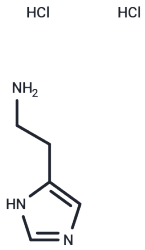
Data Sheet (Cat.No.T6534)

TargetMol

Histamine dihydrochloride

Chemical Properties



CAS No. : 56-92-8

Formula: C5H9N3·2HCl

Molecular Weight: 184.07

Storage: Pure form: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Histamine dihydrochloride (Ceplene) is the dihydrochloride salt form of histamine, an endogenous metabolic product. Histamine is an organic nitrogen-containing compound with diverse biological activities. It acts as a potent stimulator of gastric acid secretion, a bronchial smooth muscle constrictor, and a vasodilator, as well as a neurotransmitter with central nervous system activity. It is commonly used to induce gastric ulcer models in research.
Targets(IC50)	Endogenous Metabolite,Histamine Receptor
In vitro	Histamine suppresses the generation of ROS through the Histaminetype-2 receptor (H2 receptor).[1] Histamine inhibits the generation and release of reactive oxygen species (ROS) by monocytes/macrophages (MO) during respiratory burst. Histamine and interleukin-2 (IL-2) act synergistically to activate NK cell cytotoxicity (NKCC). Histamine combined with IL-2 might improve response rates and disease-free survival by protecting the cells of the immune system from oxidative stress and inducing natural endogenous immune cytotoxicity. [2]
In vivo	Histamine treatment (0.5 mg/kg or 5.0 mg/kg, twice daily) protects against liver injury as evident by normal serum transaminase levels and significantly reduced liver pathology scores in a rat model with early alcohol-induced liver injury. The protective effect of histamine is blocked by Ranitidine (10 mg/kg), an H2 receptor antagonist, indicating that the histamine effect is predominantly mediated through the H2 receptor. [1] Histamine (30 pg/rat, icv) increases both 3,4-dihydroxyphenylalanine accumulation and 3,4-dihydroxyphenylalanine acid concentrations in the nucleus accumbens in male rats, and this effect is not affect by H2 antagonist zolantidine, indicating that histamine stimulates mesolimbic DA neurons through an action at the H1 receptor. [3] Histamine (0.5 mg/kg s.c.) reduces the liver tumour weight by 46% and subcutaneous tumour weight by 41% versus rats receiving subcutaneous saline injections. The anti-tumour effect observed by subcutaneous histamine injections is inhibited by Ranitidine (50 mg/kg s.c.) in rats sarcoma. [4] Histamine (1000 mg/kg s.c.) displays acute tissue damage after 24 hours and indications of pathological inflammation at the injection sites at 5 days and 28 days in Sprague-Dawley rats. Histamine (1000 mg/kg s.c.) results in Cmax of 167 mM, tmax of 0.5 hour, t1/2 of 0.95 and AUC of 186 mmol-h/L in male Sprague-Dawley rats. [5]

Solubility Information

Solubility	H2O: 18.4 mg/mL (99.96 mM),Sonication is recommended. DMSO: 70 mg/mL (380.29 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (10.87 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	5.4327 mL	27.1636 mL	54.3272 mL
5 mM	1.0865 mL	5.4327 mL	10.8654 mL
10 mM	0.5433 mL	2.7164 mL	5.4327 mL
50 mM	0.1087 mL	0.5433 mL	1.0865 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Hornyak SC, et al. Inflammation, 2003, 27(5), 317-327.
Agarwala SS, et al. Expert Opin Biol Ther, 2001, 1(5), 869-879.
Fleckenstein AE, et al. Naunyn Schmiedebergs Arch Pharmacol, 1993, 347(1), 50-54.
Rizell M, et al. Anticancer Res, 2002, 22(4), 1943-1948.
Karavodin L, et al. Drug Chem Toxicol, 2003, 26(1), 35-49.