Rostafuroxin

Cat. No.: HY-12283

CAS No.: 156722-18-8

Molecular Formula: $C_{23}H_{34}O_4$

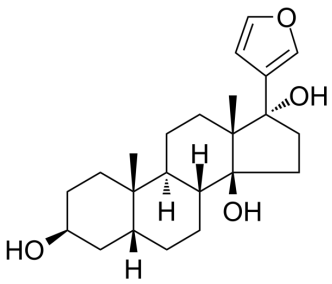
Molecular Weight: 374.51

Target: Na+/K+ ATPase; RSV

Pathway: Membrane Transporter/Ion Channel; Anti-infection

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (133.51 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6702 mL	13.3508 mL	26.7016 mL
	5 mM	0.5340 mL	2.6702 mL	5.3403 mL
	10 mM	0.2670 mL	1.3351 mL	2.6702 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 (6.68 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.68 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.68 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Rostafuroxin (PST 2238), a digitoxigenin derivative, is an orally active and potent Na^+,K^+-ATPase (ATP1A1) antognist. Rostafuroxin binds specifically to the ATP1A1 extracellular domain and blocks respiratory syncytial virus (RSV)-triggered EGFR Tyr845 phosphorylation. Rostafuroxin has antihypertensive and anti-RSV activity[1][2][3][4].

In Vitro

Rostafuroxin (PST 2238) competitively inhibits Ouabain (HY-B0542) binding and signaling. Rostafuroxin antagonizes the molecularand functional effects of Ouabain by reversing the ouabain-induced, Src-dependent Na^+,K^+-ATPase

phosphorylation and activation[3][4].
Rostafuroxin (0.125-128 μM; for 24 h post treatment) has less than 20% reduction in cell viability in A549 cells and HSAEC.
Rostafuroxin inhibits the expression of RSV-GFP in HSAEC (IC_{50}=1.8 μM) and A549 cells (IC_{50}=14.8 μM)[3].
Rostafuroxin displaced [^{3}H]Ouabain from the dog kidney Na^+,K^+-ATPase receptor (IC50=1.5 nM), is devoid of cardiac inotropic activity in isolated guinea pig atria, and shows no affinity up to 10^{-4} M with general (R1, R2, a1, a2, A1, A2, M1, M2, H1, H2, 5-HT1, 5-HT2, Ca2+ channels, TXA2/PGH2, PAF, GABAA, GABAB, DA-NE-5-HT uptake, glutammate,glycine, benzodiazepine) and hormonal (estrogenic, progestinic, androgenic, mineralcorticoid) receptors[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Rostafuroxin (PST 2238; 1 mg/kg/day; gavage; for 3 weeks) decreases SBP and improves acetylcholine-induced relaxation[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male 7-week-old Wistar rats[4]
Dosage:	1 mg/kg
Administration:	Gavage; daily; for 3 weeks
Result:	Decreased SBP, improved acetylcholine-induced relaxation via enhanced nitric oxide synthesis and bioavailability, decreased superoxide anion generation from NAD(P)H oxidase and cyclooxygenase-2 and reduced cytoplasmic tyrosine kinase Src phosphorylation.

CUSTOMER VALIDATION

- Int J Mol Sci. 2023 Feb 16;24(4):4000.

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REFERENCES

[1]. Quadri L, et al. 17 beta-(3-furyl)-5 beta-androstane-3 beta, 14 beta, 17 alpha-triol (PST 2238). A very potent antihypertensive agent with a novel mechanism of action. J Med Chem. 1997 May 23;40(11):1561-4.

[2]. Ferrari P, et al. PST 2238: A new antihypertensive compound that modulates Na,K-ATPase in genetic hypertension. J Pharmacol Exp Ther. 1999 Mar;288(3):1074-83.

[3]. Matthias Lingemann, et al. The alpha-1 subunit of the Na^+,K^+-ATPase (ATP1A1) is required for macropinocytic entry of respiratory syncytial virus (RSV) in human respiratory epithelial cells. PLoS Pathog. 2019 Aug 5;15(8):e1007963.

[4]. Camilla F Wenceslau, et al. Rostafuroxin ameliorates endothelial dysfunction and oxidative stress in resistance arteries from deoxycorticosterone acetate-salt hypertensive rats: the role of Na^+,K^+-ATPase/cSRC pathway. J Hypertens. 20

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