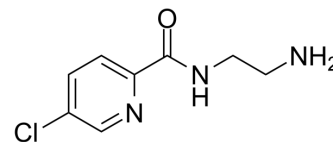


## Lazabemide

Cat. No.:	HY-14201	
CAS No.:	103878-84-8	
Molecular Formula:	C <sub>8</sub> H <sub>10</sub> ClN <sub>3</sub> O	
Molecular Weight:	199.64	
Target:	Monoamine Oxidase	
Pathway:	Neuronal Signaling	
Storage:	Powder	-20°C 3 years
		4°C 2 years
	In solvent	-80°C 6 months
		-20°C 1 month



### SOLVENT & SOLUBILITY

In Vitro	DMSO : 5 mg/mL (25.05 mM; Need ultrasonic)			
		Solvent Concentration	Mass	
			1 mg	5 mg
			10 mg	
Preparing Stock Solutions	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 <sub>50</sub> =0.03 μM) but less active for MAO-A (IC <sub>50</sub> >100 μM). Lazabemide inhibits monoamine uptake at high concentrations, the IC <sub>50</sub> 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 <sup>[1]</sup> .
IC <sub>50</sub> & Target	IC50: 30 nM (MAO-B) <sup>[1]</sup> .

<p><b>In Vitro</b></p>	<p>The in vitro binding characteristics of both radiolabeled inhibitors revealed them to be selective, high-affinity ligands for the respective enzymes. <math>K_D</math> and <math>B_{max}</math> values for <math>^3H</math>-Ro 19-6327 in rat cerebral cortex are 18.4 nM and 3.45 pmol/mg protein, respectively<sup>[1]</sup>.</p> <p>The <math>IC_{50}</math> values for lazabemide are: 86 <math>\mu</math>M for NA uptake; 123 <math>\mu</math>M for 5HT uptake; &gt; 500 <math>\mu</math>M for DA uptake, respectively<sup>[1]</sup>.</p> <p>. Lazabemide (5 <math>\mu</math>M) inhibits human MAO-B and MAO-A with <math>IC_{50}</math> of 6.9 nM and &gt;10 nM, respectively. And it inhibits rat MAO-B and MAO-A with <math>IC_{50}</math> of 37 nM and &gt;10 <math>\mu</math>M, respectively in an enzymatic assay<sup>[2]</sup>.</p> <p>Lazabemide differs from L-deprenyl in their ability to induce release of endogenous monoamines from synaptosomes. Thus, Lazabemide (500 <math>\mu</math>M) 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<sup>[2]</sup>.</p> <p>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<sup>[3]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
<p><b>In Vivo</b></p>	<p>Lazabemide (3 mg/kg) attenuates ischemia reperfusion-induced hydroxyl radical generation and pretreatment with Lazabemide showed decreased DOPAC levels in comparison with those of their respective vehicle-treated control groups<sup>[4]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

## 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.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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