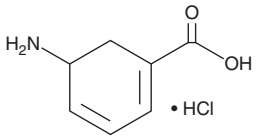
PRODUCT INFORMATION

Gabaculine (hydrochloride)

Item No. 24209

CAS Registry No.: 59556-17-1
Formal Name: 5-amino-1,3-cyclohexadiene-1-carboxylic acid, monohydrochloride
MF: $C_7H_9NO_2$ • HCl
FW: 175.6
Purity: ≥95%
UV/Vis.: λ_{max}: 276 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

Gabaculine (hydrochloride) is supplied as a crystalline solid. A stock solution may be made by dissolving the gabaculine (hydrochloride) in the solvent of choice, which should be purged with an inert gas. Gabaculine (hydrochloride) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of gabaculine (hydrochloride) in ethanol is approximately 0.2 mg/ml and approximately 20 mg/ml in DMSO and DMF.

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 gabaculine (hydrochloride) can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of gabaculine (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

Gabaculine is a naturally occurring, conformationally constrained analog of GABA and an irreversible inhibitor of GABA transaminase (GABA-T; K_i = 2.9 µM).[1] It irreversibly inhibits D-amino acid transaminase, L-alanine transaminase, and L-aspartate transaminase with K_i values of 0.1, 1, and 55 mM, respectively.[2] Gabaculine also irreversibly inhibits ornithine aminotransferase *in vitro* and in mouse brain and liver homogenates, where ornithine aminotransferase activity is suppressed for over 24 hours when administered at a dose of 50 mg/kg.[3] Gabaculine increases latency to convulsion in the 3-mercaptopropionic acid-induced and minimal electroshock-induced seizure models (ED_{50}s = 135 and 200 mg/kg, respectively) and inhibits 3-mercaptopropionic acid-induced increases in glutamic acid decarboxylase (GAD) activity and GABA-T activity in mice (ED_{50}s =135 mg/kg), however, the doses fall above the LD_{50} value of 62 mg/kg.[4] Gabaculine (135 mg/kg, i.p.) elevates concentrations of GABA in mouse brain by over 500% and knocks out GABA-T activity to below detection limits.

References

1. Rando, R.R. *Biochem J.* **16(21)**, 4604-4610 (1977).
2. Soper, T.S. and Manning, J.M. *J. Biol. Chem.* **257(23),** 13930-13936 (1982).
3. Jung, M.J. and Seiler, N. *J. Biol. Chem.* **253(20)**, 7431-7439 (1978).
4. Löscher, W. *Biochem. Pharmacol.* **28(8)**, 1397-1407 (1979).

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