Lazabemide hydrochloride

Cat. No.: HY-14202

CAS No.: 103878-83-7

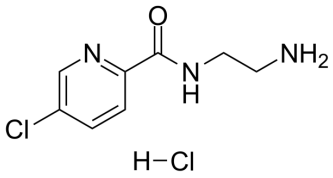
Molecular Formula: $C_8H_{11}Cl_2N_3O$

Molecular Weight: 236.1

Target: Monoamine Oxidase

Pathway: Neuronal Signaling

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Lazabemide hydrochloride (Ro 19-6327 hydrochloride) is a selective, reversible inhibitor of monoamine oxidase B (MAO-B) (IC_{50}=0.03 μM) but less active for MAO-A (IC_{50}>100 μM). Lazabemide inhibits monoamine uptake at high concentrations, the IC_{50} values are 86 μM, 123 μM and >500 μM for noradrenalin, serotonin and dopamine uptake, respectively. Lazabemide can be used for the research of parkinson and alzheimer's disease[1].
IC_{50} & Target	MAO-B 0.4 nM (IC_{50})
In Vitro	The in vitro binding characteristics of both radiolabeled inhibitors revealed them to be selective, high-affinity ligands for the respective enzymes. K_D and B_{max} values for ^{3}H-Ro 19-6327 in rat cerebral cortex are 18.4 nM and 3.45 pmol/mg protein, respectively[1]. The IC_{50} values for lazabemide are: 86 μM for NA uptake; 123 μM for 5HT uptake; > 500 μM for DA uptake, respectively[1]. . Lazabemide (5 μM) inhibits human MAO-B and MAO-A with IC_{50} of 6.9 nM and >10 nM, respectively. And it inhibits rat MAO-B and MAO-A with $IC_{50}$of 37 nM and >10 μM, respectively ina enzymatic assay[2]. Lazabemide differs from L-deprenyl in their ability to induce release of endogenous monoamines from synaptosomes. Thus, Lazabemide (500 μM) induces a greater 5 HT release than does L-deprenyl, but is less effective than L-deprenyl in releasing DA. On the contrary, lazabemide was almost completely inactive on either 5 HT and DA release[2]. Lazabemide (250 nM) results in a clear inhibition of DOPAC formation, while does not increase the accumulation of newly-formed DA in those tubular epithelial cells loaded with 50 microM L-DOPA[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Lazabemide (3 mg/kg) attenuates ichemia reperfusion-induced hydroxyl radical generation and pretreatment with Lazabemide showed decreased DOPAC levels in comparison with those of their respective vehicle-treated control groups[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Saura J, et al. Quantitative enzyme radioautography with 3H-Ro 41-1049 and 3H-Ro 19-6327 in vitro: localization and abundance of MAO-A and MAO-B in rat CNS, peripheral organs, and human brain. J Neurosci. 1992 May;12(5):1977-99.

[2]. Bondiolotti GP, et al. In vitro effects on monoamine uptake and release by the reversible monoamine oxidase-B inhibitors lazabemide and N-(2-aminoethyl)-p-chlorobenzamide: a comparison with L-deprenyl. Biochem Pharmacol. 1995 Jun 29;50(1):97-102.

[3]. Guimaraes J, et al. The activity of MAO A and B in rat renal cells and tubules. Life Sci. 1998;62(8):727-37.

[4]. Suzuki T, et al. MAO inhibitors, clorgyline and lazabemide, prevent hydroxyl radical generation caused by brain ischemia/reperfusion in mice. Pharmacology. 1995 Jun;50(6):357-62.

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