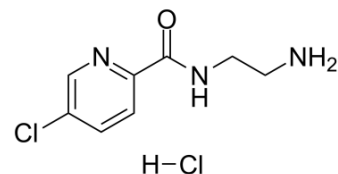


## Lazabemide hydrochloride

<b>Cat. No.:</b>	HY-14202
<b>CAS No.:</b>	103878-83-7
<b>Molecular Formula:</b>	C <sub>8</sub> H <sub>11</sub> Cl <sub>2</sub> N <sub>3</sub> O
<b>Molecular Weight:</b>	236.1
<b>Target:</b>	Monoamine Oxidase
<b>Pathway:</b>	Neuronal Signaling
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	Lazabemide hydrochloride (Ro 19-6327 hydrochloride) 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> .
<b>IC<sub>50</sub> &amp; Target</b>	MAO-B 0.4 nM (IC <sub>50</sub> )
<b>In Vitro</b>	<p>The in vitro binding characteristics of both radiolabeled inhibitors revealed them to be selective, high-affinity ligands for the respective enzymes. K<sub>D</sub> and B<sub>max</sub> values for <sup>3</sup>H-Ro 19-6327 in rat cerebral cortex are 18.4 nM and 3.45 pmol/mg protein, respectively<sup>[1]</sup>.</p> <p>The IC<sub>50</sub> values for lazabemide are: 86 μM for NA uptake; 123 μM for 5HT uptake; &gt; 500 μM for DA uptake, respectively<sup>[1]</sup>.</p> <p>Lazabemide (5 μM) inhibits human MAO-B and MAO-A with IC<sub>50</sub> of 6.9 nM and &gt;10 nM, respectively. And it inhibits rat MAO-B and MAO-A with IC<sub>50</sub> of 37 nM and &gt;10 μM, respectively in a 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 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<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>
<b>In Vivo</b>	<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 <sup>3</sup>H-Ro 41-1049 and <sup>3</sup>H-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.

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

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