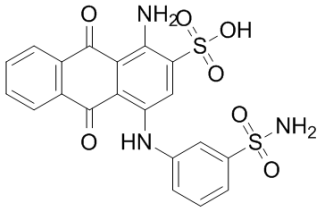
NSC117079

Cat. No.:	HY-19819
CAS No.:	500363-63-3
Molecular Formula:	$C_{20}H_{15}N_3O_7S_2$
Molecular Weight:	473.48
Target:	Others
Pathway:	Others

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (105.60 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1120 mL	10.5601 mL	21.1202 mL
	5 mM	0.4224 mL	2.1120 mL	4.2240 mL
	10 mM	0.2112 mL	1.0560 mL	2.1120 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (5.28 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.28 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

NSC117079 is a novel PHLPP inhibitor.

IC_{50} & Target

PHLPP[1]

In Vitro

NSC-117079 at 30 μM induces neutrophil adhesion to plated fibrinogen from 9.0±2.4% to 27.0±8.0% and enhanced neutrophil adhesion caused by 50 ng/mL GM-CSF from 22.9±6.0% to 47.6±10.9%. Neutrophil adhesion is followed by neutrophil transendothelial migration. Results suggest that PHLPP inhibitor NSC-117079 is effective in preventing Akt from dephosphorylation in neutrophils, and Akt phosphatase PHLPP serves to attenuate neutrophil adhesion but not migration[2].

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

In Vivo	A single intraarticular injection of the Phlpp inhibitor NSC117079 attenuates mechanical allodynia and slows articular cartilage degradation in joints with a destabilized meniscus. Animals treated with the Phlpp inhibitor seven weeks after injury maintain normal activity levels, while those in the control group travel shorter distances and are less active three months after the joint injury. NSC117079 also increases production of cartilage extracellular matrix components (glycosaminoglycans and aggrecan) in over 90% of human articular cartilage explants from osteoarthritis patients and increased phosphorylation of Phlpp1 substrates (AKT2, ERK1/2 and PKC) in human articular chondrocytes[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1]	Mice[1] Posttraumatic osteoarthritis is induced in male C57Bl/6 mice by surgically destabilizing the meniscus. Seven weeks after surgery, mice receive a single intra-articular injection of the PHLPP inhibitor NSC117079 (8 μM) or saline. Mechanical allodynia is measured with von Frey assays, mobility is tracked in an open field system, and cartilage damage is assessed histologically[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Jackson TC, et al. Pharmacological inhibition of pleckstrin homology domain leucine-rich repeat protein phosphatase is neuroprotective: differential effects on astrocytes. J Pharmacol Exp Ther. 2013 Nov;347(2):516-28.

[2]. Zhu X, et al. Regulation Of Neutrophil Adhesion And Migration By Ph Domain And Leucine Rich Repeat Protein Phosphatase. A35 RECENT ADVANCES IN PHAGOCYTE BIOLOGY / Thematic Poster Session / Sunday, May 20/8:15 AM-4:30 PM / Area G (Hall D, North Building, Lower Level), Moscone Center

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA