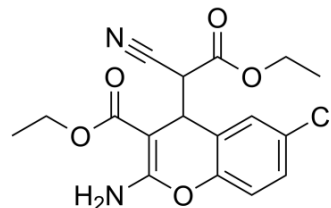


SC79

Cat. No.:	HY-18749
CAS No.:	305834-79-1
Molecular Formula:	C ₁₇ H ₁₇ ClN ₂ O ₅
Molecular Weight:	364.78
Target:	Akt
Pathway:	PI3K/Akt/mTOR
Storage:	Powder -20°C 3 years 4°C 2 years



* The compound is unstable in solutions, freshly prepared is recommended.

SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (274.14 mM)
* "≥" means soluble, but saturation unknown.

Concentration	Solvent	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	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

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: 10 mg/mL (27.41 mM); Suspended solution; Need ultrasonic
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (6.85 mM); Clear solution
- Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
Solubility: 5 mg/mL (13.71 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

SC79, a unique specific and BBB permeable Akt activator, activates Akt in the cytosol and inhibits Akt membrane translocation. SC79 specifically binds to the PH domain of Akt^{[1][2][3]}.

IC₅₀ & Target

Akt

In Vitro

SC79 augments Akt phosphorylation at both the Thr308 and S473 sites^[1]. SC79 (10.96 μM) induces cytosolic phosphorylation of Akt. SC79 enhances IGF1-induced Akt phosphorylation in both serum-

starved cells and cells grown in serum-rich medium^[1].

SC79 reduces neuronal excitotoxicity and prevents stroke-induced neuronal death. SC79 suppresses PHAKTM-GFP plasma membrane translocation^[1].

SC79 restores proliferation of BRAT1 knockdown cells, and reduces the production of superoxide in mitochondria of MitoSox positive cells^[2].

SC79 upregulates FLIPL/S expression and consequently suppresses caspase-8 activation^[5].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis^[1]

Cell Line:	HeLa cells.
Concentration:	4 µg/mL (10.96 µM).
Incubation Time:	30 min.
Result:	Induced cytosolic phosphorylation of Akt.

In Vivo

SC79 treatment, even at much high dose (0.4 mg/g of body weight), does not induce any detectable changes in body weight, survival rate, appearance, and behavior in mice^[1].

SC79 (10 mg/kg, i.p.) Protects C57BL/6 mice from fas-induced fulminant hepatic failure^[4].

SC79 protects hepatocytes from TNFα-mediated apoptosis and mice from Gal/LPS-induced liver injury and damage^[5].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male, age-matched (6- to 8-week-old) C57BL/6 or BALB/c mice weighing 16 to 18 g ^[4] .
Dosage:	10 mg/kg.
Administration:	Intraperitoneally at 0.5 hour before the i.p. administration of an agonistic anti-Fas Jo2 antibody at a lethal dose of 0.5 and 0.4 mg/kg for C57BL/6 and BALB/c mice, respectively.
Result:	Treatment of mice with 10 mg/kg of SC79 at 0.5 hour before Jo2 injection increased mouse survival at 12 hours after Jo2 injection from 0% to 35%, and no additional mortality was observed to the end of the 2-month observation period.

Animal Model:	Male, age-matched (6 to 8 weeks old) C57BL/6 mice weighing 16-18 g ^[5] .
Dosage:	10 mg/kg.
Administration:	Intraperitoneally at 0.5 h before i.p. administration of 400 mg/kg of D-galactosamine (D-Gal) and 60 µg/kg of lipopolysaccharide (LPS) for C57BL/6 mice.
Result:	Gal/LPS challenge there was more bleeding on the liver of the vehicle control-treated mice as compared to that of SC79-treated mice. A single dose of SC79 significantly reduced Gal/LPS-mediated liver damage but not an infiltration of inflammatory cells in liver sections.

CUSTOMER VALIDATION

- Acta Biomater. 2018 Nov;81:278-292.
- Acta Pharm Sin B. 2021 Apr 2.
- Acta Pharm Sin B. 2021 Jan;11(1):71-88.

- J Exp Clin Cancer Res. 2019 May 15;38(1):197.
- Mol Ther Nucleic Acids. 2019 Jun 7;16:105-117.

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REFERENCES

- [1]. Jo H, et al. Small molecule-induced cytosolic activation of protein kinase Akt rescues ischemia-elicited neuronal death. Proc Natl Acad Sci U S A. 2012 Jun 26;109(26):10581-10586.
- [2]. So EY, et al. BRAT1 deficiency causes increased glucose metabolism and mitochondrial malfunction. BMC Cancer. 2014 Jul 29;14:548
- [3]. Liu X, et al. Activation of Akt by SC79 decreased cerebral infarct in early cerebral ischemia-reperfusion despite increased BBB disruption. Neurosci Lett. 2018 Aug 10;681:78-82.
- [4]. Liu W, et al. A Novel AKT Activator, SC79, Prevents Acute Hepatic Failure Induced by Fas-Mediated Apoptosis of Hepatocytes. Am J Pathol. 2018 May;188(5):1171-1182.
- [5]. Jing ZT, et al. AKT activator SC79 protects hepatocytes from TNF- α -mediated apoptosis and alleviates d-Gal/LPS-induced liver injury. Am J Physiol Gastrointest Liver Physiol. 2019 Mar 1;316(3):G387-G396.
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Caution: Product has not been fully validated for medical applications. For research use only.

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