SC79

Cat. No.: HY-18749

CAS No.: 305834-79-1

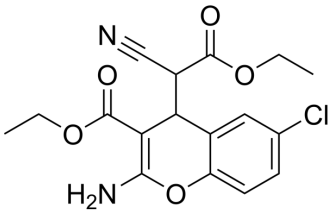
Molecular Formula: $C_{17}H_{17}ClN_2O_5$

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; Need ultrasonic)
Ethanol : 50 mg/mL (137.07 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	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

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

In Vivo

1. Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 5 mg/mL (13.71 mM); Clear solution
2. Add each solvent one by one: 10% EtOH >> 90% corn oil
   Solubility: ≥ 5 mg/mL (13.71 mM); Clear solution
3. 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
4. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (6.85 mM); Clear solution
5. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.85 mM); Clear solution
6. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.85 mM); Clear solution

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].

In Vitro

SC79 augmentes 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-weekeold) 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

- Nat Commun. 2023 Sep 28;14(1):6069.
- Nat Commun. 2023 Sep 25;14(1):5977.
- Acta Pharm Sin B. 2021 Jun;11(6):1592-1606.
- Acta Pharm Sin B. 2021 Jan;11(1):71-88.
- Biomaterials. 2021 Jul;274:120850.

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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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