

产品说明书

基本信息

产品编号:	L10234				
产品名称:	Lazabemide				
CAS:	103878-84-8	储存条件	粉末	-20℃	四年
分子式:	C21H19ClFN04S		溶于液体	-80℃	两年
分子量	435.90			-20℃	一个月
化学名:	N-(2-Aminoethyl)-5-chloro-2-pyridinecarboxamide;N-(2-Aminoethyl)-5-chloro-2-pyridinecarboxamide				
Solubility (25° C):					
体外:	DMSO	5mg/mL (25.05mM; Need ultrasonic)			
	Ethanol				
	Water				
体内 (现配现用):	1. 请依序添加每种溶剂: 10% DMSO→40% PEG300→5% Tween-80→45% saline, Solubility: ≥ 0.5mg/mL (2.50mM); Clear solution				
	此方案可获得 ≥ 0.5mg/mL (2.50mM, 饱和度未知) 的澄清溶液。以 1mL 工作液为例, 取 100 μL 5.0mg/mL 的澄清 DMSO 储备液加到 400 μL PEG300 中, 混合均匀; 向上述体系中加入 50 μL Tween-80, 混合均匀; 然后继续加入 450 μL 生理盐水定容至 1mL。				
	2. 请依序添加每种溶剂: 10% DMSO→90% (20% SBE-β-CD in saline) Solubility: ≥ 0.5mg/mL (2.50mM); Clear solution				
	此方案可获得 ≥ 0.5 mg/mL (2.50mM, 饱和度未知) 的澄清溶液。以 1mL 工作液为例, 取 100 μL 5.0 mg/mL 的澄清 DMSO 储备液加到 900 μL 20% 的 SBE-β-CD 生理盐水水溶液中, 混合均匀。				
	3. 请依序添加每种溶剂: 10% DMSO→90% corn oil Solubility:: ≥ 0.5mg/mL (2.50mM); Clear solution				
	此方案可获得 ≥ 0.5mg/mL (2.50mM, 饱和度未知) 的澄清溶液, 此方案不适用于实验周期在半个月以上的实验。以 1mL 工作液为例, 取 100 μL 5.0mg/mL 的澄清 DMSO 储备液加到 900 μL 玉米油中, 混合均匀。				
<1mg/ml 表示微溶或不溶。					
普西唐提供的所有化合物浓度为内部测试所得, 实际溶液度可能与公布值有所偏差, 属于正常的批间细微差异现象。					
请根据产品在不同溶剂中的溶解度选择合适的溶剂配制储备液; 一旦配成溶液, 请分装保存, 避免反复冻融造成的产品失效。					

制备储备液

浓度	质量			
	溶液体积	1mg	5mg	10mg
1mM		5.0090mL	25.0451mL	50.0902mL
5mM		1.0018mL	5.0090mL	10.0180mL
10mM		0.5009mL	2.5045mL	5.0090mL

生物活性

产品描述	一种单胺氧化酶 B (MAO-B) 的选择性的可逆抑制剂, IC ₅₀ =0.03 μM 。
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靶点	IC50: 30nM (MAO-B)
体外研究	The in vitro binding characteristics of both radiolabeled inhibitors revealed them to be selective, high-affinity ligands for the respective enzymes. KD and Bmax values for 3H-Ro 19-6327 in rat cerebral cortex are 18.4 nM and 3.45 pmol/mg protein, respectively. The IC50 values for lazabemide are: 86 μM for NA uptake; 123 μM for 5HT uptake; > 500 μM for DA uptake, respectively. Lazabemide (5 μM) inhibits human MAO-B and MAO-A with IC50 of 6.9nM and >10nM, respectively. And it inhibits rat MAO-B and MAO-A with IC50 of 37nM and >10 μM, respectively in an enzymatic assay. 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. Lazabemide (250nM) results in a clear inhibition of DOPAC formation, while does not increase the accumulation of newlyformed DA in those tubular epithelial cells loaded with 50microM L-DOPA.
体内研究	Lazabemide (3mg/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.