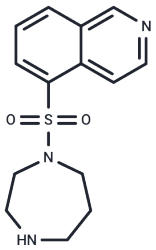
Data Sheet (Cat.No.T1606)

TargetMol

Fasudil

Chemical Properties



CAS No. : 103745-39-7

Formula: C14H17N3O2S

Molecular Weight: 291.37

Appearance: no data available

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

Biological Description

Description	Fasudil (HA-1077) is a potent inhibitor of ROCK1, PKA, PKC, and MLCK.
Targets(IC50)	Calcium Channel,Autophagy,PKA,PKC,ROCK,Serine/threonin kinase
In vitro	Fasudil (Hydrochloride) has vasodilatory action and occupies the adenine pocket of the ATP-binding site of the enzyme[1]. Fasudil is a class of calcium antagonists. Fasudil produces a competitive inhibition of the Ca2+-induced contraction of the depolarized rabbit aorta. Fasudil is able to inhibit contractile responses to KCl, phenylephnne (PHE) and prostaglandin (PG) F2a[2]. Fasudil also exhibits vasodilator actions by inhibition of 5-hydroxytryptamine, noradrenaline, histamine, angiotensin, and dopamine induced spiral strips contraction[3]. Fasudil induces disorganization of actin stress fiber and cell migration inhibition[4]. Fasudil inhibits hepatic stellate cells spreading, the formation of stress fibers, and expression of α-SMA with concomitant suppression of cell growth, but does not induce apoptosis. Fasudil suppresses the LPA-induced phosphorylation of ERK1/2, JNK and p38 MAPK[5].
In vivo	Fasudil (30 μg) produces an approximate 50% increase in CBF via intra-coronary injection to dogs. Fasudil (0.01, 0.03, 0.1 and 0.3 mg/kg, bolus, i.v.) dose-dependently decreases MBP and increases HR, VBF, CBF, RBF, and FBF. A total dose of 1.0 ng/mL Fasudil increases cardiac output. The infusion of Fasudil i.v. produces a significant fall in MBP, left ventricular systolic pressure and total peripheral resistance with an increase in HR and cardiac output, but without significant changes in right atrial pressure, dP/dt or left ventricular minute work in dogs[3]. Fasudil administration displays protectable effects on cardiovascular disease and reduces the activation of JNK and attenuates mitochondrial-nuclear translocation of AIF under ischemic injury[6]. The oral administration of Fasudil (a dosage of 100 mg/kg/day) significantly reduces incidence and mean maximum clinical score of EAE in SJL/J mice immunized with PLP p139-151. Treatment of mice with Fasudil suppresses the proliferative response of splenocytes to the antigen. Oral administration of Fasudil decreases inflammation, demyelination, axonal loss and APP positivein spinal cord of Fasudil-treated mice[7].
Kinase Assay	Cyclic AMP-dependent protein kinase activity is assayed in a reaction mixture containing, in a final volume of 0.2 mL, 50 mM Tris-HCl (pH 7.0), 10 mM magnesium acetate, 2 mM EGTA, 1 μM cyclic AMP or absence of cyclic AMP, 3.3 to 20 μM [r-32P] ATP (4×105 c.p.m.), 0.5 μg of the enzyme, 100 μg of histone H2B and compound. The mixture

is incubated at 30°C for 5 min. The reaction is terminated by adding 1mL of ice-cold 20% trichloroacetic acid after adding 500 μg of bovine serum albumin as a carrier protein. The sample is centrifuged at 3000 r.p.m. for 15min, the pellet is resuspended in ice-cold 10% trichloro-acetic acid solution and the centrifugation-resuspension cycle is repeated three times. The final pellet is dissolved in 1 mL of 1 N NaOH and radioactivity is measured with a liquid scintillation counter[1].

Solubility Information

Solubility	DMSO: Soluble, (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.4321 mL	17.1603 mL	34.3206 mL
5 mM	0.6864 mL	3.4321 mL	6.8641 mL
10 mM	0.3432 mL	1.716 mL	3.4321 mL
50 mM	0.0686 mL	0.3432 mL	0.6864 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

Ono-Saito N, et al. H-series protein kinase inhibitors and potential clinical applications. Pharmacol Ther. 1999 May-Jun;82(2-3):123-31.

Zhang S, Xiang H, Tao Y, et al.Inhibitor Development for α-Synuclein Fibril's Disordered Region to Alleviate Parkinson's Disease Pathology.Journal of the American Chemical Society.2024

Ge W, Zhang X, Lin J, et al.Rnd3 protects against doxorubicin-induced cardiotoxicity through inhibition of PANoptosis in a Rock1/Drp1/mitochondrial fission-dependent manner.Cell Death & Disease.2025, 16(1): 1-16.

Asano T, et al. Mechanism of action of a novel antivasospasm drug, HA1077. J Pharmacol Exp Ther. 1987 Jun;241 (3):1033-40.

Asano T, et al. Vasodilator actions of HA1077 in vitro and in vivo putatively mediated by the inhibition of protein kinase. Br J Pharmacol. 1989 Dec;98(4):1091-100.

Negoro N, et al. The kinase inhibitor fasudil (HA-1077) reduces intimal hyperplasia through inhibiting migration and enhancing cell loss of vascular smooth muscle cells. Biochem Biophys Res Commun. 1999 Aug 19;262(1):211-5.

Fukushima M, et al. Fasudil hydrochloride hydrate, a Rho-kinase (ROCK) inhibitor, suppresses collagen production and enhances collagenase activity in hepatic stellate cells. Liver Int. 2005 Aug;25(4):829-38.