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Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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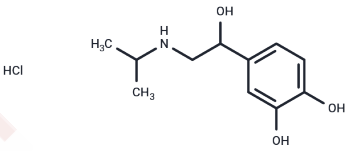
Data Sheet (Cat.No.T1056)

TargetMol

Isoprenaline hydrochloride

Chemical Properties



CAS No. : 51-30-9

Formula: C11H18ClNO3

Molecular Weight: 247.719

Appearance: no data available

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

Biological Description

Description	Isoprenaline hydrochloride (NCI-c55630) is a potent beta-adrenergic agonist with the peripheral vasodilator, bronchodilator, and cardiac stimulating properties.
Targets(IC50)	PI3K,Endogenous Metabolite,Adrenergic Receptor,PDE
In vitro	Incubation of intact rat fat cells with isoprenaline (300 nM, 3 min) increased particulate cGMP- and cilostamide-inhibited, low-Km cAMP phosphodiesterase (cAMP-PDE) activity by about 50% and 100%, respectively [1]. Relaxation induced by isoprenaline was also potentiated by the cyclic GMP-inhibited PDE (PDE 3) inhibitor cilostamide (100 nM). Isoprenaline (5 nM and 10 microM) increased cyclic AMP levels and this effect was potentiated by cilostamide (10 microM), by rolipram, a cyclic AMP-specific PDE (PDE 4) inhibitor (10 microM) and by cyclic GMP-elevating agents (50 nM ANF or 30 nM SNP plus 100 nM DMPPO) [2]. Isoprenaline increased the phosphorylation levels of Akt, and the downstream FoxO1, FoxO3a, and CREB. When catecholamine binding to β-adrenoceptors, the G protein-coupled receptor kinase-2 (GRK2) mediates the translocation of PI3K to β-adrenoceptors and then enhances the recruitment of β-arrestin and AP-2, which finally results in the internalization and downregulation of β-adrenoceptors. It has reported that disrupts the interaction between PI3K and GRK2 by displacing class I PI3K isoforms blocks agonist-stimulated β-adrenoceptors internalization [3].
Cell Research	Cells are seeded in 24-well culture dishes at a density of 2 to 5×10^4 cells per well. Experiments are performed after 3 to 5 days in culture when cells has reached confluence. Culture medium is aspirated and replaced by 0.5 mL of PBS containing the pharmacological agents. Treatments are performed in quadruplicate at 37°C. The type 3, 4 and 5 PDE inhibitors cilostamide (10 gM), rolipram (10 pM) and DMPPO (10 gM), respectively, are incubated with cells for 30 min before addition of adenylate or guanylate cyclase activators. Cyclic GMP and cyclic AMP are respectively increased in RASMC by stimulation of particulate guanylate cyclase with ANF (50 nM for 10 min) or fl-adrenoceptors with isoprenaline (5 nm for 5 min). At the end of the incubation period, the medium is removed and intracellular cyclic nucleotides are extracted by two ethanolic (65%) ishes at 4°C for 5 min. Ethanolic extracts are pooled, evaporated to dryness by a Speed-Vac system. The dried extract is dissolved in a suitable amount of assay buffer and cyclic nucleotide levels are measured by scintillation proximity assay [3].

Solubility Information

Solubility	H2O: 201.8 mM, DMSO: 60 mg/mL (242.21 mM), (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	4.0368 mL	20.1841 mL	40.3682 mL
5 mM	0.8074 mL	4.0368 mL	8.0736 mL
10 mM	0.4037 mL	2.0184 mL	4.0368 mL
50 mM	0.0807 mL	0.4037 mL	0.8074 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

Cai S, Chang J, Su M, et al.miR-455-5p promotes pathological cardiac remodeling via suppression of PRMT1-mediated Notch signaling pathway.Cellular and Molecular Life Sciences.2023, 80(12): 359.