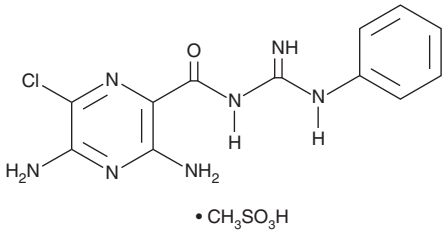
PRODUCT INFORMATION

Phenamil (methanesulfonate)

Item No. 14308

CAS Registry No.: 1161-94-0
Formal Name: 3,5-diamino-6-chloro-N-[imino(phenylamino)methyl]-2-pyrazinecarboxamide, monomethanesulfonate
MF: $C_{12}H_{12}ClN_7O \bullet CH_3SO_3H$
FW: 401.8
Purity: ≥98%
UV/Vis.: λ_{max}: 289, 362 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

Phenamil (methanesulfonate) is supplied as a crystalline solid. A stock solution may be made by dissolving the phenamil (methanesulfonate) in the solvent of choice. Phenamil (methanesulfonate) is soluble in organic solvents such as DMSO and dimethyl formamide, which should be purged with an inert gas. The solubility of phenamil (methanesulfonate) in these solvents is approximately 1 and 0.1 mg/ml, respectively.

Phenamil (methanesulfonate) is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, phenamil (methanesulfonate) should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Phenamil (methanesulfonate) 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

Phenamil is an inhibitor of transient receptor potential polycystin-L (TRPP3; IC_{50} = 140 nM) and a derivative of amiloride (Item No. 14409).[1] It also inhibits the epithelial sodium channel (ENaC; IC_{50} = 400 nM).[2] Phenamil decreases basal short-circuit currents in human and ovine bronchial epithelial cells with IC_{50} values of 75 and 116 nM, respectively.[3] It inhibits potassium chloride-induced contractions in isolated rat endothelium-denuded aortic rings (EC_{50} = 6.76 µM) and increases contractile force in isolated rat right ventricular papillary muscles (EC_{50} = 16.98 µM).[4] Phenamil (15 and 30 mg/kg per day) reduces pulmonary artery medial wall thickness and decreases right ventricular peak pressure in a rat model of chronic hypoxia-induced pulmonary hypertension.[5]

References

1. Dai, X. Q., Ramji, A., Liu, Y., *et al. Mol. Pharmacol.* **72(6)**, 1576-1585 (2007).
2. Hirsh, A.J., Molino, B.F., Zhang, J., *et al. J. Med. Chem.* **49(14)**, 4098-4115 (2006).
3. Hirsh, S.J., Sabater, J.R., Zamurs, A., et al. *J. Pharmacol. Exp. Ther.* **311(3)**, 929-938 (2004).
4. Brown, L., Cragoe, E.J., Jr., Abel, K.C., *et al. Naunyn Schmiedebergs Arch Pharmacol.* **344(2)**, 220-224 (1991).
5. Chan, M.C., Weisman, A.S., Kang, H., *et al. Mol. Cell. Biol.* **31(3)**, 517-530 (2011).

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