

产品名称: **2-Amino-6-chloro- $\alpha$ -cyano-3-(ethoxycarbonyl)-4H-1-benzopyran-4-acetic Acid Ethyl Ester**  
 产品别名: **SC79**

生物活性:						
Description		SC79 is a selective and cell-permeable Akt activator which activates Akt phosphorylation and inhibits Akt membrane translocation.				
IC <sub>50</sub> & Target		Akt [1]				
In Vitro		SC79 reduces neuronal excitotoxicity and prevents stroke-induced neuronal death. SC79 suppresses PHAKTM-GFP plasma membrane translocation, and enhances phosphorylation of all three Akt isoforms in HEK293, HeLa, HL60, NB4, and HsSulton (B cells) cells[1]. SC79 restores proliferation of BRAT1 knockdown cells, and reduces the production of superoxide in mitochondria of MitoSox positive cells[2].				
In Vivo		SC79 (0.04 mg/g, i.p.) inhibits the cytosolic activation of Akt, and recapitulates the primary cellular function of Akt signaling, resulting in augmented neuronal survival, in the permanent focal cerebral ischemia mouse model[1].				
Solvent&Solubility		<b>In Vitro:</b> <b>DMSO : ≥ 100 mg/mL (274.14 mM)</b> <b>H2O : &lt; 0.1 mg/mL (insoluble)</b>  * "≥" means soluble, but saturation unknown.				
		<div>Preparing Stock Solutions</div>	<div>Solvent / Mass / Concentration</div>	1 mg	5 mg	10 mg
			1 mM	2.7414 mL	13.7069 mL	27.4138 mL
			5 mM	0.5483 mL	2.7414 mL	5.4828 mL
			10 mM	0.2741 mL	1.3707 mL	2.7414 mL
		*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。  储备液的保存方式和期限 -80°C, 6 months; -20°C, 1 month。 -80°C 储存时, 请在 6 个月内使用, -20°C 储存时, 请在 1 个月内使用。  <b>In Vivo:</b>  请根据您的实验动物和给药方式选择适当的溶解方案。以下溶解方案都请先按照 <b>In Vitro</b> 方式配制澄清的储备液, 再依次添加助溶剂:  ——为保证实验结果的可靠性, 澄清的储备液可以根据储存条件, 适当保存; 体内实验的工作液, 建议您现用现配, 当天使用; 以下溶剂前显示的百分比是指该溶剂在您配制终溶液中的体积占比; 如在配制过程中出现沉淀、析出现象, 可以通过加热和/或超声的方式助溶				
		1.请依序添加每种溶剂: 10% DMSO→40% PEG300 →5% Tween-80 → 45% saline <b>Solubility: ≥ 2.5 mg/mL (6.85 mM); Clear solution</b>  此方案可获得 ≥ 2.5 mg/mL (6.85 mM, 饱和度未知) 的澄清溶液。  以 1 mL 工作液为例, 取 100 μL 25.0 mg/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 1 mL。				
		2.请依序添加每种溶剂: 10% DMSO →90% corn oil <b>Solubility: ≥ 2.5 mg/mL (6.85 mM); Clear solution</b>  此方案可获得 ≥ 2.5 mg/mL (6.85 mM, 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 1 mL 工作液为例，取 100 $\mu$ L 25.0 mg/mL 的澄清 DMSO 储备液加到 900 $\mu$ L 玉米油中，混合均匀。
<b>References</b>	<p>[1]. Jo H, et al. <u>Small molecule-induced cytosolic activation of protein kinase Akt rescues ischemia-elicited neuronal death</u>. Proc Natl Acad Sci U S A. 2012 Jun 26;109(26):10581-10586.</p> <p>[2]. So EY, et al. <u>BRAT1 deficiency causes increased glucose metabolism and mitochondrial malfunction</u>. BMC Cancer. 2014 Jul 29;14:548</p>
<b>实验参考：</b>	
<b>Cell Assay</b>	HsSultan or NB4 cells ( $2.5 \times 10^5$ ) are plated in a 24-well plate in 500 $\mu$ L of phenol red-free RPMI medium supplemented with 10% FBS. After incubation for 24 hours, each compound (8 $\mu$ g/mL) is added and cultured for overnight (16-20 h). Fifty $\mu$ Ls of MTT solution (5 mg/mL in PBS) are added to each well. Following 2 hrs incubation, the purple formazan crystals are dissolved by directly adding in 500 $\mu$ L of isopropanol with 0.1mol/LHCl to each well. After clearing the cell debris by centrifugation, the absorbance is measured at a wavelength of 570 nm. [1]
<b>Animal Administration</b>	The permanent focal cerebral ischemia is induced by middle cerebral artery occlusion (MCAO) essentially. Briefly, mice (C57 Black/6) weighing 17-25 g are anesthetized with 4% isoflurane/66% N <sub>2</sub> O/30% O <sub>2</sub> and maintained with 1.5% isoflurane. Permanent focal ischemia is achieved as follows: a 2-mm hole is drilled at a site superior and lateral to the left foramen ovale to expose the left middle cerebral artery. The proximal portion of the left middle cerebral artery (MCA) is permanently occluded over a 1-mm segment distal to the origin of the lenticulostriate branches through the use of a bipolar coagulator. SC79 is injected intraperitoneally (0.04 mg/g mouse body weight) 5 min before permanent MCAO. In another experiment, extra SC79 is injected (0.04 mg/g mouse body weight, once per hour for 6 hours). [1]
<b>References</b>	<p>[1]. Jo H, et al. <u>Small molecule-induced cytosolic activation of protein kinase Akt rescues ischemia-elicited neuronal death</u>. Proc Natl Acad Sci U S A. 2012 Jun 26;109(26):10581-10586.</p> <p>[2]. So EY, et al. <u>BRAT1 deficiency causes increased glucose metabolism and mitochondrial malfunction</u>. BMC Cancer. 2014 Jul 29;14:548</p>

源叶生物