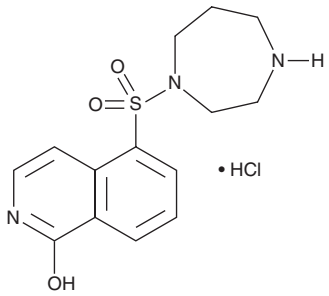
PRODUCT INFORMATION

HA-1100 (hydrochloride)

Item No. 14413

CAS Registry No.:	155558-32-0
Formal Name:	5-[(hexahydro-1H-1,4-diazepin-1-yl)sulfonyl]-1(2H)-isoquinolinone, monohydrochloride
MF:	$C_{14}H_{17}N_3O_3S \bullet HCl$
FW:	343.8
Purity:	≥98%
UV/Vis.:	λ_{max}: 246, 300 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

HA-1100 (hydrochloride) is supplied as a A crystalline solid. A stock solution may be made by dissolving the HA-1100 (hydrochloride) in the solvent of choice, which should be purged with an inert gas. HA-1100 (hydrochloride) is soluble in the organic solvent DMSO. The solubility of HA-1100 (hydrochloride) in DMSO is approximately 1.1 mg/ml.

HA-1100 (hydrochloride) is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, HA-1100 (hydrochloride) should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. HA-1100 (hydrochloride) has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

HA-1100, a cell-permeable hydroxylated metabolite of HA-1077 (Item No. 10010559), is a potent inhibitor of Rho-associated kinase (ROCK) (K_i = 150 nM).[1] It less effectively inhibits protein kinase A (K_i = 2.2 µM).[1] It has been used to explore the role of ROCK in hypoxic and ischemic signaling as well as arterial relaxation.[2-5]

References

1. Jacobs, M., Hayakawa, K., Swenson, L., *et al.* The structure of dimeric ROCK I reveals the mechanism for ligand selectivity. *J. Biol. Chem.* **281(1)**, 260-268 (2006).
2. Nakamura, K., Nishimura, J., Hirano, K., *et al.* Hydroxyfasudil, an active metabolite of fasudil hydrochloride, relaxes the rabbit basilar artery by disinhibition of myosin light chain phosphatase. *J. Cereb. Blood Flow Metab.* **21(7)**, 876-885 (2001).
3. Takemoto, M., Sun, J., Hiroki, J., *et al.* Rho-kinase mediates hypoxia-induced downregulation of endothelial nitric oxide synthase. *Circulation* **106(1)**, 57-62 (2002).
4. Wolfrum, S., Dendorfer, A., Rikitake, Y., *et al.* Inhibition of rho-kinase leads to rapid activation of phosphatidylinositol 3-kinase/protein kinase Akt and cardiovascular protection. *Arterioscler. Thromb. Vasc. Biol.* **24(10)**, 1842-1847 (2004).
5. Satoh, S., Utsunomiya, T., Tsurui, K., *et al.* Pharmacological profile of hydroxy fasudil as a selective rho kinase inhibitor on ischemic brain damage. *Life Sci.* **69(12)**, 1441-1453 (2001).

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