

Fasudil (Hydrochloride)

Catalog No: tcsc0225



Available Sizes

Size: 200mg

Size: 500mg



Specifications

CAS No:

105628-07-7

Formula:

$C_{14}H_{18}ClN_3O_2S$

Pathway:

Stem Cell/Wnt;Protein Tyrosine Kinase/RTK;TGF-beta/Smad;Stem Cell/Wnt;Cell Cycle/DNA Damage;Autophagy

Target:

PKA;PKA;ROCK;ROCK;ROCK;Autophagy

Purity / Grade:

>98%

Solubility:

DMSO : ≥ 31 mg/mL (94.56 mM); H₂O : 55 mg/mL (167.77 mM; Need ultrasonic)

Alternative Names:

HA-1077;AT-877;Fasudil HCl

Observed Molecular Weight:

327.83

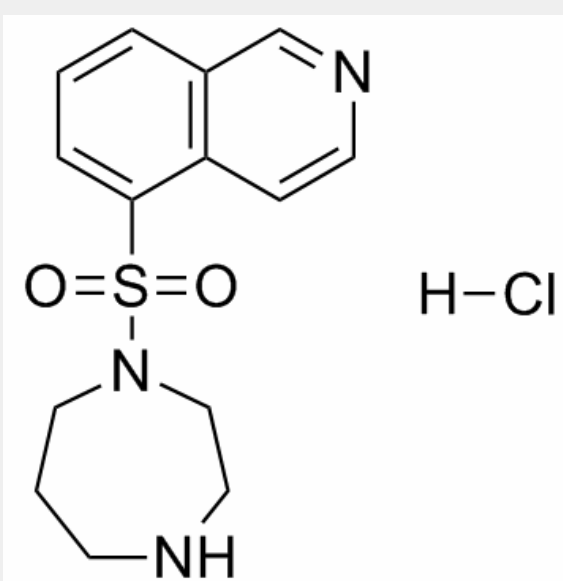
Product Description

Fasudil Hydrochloride is a potent inhibitor of **ROCK1**, **PKA**, **PKC**, and **MLCK** with **K_i**s of 0.33 μ M, 1.0 μ M, 9.3 μ M and 55 μ M, respectively.

IC50 & Target: Ki: 0.33 μ M (ROCK1), 1.0 μ M (PKA), 9.3 μ M (PKC), 55 μ M (MLCK)^[8]

In Vitro: Fasudil Hydrochloride has vasodilatory action and occupies the adenine pocket of the ATP-binding site of the enzyme^[1]. Fasudil produces a competitive inhibition of the Ca^{2+} -induced contraction of the depolarized rabbit aorta. Fasudil inhibits contractile responses to KCl, phenylephrine (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]. In addition, 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 also blocks the LPA-induced phosphorylation of ERK1/2, JNK and p38 MAPK^[5].

In Vivo: Fasudil (30 μ g) 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 positive in spinal cord of Fasudil-treated mice via p.o. administration^[7].



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