Lazabemide

Cat. No.: HY-14201

CAS No.: 103878-84-8

Molecular Formula: $C_8H_{10}ClN_3O$

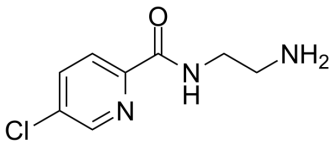
Molecular Weight: 199.64

Target: Monoamine Oxidase

Pathway: Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5 mg/mL (25.05 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	5.0090 mL	25.0451 mL	50.0902 mL
	5 mM	1.0018 mL	5.0090 mL	10.0180 mL
	10 mM	0.5009 mL	2.5045 mL	5.0090 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 0.5 mg/mL (2.50 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 0.5 mg/mL (2.50 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.5 mg/mL (2.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Lazabemide (Ro 19-6327) 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

IC50: 30 nM (MAO-B)[1].

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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