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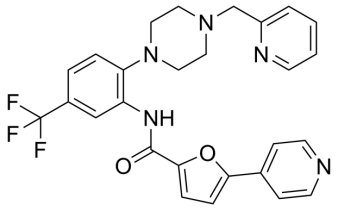
SPHINX31

Cat. No.:	HY-117661
CAS No.:	1818389-84-2
Molecular Formula:	$C_{27}H_{24}F_3N_5O_2$
Molecular Weight:	507.51
Target:	SRPK; VEGFR
Pathway:	Cell Cycle/DNA Damage; Protein Tyrosine Kinase/RTK
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 : 17.33 mg/mL (34.15 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9704 mL	9.8520 mL	19.7040 mL
	5 mM	0.3941 mL	1.9704 mL	3.9408 mL
	10 mM	0.1970 mL	0.9852 mL	1.9704 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 mg/mL (3.94 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2 mg/mL (3.94 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

SPHINX31 is a potent and selective SRPK1 inhibitor, with an IC_{50} of 5.9 nM. SPHINX31 inhibits phosphorylation of serine/arginine-rich splicing factor 1 (SRSF1). SPHINX31 also decreases the mRNA expression of pro-angiogenic VEGF-A_{165}a isoform. SPHINX31 can be used to research neovascular eye disease[1][2][3].

IC_{50} & Target

IC_{50}: 5.9 nM (SRPK1)[1]
VEGF-A_{165}a[2]

In Vitro

SPHINX31 (0.3-10 μM; 24 h) significantly down-regulates the expression of VEGF-A_{165}a mRNA[2].
SPHINX31 (0.3 μM; 24 h) suppresses SRSF1 phosphorylation and nuclear localization[2].
SPHINX31 (0.3-10 μM; 24 h) decreases pre-tube formation and the rate of migration for human umbilical vein endothelial

cells (HUVECs)[2].

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

RT-PCR[2]

Cell Line:	HuCCA-1 cells
Concentration:	0.3, 1, 3 and 10 μM
Incubation Time:	24 h
Result:	Significantly down-regulated about 60% of the expression of VEGF-A_{165}a mRNA compared with the control cells.

Immunofluorescence[2]

Cell Line:	HuCCA-1 cells
Concentration:	0.3 μM
Incubation Time:	24 h
Result:	Suppressed SRSF1 phosphorylation and nuclear localization, which thereby induced less expression of pro-angiogenic VEGF-A_{165}a in HuCCA-1 cells.

Cell Migration Assay [2]

Cell Line:	HuCCA-1 cells
Concentration:	0.3, 1, 3 and 10 μM
Incubation Time:	24 h
Result:	Decreased pre-tube formation of the cells network area to about 50% of the control group. Decreased the rate of migration for HUVECs.

In Vivo

SPHINX31 (200 μg/mL; twice daily topical eye drops) protects the retinal endothelial permeability barrier from diabetes-associated loss of integrity[3].

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

Animal Model:	Norway Brown rats (intraperitoneally injected with 50 mg/kg Streptozotocin (HY-10219) to induce type I diabetes)[3]
Dosage:	200 μg/mL
Administration:	Twice daily topical eye drops
Result:	Reduced retinal permeability in the diabetics ($7.92 \pm 1.65 \times 10^{-4}$ cms^{-1}) less than before induction of diabetes ($8.15 \pm 2.33 \times 10^{-4}$ cms^{-1}), and less than the control group ($8.85 \pm 1.29 \times 10^{-4}$ cms^{-1}), while the diabetes group was $12.67 \pm 1.09 \times 10^{-4}$ cms^{-1}.

CUSTOMER VALIDATION

- Sci Signal. 2022 Oct 25;15(757):eabm0808.
- Front Pharmacol. 2022 Jun 30;13:920601.

- BMC Cancer. 2022 Oct 27;22(1):1100.
- SSRN. 2023 Jun 20.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. C. Allen, et al. The SRPK1 inhibitor SPHINX31 prevents increased retinal permeability in a rodent model of diabetes. Acta Ophthalmologica. Volume 95, Issue S259.

[2]. Supradit K, et al. Inhibition of serine/arginine-rich protein kinase-1 (SRPK1) prevents cholangiocarcinoma cells induced angiogenesis. Toxicol In Vitro. 2022 Aug;82:105385.

[3]. Batson J, et al. Development of Potent, Selective SRPK1 Inhibitors as Potential Topical Therapeutics for Neovascular Eye Disease. ACS Chem Biol. 2017 Mar 17;12(3):825-832.

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