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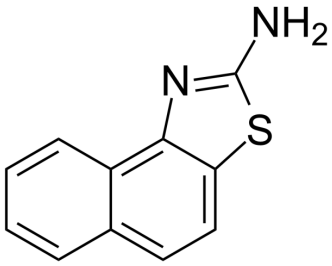
SKA-31

Cat. No.:	HY-111655
CAS No.:	40172-65-4
Molecular Formula:	$C_{11}H_8N_2S$
Molecular Weight:	200.26
Target:	Potassium Channel
Pathway:	Membrane Transporter/Ion Channel
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (624.19 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.9935 mL	24.9675 mL	49.9351 mL
	5 mM	0.9987 mL	4.9935 mL	9.9870 mL
	10 mM	0.4994 mL	2.4968 mL	4.9935 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 (12.48 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (12.48 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (10.39 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

SKA-31 is a potent potassium channel activator with EC_{50s} of 260 nM, 1.9 μM, 2.9 μM, and 2.9 μM for KCa3.1, KCa2.2, KCa2.1 and KCa2.3, respectively. SKA-31 potentiates endothelium-derived hyperpolarizing factor response and lowers blood pressure[1].

IC_{50} & Target

EC50: 2.9 μM (KCa2.1), 1.9 μM (KCa2.2), 2.9 μM (KCa2.3), 260 nM (KCa3.1)[1]

In Vitro

SKA-31 activates KCa2/3 channels more potently than PK 26124, and is more selective over other Ion channels[1].
SKA-31 reduces cell viability with IC_{50s} of 5.3 μM , 46.9 μM in HCT-116 cells and HCT-8 cells, respectively[2].

SKA-31 (5.3 μM; 0-96 hours) reduces HCT-116 cells proliferation when added at time zero at 5.3 μM[2].
SKA-31 triggers apoptosis in HCT-116 cells at 5 μM, and the effect is smaller in HCT-8 cells at 45 μM[2].
SKA-31 increases the percentage of cells in G0/G1 phase in HCT-116 and HCT-8 cell lines at 5 μM and 45 μM, respectively[2].
SKA-31 further activates Caspase 3 and reduces Akt phosphorylation induced by CDDP[2].
SKA-31 has a synergic effect with CDDP also on the inhibition of HCT-116 cell proliferation[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	HCT-116 cells, HCT-8 cells
Concentration:	
Incubation Time:	24 hours
Result:	Reduced cell viability with IC_{50s} of 5.3 μM , 46.9 μM in HCT-116 and HCT-8, respectively.

Cell Proliferation Assay[2]

Cell Line:	HCT-116 cells
Concentration:	5.3 μM
Incubation Time:	0-96 hours
Result:	Reduced HCT-116 cells proliferation when added at time zero at IC_{50S} value.

Apoptosis Analysis[2]

Cell Line:	HCT-116 cells, HCT-8 cells
Concentration:	5 μM (HCT-116 cells), 45 μM (HCT-8 cells)
Incubation Time:	24 hours
Result:	Triggered apoptosis in HCT-116 cells, and the effect was smaller in HCT-8 cells.

Cell Cycle Analysis[2]

Cell Line:	HCT-116 cells, HCT-8 cells
Concentration:	5 μM (HCT-116), 45 μM (HCT-8)
Incubation Time:	24 hours
Result:	Increased the percentage of cells in G0/G1 phase in HCT-116 and HCT-8 cell lines.

Western Blot Analysis[2]

Cell Line:	HCT-116 cells
Concentration:	
Incubation Time:	24 hours
Result:	Further activated Caspase 3 and reduced Akt phosphorylation when co-treatment with CDDP in HCT-116 cells.

In Vivo

SKA-31 is not acutely toxic and has good pharmacokinetic properties[1].
SKA-31 potentiates native KCa3.1 and KCa2.3 in murine carotid endothelium with EC_{50} values of 225 nM and 1.6 μM for

KCa3.1 and KCa2.3, respectively[1].
SKA-31 stimulates KCa3.1 and KCa2.3 in vascular endothelial cells and increases acetylcholine-induced endothelium-derived hyperpolarizing factor (EDHF) -mediated vasodilation[1].
SKA-31 potentiates EDHF-type vasodilations and lowers blood pressure in mice. Injections of SKA-31 (1-30 mg/kg; i.p.) lower MAP over 24 hours in normotensive wild-type mice but not in KCa3.1(-/-) mice (-/-)[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	16-25 weeks mice[1]
Dosage:	1 mg/kg, 10 mg/kg, and 30 mg/kg
Administration:	Intraperitoneal injection
Result:	Lower MAP over 24 hours in normotensive wild-type mice but not in KCa3.1(-/-) mice (-/-).

CUSTOMER VALIDATION

- Front Physiol. 2021 Mar 9;12:639857.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Sankaranarayanan A, et al. Naphtho[1,2-d]thiazol-2-ylamine (SKA-31), a new activator of KCa2 and KCa3.1 potassium channels, potentiates the endothelium-derived hyperpolarizing factor response and lowers blood pressure. Mol Pharmacol. 2009 Feb;75(2):281-95

[2]. Serena Pillozzi, et al. The combined activation of KCa3.1 and inhibition of Kv11.1/hERG1 currents contribute to overcome CDDP resistance in colorectal cancer cells. Br J Cancer. 2018 Jan; 118(2): 200–212.

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