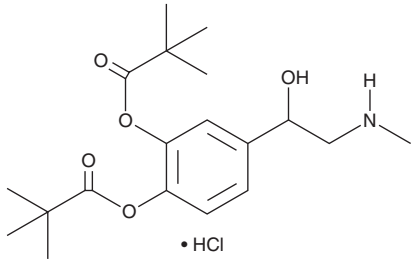
PRODUCT INFORMATION

Dipivefrin (hydrochloride)

Item No. 24028

CAS Registry No.: 64019-93-8
Formal Name: 2,2-dimethyl-propanoic acid, 1,1'-[4-[1-hydroxy-2-(methylamino)ethyl]-1,2-phenylene] ester, monohydrochloride
MF: $C_{19}H_{29}NO_5 \bullet HCl$
FW: 387.9
Purity: ≥98%
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

Dipivefrin (hydrochloride) is supplied as a crystalline solid. A stock solution may be made by dissolving the dipivefrin (hydrochloride) in the solvent of choice. Dipivefrin (hydrochloride) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of dipivefrin (hydrochloride) in these solvents is approximately 33 mg/ml.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of dipivefrin (hydrochloride) can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of dipivefrin (hydrochloride) in PBS, pH 7.2, is approximately 10 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Dipivefrin is a prodrug of epinephrine that is hydrolyzed by cholinesterase and other esterases in the cornea to epinephrine.[1] It reduces the density of cultured bovine primary trabecular meshwork cells (IC_{50} = 115 µM).[2] Additionally, it induces an elongated, fibroblast-like morphology and disrupts the actin cytoskeleton in bovine primary trabecular meshwork cells when used at a concentration of 103 µM.[2] In cultured bovine corneal endothelial cells, dipivefrin (28 µM) enhances calcium signaling and induces cytotoxicity.[3,4] Dipivefrin also suppresses primary human corneal keratinocyte proliferation when used at a concentration of 280 µM.[5] Formulations containing dipivefrin have been used alone and in combination with β-adrenergic receptor antagonists for the treatment of glaucoma.

References

1. Nakamura, M., Shirasawa, E., and Hikida, M. Characterization of esterases involved in the hydrolysis of dipivefrin hydrochloride. *Ophthalmic Res.* **25(1)**, 46-51 (1993).
2. Kawa, J.E., Higginbotham, E.J., Change, I.L., *et al.* Effects of antiglaucoma medications on bovine trabecular meshwork cells in vitro. *Exp. Eye Res.* **57(5)**, 557-565 (1993).
3. Wu, K.Y., Hong, S.J., and Wang, H.Z. Effects of antiglaucoma drugs on calcium mobility in cultured corneal endothelial cells. *Kaohsiung J. Med. Sci.* **22(2)**, 60-67 (2006).
4. Wu, K.Y., Wang, H.Z., and Hong, S.J. Cellular cytotoxicity of antiglaucoma drugs in cultured corneal endothelial cells. *Kaohsiung J. Med. Sci.* **23(3)**, 105-111 (2007).
5. Wu, K.Y., Wang, H.Z., and Hong, S.J. Effects of antiglaucoma drugs on cellular proliferation in cultured human corneal keratocytes. *Kaohsiung J. Med. Sci.* **22(3)**, 120-125 (2006).

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