PRODUCT INFORMATION

Cl-Amidine (hydrochloride)

Item No. 10599

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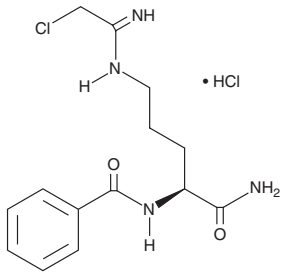
CAS Registry No.: 1373232-26-8
Formal Name: N-[(1S)-1-(aminocarbonyl)-4-[(2-chloro-1-iminoethyl)amino]butyl]-benzamide, monohydrochloride
MF: $C_{14}H_{19}ClN_4O_2 \bullet HCl$
FW: 347.2
Purity: ≥95%
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥2 years



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

Laboratory Procedures

Cl-Amidine (hydrochloride) is supplied as a crystalline solid. A stock solution may be made by dissolving the Cl-amidine (hydrochloride) in the solvent of choice. Cl-Amidine (hydrochloride) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of Cl-amidine (hydrochloride) in these solvents is approximately 20, 50, and 14 mg/ml, respectively.

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 Cl-amidine (hydrochloride) can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of Cl-amidine (hydrochloride) in PBS, pH 7.2, is approximately 3 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Cl-Amidine is an irreversible inhibitor of protein arginine deiminases (PADs; IC_{50}s = 0.8, 6.2, and 5.9 µM for PAD1, PAD3, and PAD4, respectively).[1-3] It is cytotoxic to HL-60, MCF-7, and HT-29 cancer cells (IC_{50}s = 0.25, 0.05, and 1 µM, respectively).[4] Cl-amidine (50 mg/kg) reduces *ex vivo* extracellular neutrophil extracellular trap (NET) formation and increases survival in a mouse model of sepsis induced by cecal ligation and puncture (CLP).[6] It also decreases the citrulline content in serum and joints and reduces the development of IgG autoantibodies in a mouse model of collagen-induced arthritis in a dose-dependent manner.[5]

References

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4. Slack, J.L., Causey, C.P., and Thompson, P.R. *Cell. Mol. Life Sci.* **68(4)**, 709-720 (2011).
5. Willis, V.C., Gizinski, A.M., Banda, N.K., *et al. J. Immunol.* **186**, 4396-4404 (2011).
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