PD146176

Cat. No.: HY-103157

CAS No.: 4079-26-9

Molecular Formula: $C_{15}H_{11}NS$

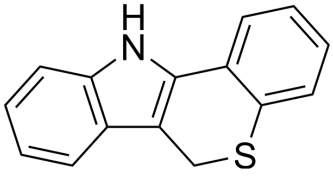
Molecular Weight: 237

Target: Autophagy; Ferroptosis

Pathway: Autophagy; Apoptosis

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (105.49 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.2194 mL	21.0970 mL	42.1941 mL
	5 mM	0.8439 mL	4.2194 mL	8.4388 mL
	10 mM	0.4219 mL	2.1097 mL	4.2194 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 (10.55 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (10.55 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.55 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

PD146176 (NSC168807), a 15-Lipoxygenase (15-LO) inhibitor, inhibits rabbit reticulocyte 15-LO (K_i=197 nM, IC_{50}=0.54 μM). PD146176 reverses cognitive impairment, brain amyloidosis, and tau pathology by stimulating autophagy in aged triple transgenic mice[1][2][3].

IC_{50} & Target

Ki: 197 nM (Rabbit reticulocyte 15-LO)[1]
IC50: 0.54 μM[2]

In Vitro

In intact IC21 cells transfected with human 15-LO, PD146176 inhibits 13-HODE production with an IC_{50} of 0.81 μM[2].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	PD146176 (80 mg/kg; Chowing; 12 weeks) reverses cognitive impairment, brain amyloidosis, and Tau pathology by stimulating autophagy in aged triple transgenic mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Triple transgenic (3xTg) mice[3]
Dosage:	80 mg/kg
Administration:	Chowing; 12 weeks
Result:	Significantly lower amyloid beta levels and deposition, less tau neuropathology, increased synaptic integrity, and autophagy activation.

CUSTOMER VALIDATION

- Biosci Trends. 2022 Aug 7.

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REFERENCES

[1]. Sendobry SM, et al. Attenuation of diet-induced atherosclerosis in rabbits with a highly selective 15-lipoxygenase inhibitor lacking significant antioxidant properties. Br J Pharmacol. 1997;120(7):1199-1206.

[2]. Bocan TM, et al. A specific 15-lipoxygenase inhibitor limits the progression and monocyte-macrophage enrichment of hypercholesterolemia-induced atherosclerosis in the rabbit [published correction appears in Atherosclerosis 1998 Jul;139(1):201]. Atherosclerosis. 1998;136(2):203-216.

[3]. Di Meco A, et al. 12/15-Lipoxygenase Inhibition Reverses Cognitive Impairment, Brain Amyloidosis, and Tau Pathology by Stimulating Autophagy in Aged Triple Transgenic Mice. Biol Psychiatry. 2017;81(2):92-100.

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