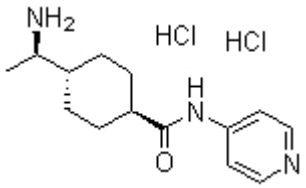
Product Introduction

Y-27632 2HCl

Y-27632 2HCl is a selective **ROCK1 (p160ROCK)** inhibitor with K_I of 140 nM, exhibits >200-fold selectivity over other kinases, including PKC, cAMP-dependent protein kinase, MLCK and PAK.

Technical Data:

Molecular Weight (MW):	320.26	
Formula:	$C_{14}H_{21}N_3O.2HCl$	
Solubility (25°C) * <1 mg/ml means slightly soluble or insoluble:	DMSO 64 mg/mL	
	Water 64 mg/mL	
	Ethanol <1 mg/mL	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	129830-38-2	

Biological Activity

Y-27632 2HCl inhibits ROCK-II while displaying little activity against PKC, cAMP-dependent protein kinase and myosin light-chain kinase (MLCK) with K_i of 26 μM, 25 μM and > 250 μM, respectively, as well as PKA activated by another Rho-family GTPase member, Cdc42. Y-27632 2HCl inhibits smooth-muscle contraction induces by various agonists including phenylephrine, histamine, acetylcholine, serotonin, endothelin, and thromboxane with IC50 of 0.3-1 μM, by selectively inhibiting Ca^{2+} sensitization. Y-27632 2HCl suppresses Rho-induced, p160ROCK-mediated formation of stress fibres in cultured cells. [1] Y-27632

2HCl treatment blocks both Rho-mediated activation of actomyosin and LPA-stimulated invasive activity of MM1 cells in a concentration-dependent manner. [2] Y-27632 2HCl treatment is not only sufficient to initiate formation of exuberant axonal processes but also facilitates axonal maturation during the very early stages of axonogenesis, while largely sparing axon elongation. [3] In human embryonic stem (hES) cells, Y-27632 2HCl treatment at 10 μM markedly diminishes dissociation-induced apoptosis even in serum-free suspension (SFEB) culture, increases cloning efficiency (from ~1% to ~27%), facilitates subcloning after gene transfer, and enables SFEB-cultured hES cells to survive and differentiate into $Bf1^+$ cortical and basal telencephalic progenitors. [4]

Oral administration of Y-27632 2HCl at 30 mg/kg significantly decreases the blood pressure in a dose-dependent manner in spontaneous hypertensive rats, renal hypertensive rats, as well as deoxycorticosterone acetate (DOCA)-salt hypertensive rats. [1] When Y-27632 2HCl is continuously administered at a rate of 0.55 μL per hour by implanted pumps for 11 days tumor cell invasion (MM1 cells expressing Val14-RhoA in rats) is significantly delayed. [2] By inhibiting ROCK, Y-27632 2HCl treatment attenuates hypoxia-induced angiogenesis and vascular remodeling in the pulmonary circulation. [5]

References

[1] Uehata M, et al. Nature, 1997, 389(6654), 990-994.

[2] Itoh K, et al. Nat Med, 1999, 5(2), 221-225.

[3] Bito H, et al. Neuron, 2000, 26(2), 431-441.

[4] Watanabe K, et al. Nat Biotechnol, 2007, 25(6), 681-686.

[5] Hyvelin JM, et al. Circ Res, 2005, 97(2), 185-191.

[6] Ishizaki T, et al. Mol Pharmacol. 2000, 57(5), 976-983.

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