oxidation (high calcium, low FA; 15 ml/min) in Langendorff hearts in normoxic rats. Ranolazine significantly improves the function of the reperfused ischemic working heart, which is associated with a significant increase in glucose oxidation function.
Kinase Assay	In vitro kinase assay for CDK1 and Aurora kinases:CDK1 kinase activity is tested by the CDK1/cyclin B complex purified from baculovirus to phosphorylate a biotinylated peptide substrate containing the consensus phosphorylation site for histone H1, which is phosphorylated in vivo by CDK1. Inhibition of CDK1 activity is measured by observing a decreased amount of 33P-γ-ATP incorporation into the immobilized substrate in streptavidin-coated 96-well scintillating microplates. CDK1 enzyme is diluted in 50 mM Tris-HCl (pH 8), 10 mM MgCl2, 0.1 mM Na3VO4, 1 mM DTT, 1% DMSO, 0.25 μM peptide, 0.1 μCi per well 33P-γ-ATP, and 5 μM ATP in the presence or absence of various concentrations of JNJ-7706621 and incubated at 30 °C for 1 hour. The reaction is terminated by washing with PBS containing 100 mM EDTA and plates are counted in a scintillation counter. IC50 is determined by Linear regression analysis of the percent inhibition by JNJ-7706621.The Aurora kinase activity is measured with 10 μM ATP and a

	peptide containing a dual repeat of the kemptide phosphorylation motif.

Solubility Information

Solubility	H2O: < 1 mg/mL (insoluble or slightly soluble), Ethanol: 16 mg/mL (37.42 mM),Sonication is recommended. DMSO: 60 mg/mL (140.34 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.339 mL	11.6948 mL	23.3896 mL
5 mM	0.4678 mL	2.339 mL	4.6779 mL
10 mM	0.2339 mL	1.1695 mL	2.339 mL
50 mM	0.0468 mL	0.2339 mL	0.4678 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Reference

Undrovinas AI, et al. J Cardiovasc Electrophysiol,2006, 17 Suppl 1, S169-S177.
Lenaeus M, Gamal El-Din T M, Tonggu L, et al.Structural basis for inhibition of the cardiac sodium channel by the atypical antiarrhythmic drug ranolazine.Nature Cardiovascular Research.2023: 1-8.
Song Y, et al. J Cardiovasc Pharmacol,2004, 44(2), 192-199.
Baptista T, et al. Circulation,1996, 93(1), 135-142.