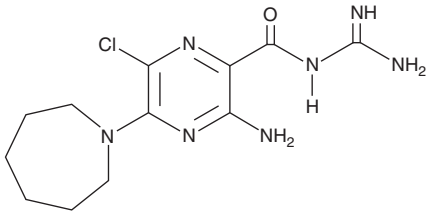
PRODUCT INFORMATION

5-(N,N-hexamethylene)-Amiloride

Item No. 29788

CAS Registry No.: 1428-95-1
Formal Name: 3-amino-N-(aminoiminomethyl)-6-chloro-5-(hexahydro-1H-azepin-1-yl)-2-pyrazinecarboxamide
Synonym: HMA
MF: $C_{12}H_{18}ClN_7O$
FW: 311.8
Purity: ≥98%
UV/Vis.: λ_{max}: 232, 295, 375 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

5-(N,N-hexamethylene)-Amiloride (HMA) is supplied as a crystalline solid. A stock solution may be made by dissolving the HMA in the solvent of choice, which should be purged with an inert gas. HMA is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of HMA in these solvents is approximately 10 and 3 mg/ml, respectively.

HMA is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, HMA should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. HMA has a solubility of approximately 0.2 mg/ml in a 1:4 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

HMA is a derivative of amiloride (Item No. 14409) with diverse biological activities.[1-5] It is an allosteric antagonist of adenosine A_{2A} receptors (K_i = 3.3 µM).[2] HMA inhibits the cation-selective ion channel formed by the HIV-1 viral protein Vpu when used at a concentration of 50 µM, as well as budding of virus-like particles in HeLa cells expressing the HIV-1 proteins Gag and Vpu when used at a concentration of 10 µM.[1] It also blocks the cation-selective ion channels formed by the hepatitis C virus (HCV) protein p7.[3] HMA (40 µM) induces necrosis in and reduces the viability of MCF-7, MDA-MB-231, T47D, SK-BR-3, Met-1, and NDL breast cancer cells but not cardiomyocytes or uterine, pulmonary, and renal epithelial cells.[4] HMA protects against post-ischemic contractile dysfunction and reduces coronary effluent creatine phosphokinase activity in a model of ischemia-reperfusion injury using isolated rat right ventricular free walls.[5]

References

1. Ewart, G.D., Mills, K., Cox, G.B., *et al. Eur. Biophys. J.* **31(1)**, 26-35 (2002).
2. Gao, Z.-G. and Ijzerman, A.P. *Biochem. Pharmacol.* **60(5)**, 669-679 (2000).
3. Premkumar, A., Wilson, L., Ewart, G.D., *et al. FEBS Lett.* **557(1-3)**, 99-103 (2004).
4. Rowson-Hodel, A.R., Berg, A.L., Wald, J.H., *et al. Cancer Lett.* **375(1)**, 62-72 (2016).
5. Meng, H.-P., Maddaford, T.G., and Pierce, G.N. *Am. J. Physiol.* **264(6 Pt. 2)**, H1831-H1835 (1993).

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