SKI V

Cat. No.: HY-12895

CAS No.: 24418-86-8

Molecular Formula: $C_{15}H_{10}O_4$

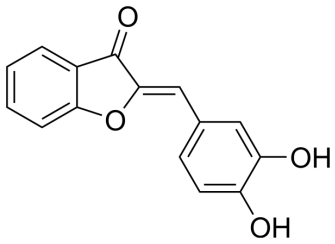
Molecular Weight: 254.24

Target: SphK; PI3K; Apoptosis

Pathway: Immunology/Inflammation; PI3K/Akt/mTOR; Apoptosis

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 (196.66 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.9333 mL	19.6665 mL	39.3329 mL
	5 mM	0.7867 mL	3.9333 mL	7.8666 mL
	10 mM	0.3933 mL	1.9666 mL	3.9333 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 (9.83 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (9.83 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

SKI V is a noncompetitive and potent non-lipid sphingosine kinase (SPHK; SK) inhibitor with an IC_{50} of 2 μM for GST-hSK. SKI V potently inhibits PI3K with an IC_{50} of 6 μM for hPI3k. SKI V decreases formation of the mitogenic second messenger sphingosine-1-phosphate (S1P). SKI V induces apoptosis and has antitumor activity[1][2].

IC_{50} & Target

IC50: 2 μM (GST-hSK), 6 μM (hPI3k) and 80 μM (ERK2)[1]

In Vitro

SKI V has weak activity toward ERK2 (IC_{50} of 80 μM for hERK2) and does not inhibit PKC-α[1].
SKI V (10 μM; for 24 hours) inhibits cancer cell proliferation and induces apoptosis[1].
SKI V (0.2, 1, 5 μM; pretreated for 1 hour) decreases phospho-Akt and phospho-MEK levels. Near-confluent cultures of JC cells are serum-starved for 16 hours, followed by pretreatment SKI V for 1 hour[2].

SKI V has IC_{50}s for inhibition of sphingosine kinase (SK) and tumor cell proliferation of ~2 μM[1].
SKI V (20 μg/ml) inhibits not only purified but endogenous SK in in MDA-MB-231 cells[1].
SKI V (0.2, 1, 5 μM) inhibits intracellular S1P formation in JC cells in a dose-dependent fashion[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	T24 tumor cells
Concentration:	10 μM
Incubation Time:	For 24 hours
Result:	Inhibited cancer cell proliferation.

Apoptosis Analysis[1]

Cell Line:	T24 tumor cells
Concentration:	10 μM
Incubation Time:	For 24 hours
Result:	Induced apoptosis.

Western Blot Analysis[2]

Cell Line:	JC cells
Concentration:	0.2, 1, 5 μM
Incubation Time:	Pretreated for 1 hour
Result:	Decreased phospho-Akt and phospho-MEK levels.

In Vivo

SKI V (75 mg/kg; i.p.; days 1, 5, 9, 15) significantly lowers tumor growth (>50% decreased at day 18) than control animals[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	6-8 weeks old BALB/c female mice with JC mammary adenocarcinoma cells[1]
Dosage:	75 mg/kg
Administration:	IP; days 1, 5, 9, 15
Result:	Tumor growth was significantly lower (>50% decreased at day 18) than tumor growth in control animals.

CUSTOMER VALIDATION

- Int J Biol Sci. 2022 Apr 18;18(7):2994-3005.

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REFERENCES

[1]. French KJ, et al. Discovery and evaluation of inhibitors of human sphingosine kinase. Cancer Res. 2003 Sep 15;63(18):5962-9.

[2]. French KJ, et al. Antitumor activity of sphingosine kinase inhibitors. J Pharmacol Exp Ther. 2006 Aug;318(2):596-603.

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