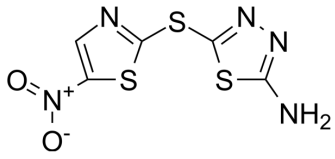
Halicin

Cat. No.:	HY-107597
CAS No.:	40045-50-9
Molecular Formula:	$C_5H_3N_5O_2S_3$
Molecular Weight:	261.3
Target:	JNK
Pathway:	MAPK/ERK Pathway
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 62.5 mg/mL (239.19 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.8270 mL	19.1351 mL	38.2702 mL
	5 mM	0.7654 mL	3.8270 mL	7.6540 mL
	10 mM	0.3827 mL	1.9135 mL	3.8270 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: ≥ 2.08 mg/mL (7.96 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (7.96 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Halicin (SU3327) is a potent, selective and substrate-competitive JNK inhibitor with an IC_{50} of 0.7 μM. Halicin also inhibits protein-protein interactions between JNK and JNK Interacting Protein (JIP) with an IC_{50} of 239 nM. Halicin shows less active against p38α and Akt kinase[1][2].

IC_{50} & Target

IC50: 0.7 μM (JNK); 239 nM (JNK-JIP interactions)[1]

In Vitro

Halicin (compound 9) is able to inhibit TNF-α stimulated phosphorylation of c-Jun in HeLa cells (EC_{50} = 6.23 μM)[1].
Halicin (25 nM) pretreatment of? human-derived cerebral microvascular endothelial cells (hCMEC/D3) effectively reduces LPS-induced polymorphonuclear leukocytes (PMN) rolling/adhesion to hCMEC/D3, prevents activation of AP-1, and significantly reduces expression of VCAM-1[3].

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

In Vivo

Halicin (Compound 9; 25 mg/kg; intraperitoneal injection; male BKS.Cg-+Lepr^{db}/+Lepr^{db}/OlaHsd db/db mice) treatment possesses the ability to restore insulin sensitivity in mice models of diabetes[1].
Halicin (Compound 9) has favorable microsomal and plasma stability ($T_{1/2}$ = 27 min)[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male BKS.Cg-+Lepr^{db}/+Lepr^{db}/OlaHsd db/db mice (11-week-old) injected with insulin[1]
Dosage:	25 mg/kg
Administration:	Intraperitoneal injection
Result:	Resulted in a statistically significant reduction in blood glucose levels.

CUSTOMER VALIDATION

- Biol Pharm Bull. 2022 May 31.

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REFERENCES

[1]. De SK, et al. Design, synthesis, and structure-activity relationship of substrate competitive, selective, and in vivo active triazole and thiadiazole inhibitors of the c-Jun N-terminal kinase. J Med Chem. 2009 Apr 9;52(7):1943-52.

[2]. Augustine C, et al. Traumatic injury elicits JNK-mediated human astrocyte retraction in vitro. Neuroscience. 2014 Aug 22;274:1-10.

[3]. Serizawa F, et al. Pretreatment of human cerebrovascular endothelial cells with CO-releasing molecule-3 interferes with JNK/AP-1 signaling and suppresses LPS-induced proadhesive phenotype. Microcirculation. 2015 Jan;22(1):28-36.

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