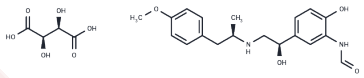
Data Sheet (Cat.No.T22245)

TargetMol

Arformoterol Tartrate

Chemical Properties



CAS No. : 200815-49-2

Formula: C23H30N2O10

Molecular Weight: 494.49

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Arformoterol Tartrate ((R,R)-Formoterol tartrate) is the tartrate salt of arformoterol. Arformoterol is a long-acting beta-2 adrenergic agonist with bronchodilator activity[2].
Targets(IC50)	Adrenergic Receptor
In vivo	Arformoterol (R,R-formoterol) is a active isomer of racemic formoterol and is indicated for the long-term, maintenance treatment of bronchoconstriction in patients with COPD including chronic bronchitis and emphysema. It is a potent and selective agent which causes bronchial smooth muscle relaxation and inhibits the release of inflammatory mediators. Its pharmacological effects can be attributed to the increased intracellular cyclic adenosine monophosphate (cAMP) levels that result from the stimulation of intracellular adenyl cyclase. Arformoterol tartrate is well absorbed through lungs when administered by a nebulizer. The mean peak plasma concentration (Cmax) and systemic exposure (AUC0-12h) are 4.3 pg/mL and 34.5 pg.h/mL, respectively, when 15 µg arformoterol is administered every 12 h for 14 days in COPD patients. The time to achieve median steady state peak plasma concentration (tmax) is approximately half an hour after drug administration. The mean terminal half-life is 26 h in COPD patients when treated with 15 µg inhaled arformoterol twice daily for 14 days. The binding of arformoterol to human plasma proteins in vitro is 52-65% at concentrations of 0.25, 0.5 and 1.0 ng/mL of radiolabeled arformoterol. Metabolism occurs primarily by direct conjugation (glucuronidation) and secondary route of metabolism is via O-demethylation. Metabolism is mediated by atleast five human uridine diphosphoglucuronosyltransferase (UGT) isozymes as well as CYP2D6 and CYP2C19. After administration of a single oral dose of radiolabeled arformoterol, 63% of the radioactive amount was recovered in urine and 11% in feces within 48 h. A total of 89% of the total radioactive dose was recovered within 14 days, with 67% in urine and 22% in faeces[1].
Animal Research	Exposure of C57BL/6 mice(8 wk old) to 400 ppm Cl(2) for 30 minutes increased respiratory system resistance and airway responsiveness to aerosolized methacholine. Intranasal administration of arformoterol mitigated the Cl(2) effects on airway reactivity and AFC, presumably by increasing lung cyclic AMP level. Arformoterol did not modify the inflammatory responses, as evidenced by the number of inflammatory cells and concentrations of IL-6 and TNF-α in the bronchoalveolar lavage. NF-κB activity (assessed by p65 Western blots and electrophoretic mobility shift assay) remained at control levels up to 24 hours after Cl(2) exposure. Our results provide mechanistic insight

Animal Research	effectiveness of long-term β(2)-agonists in reversing Cl(2)-induced reactive airway dysfunction syndrome and injury to distal lung epithelial cells[2].

Solubility Information

Solubility	DMSO: 55 mg/mL (111.23 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.0223 mL	10.1114 mL	20.2229 mL
5 mM	0.4045 mL	2.0223 mL	4.0446 mL
10 mM	0.2022 mL	1.0111 mL	2.0223 mL
50 mM	0.0404 mL	0.2022 mL	0.4045 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.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Pahwa R , Soni V , Sharma P C , et al. Arformoterol Tartrate: A Review of Pharmacology, Analysis and Clinical Studies[J]. Tropical Journal of Pharmaceutical Research, 2011, 9(6).

Song W, et al. Postexposure administration of a {beta}2-agonist decreases chlorine-induced airway hyperreactivity in mice. Am J Respir Cell Mol Biol. 2011 Jul;45(1):88-94.