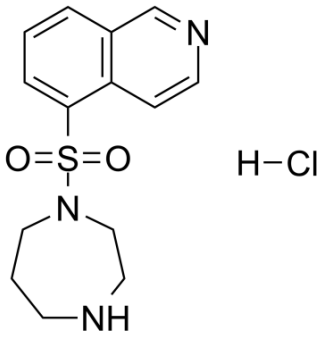
Fasudil Hydrochloride



Cat. No.:	HY-10341
CAS No.:	105628-07-7
Molecular Formula:	$C_{14}H_{18}ClN_3O_2S$
Molecular Weight:	327.83
Target:	ROCK; Calcium Channel; Autophagy; PKA; PKC
Pathway:	Cell Cycle/DNA Damage; Stem Cell/Wnt; TGF-beta/Smad; Membrane Transporter/Ion Channel; Neuronal Signaling; Autophagy; Protein Tyrosine Kinase/RTK; Epigenetics

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month

SOLVENT & SOLUBILITY

In Vitro

H_2O : 55 mg/mL (**167.77 mM;** Need ultrasonic)

DMSO : ≥ 31 mg/mL (**94.56 mM**)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.0504 mL	15.2518 mL	30.5036 mL
5 mM	0.6101 mL	3.0504 mL	6.1007 mL
10 mM	0.3050 mL	1.5252 mL	3.0504 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

Fasudil Hydrochloride (HA-1077 Hydrochloride; AT877 Hydrochloride), is a nonspecific **ROCK** inhibitor and also has inhibitory effect on protein kinases, with an **K_i** of 0.33 μM for ROCK1, **IC_{50}**s of 0.158 μM and 4.58 μM, 12.30 μM, 1.650 μM for ROCK2 and PKA, PKC, PKG, respectively[1]. Fasudil Hydrochloride is also a potent Ca^{2+} channel antagonist and vasodilator[2].

IC_{50} & Target

p160ROCK 0.33 μM (Ki)	ROCK2 0.158 μM (IC_{50})	PKA 4.58 μM (IC_{50})	PKC 12.30 μM (IC_{50})
PKG 1.65 μM (IC_{50})			

In Vitro

Fasudil Hydrochloride (100 μM) inhibits cell spreading, the formation of stress fibers, and expression of α-SMA with concomitant suppression of cell growth in rat HSCs and human HSC-derived TWNT-4 cells[4].
Fasudil Hydrochloride (50-100 μM; 24 hours) inhibits the LPA-induced phosphorylation of ERK1/2, JNK, and p38 detected by western blotting in rat HSCs and human HSC-derived TWNT-4 cells[4].
Fasudil Hydrochloride (25-100 μM; 24 hours) suppresses transcription of collagen and TIMP, stimulates transcription of MMP-1 in human HSC-derived TWNT-4 cells[4].

Western Blot Analysis[4]

Cell Line:	Rat HSCs and human HSC-derived TWNT-4 cells
Concentration:	50 μM; 100 μM
Incubation Time:	24 hours
Result:	Suppressed the LPA-induced phosphorylation of ERK1/2, JNK and p38 MAPK by 60%, 70%,and 90%, respectively.

RT-PCR[4]

Cell Line:	Rat HSCs and human HSC-derived TWNT-4 cells
Concentration:	25 μM; 50 μM; 100 μM 24 hours
Incubation Time:	24 hours
Result:	Reduced the expression of type I collagen, a-SMA, and TIMP-1.

In Vivo

Fasudil (30 μg) increases CBF by 50% via intra-coronary injection to dogs. Fasudil (0.01, 0.03, 0.1 and 0.3 mg/kg, bolus, i.v.) decreases MBP and increases HR, VBF, CBF, RBF, and FBF. Fasudil (1.0 ng/mL) increases cardiac output. Fasudil via 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 obvious effect on right atrial pressure, dP/dt or left ventricular minute work in dogs[3]. Fasudil exhibits protectable effects on cardiovascular disease and reduces the activation of JNK and attenuates mitochondrial-nuclear translocation of AIF under ischemic injury[6]. Fasudil (100 mg/kg/day, p.o.) significantly reduces incidence and mean maximum clinical score of EAE in SJL/J mice immunized with PLP p139-151. Fasudil inhibits the proliferative response of splenocytes to the antigen in mice. Fasudil decreases inflammation, demyelination, axonal loss and APP positivein spinal cord of Fasudil-treated mice via p.o. administration[7].

CUSTOMER VALIDATION

- **Sci Transl Med**. 2018 Jul 18;10(450). pii: eaaq1093.
- **Sci Rep**. 2018 Feb 6;8(1):2477.
- **Adipocyte**. 2019 Dec;8(1):114-124.
- **Biomed Rep**. 2015 May;3(3):361-364.

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[2]. Huang XN, et al. The effects of fasudil on the permeability of the rat blood-brain barrier and blood-spinal cordbarrier following experimental autoimmune encephalomyelitis. J Neuroimmunol. 2011 Oct 28;239(1-2):61-7.

[3]. Uehata M, et al. Calcium sensitization of smooth muscle mediated by a Rho-associated protein kinase in hypertension. Nature. 1997 Oct 30;389(6654):990-4.

[4]. 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.

[5]. Corbin KD, et al. Choline metabolism provides novel insights into nonalcoholic fatty liver disease and its progression. Curr Opin Gastroenterol. 2012 Mar;28(2):159-65.

[6]. Zhang J, et al. Inhibition of the activity of Rho-kinase reduces cardiomyocyte apoptosis in heart ischemia/reperfusion via suppressing JNK-mediated AIF translocation. Clin Chim Acta. 2009 Mar;401(1-2):76-80.

[7]. Sun X, et al. The selective Rho-kinase inhibitor Fasudil is protective and therapeutic in experimental autoimmune encephalomyelitis. J Neuroimmunol. 2006 Nov;180(1-2):126-34. Epub 2006 Sep 22.

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