SKF-96365 hydrochloride

Cat. No.: HY-100001

CAS No.: 130495-35-1

Molecular Formula: $C_{22}H_{27}ClN_2O_3$

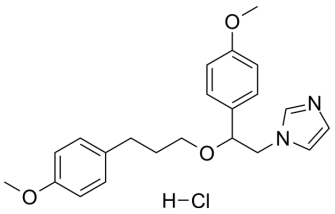
Molecular Weight: 402.91

Target: TRP Channel; Autophagy; Apoptosis; CRAC Channel; Potassium Channel

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling; Autophagy; Apoptosis

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (248.19 mM; Need ultrasonic)

H_2O : 100 mg/mL (248.19 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4819 mL	12.4097 mL	24.8194 mL
5 mM	0.4964 mL	2.4819 mL	4.9639 mL
10 mM	0.2482 mL	1.2410 mL	2.4819 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: Saline
   Solubility: 100 mg/mL (248.19 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (6.20 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.20 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.20 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

SKF-96365 hydrochloride is a potent TRP channel blocker and a store-operated Ca^{2+} entry (SOCE) inhibitor. SKF-96365 hydrochloride significantly inhibits hERG, hKCNQ1/hKCNE1, hKir2.1 and hKv4.3 current, and significantly prolongs the QTc interval in isolated guinea pig hearts. SKF-96365 hydrochloride exhibits potent anti-neoplastic activity by inducing cell-cycle arrest and apoptosis in colorectal cancer cells[1][2].

In Vitro

SKF-96365 exhibits potent anti-neoplastic activity by inducing cell-cycle arrest and apoptosis in colorectal cancer cells. SKF-

	96365 inhibits hERG current in a concentration-dependent manner[1]. SKF-96365 can induces cytoprotective autophagy to delay apoptosis by preventing the release of cytochrome c (cyt c) from the mitochondria into the cytoplasm. Mechanistically, SKF-96365 treatment inhibits the calcium/calmodulin-dependent protein kinase IIγ (CaMKIIγ)/AKT signaling cascade. Overexpression of CaMKIIγ or AKT abolishes the effects of SKF-96365 on cancer cells, suggesting a critical role of the CaMKIIγ/AKT signaling pathway in SFK-96365-induced biological effects[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	SKF-96365 inhibits CRC cell growth in vivo. SKF-96365 treatment results in a decrease of p-CaMKII and p-AKT as well as an increase in LC3-II, cleaved PARP, caspase-3, and caspase-9 in mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [2]	Mice: Five to six-week-old female athymic BALB/c mice are inoculated into the right oxter with HCT116 cells. When the diameter of the subcutaneous tumor reaches approximately 0.5 cm, animals are randomLy assigned to the vehicle, SKF-96365 alone, HCQ alone or SKF-96365+HCQ. SKF-96365 is applied (20 mg/kg) and HCQ is applied (60 mg/kg) daily for 14 successive days by i.p. injection. Tumor sizes and volume are determined. Eight mice are included in each group. Mice are sacrificed 24 h after the last treatment. The tumors are weighed and processed for western blot analysis or paraffin embedding[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Bioact Mater. 2021 Apr 21;6(11):4073-4082.
- Adv Sci (Weinh). 2022 Oct 19;e2202857.
- Autophagy. 2021 Nov;17(11):3592-3606.
- Theranostics. 2021 May 25;11(15):7379-7390.
- Theranostics. 2021 Mar 5;11(10):5045-5060.

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REFERENCES

[1]. Liu H, et al. SKF-96365 blocks human ether-à-go-go-related gene potassium channels stably expressed in HEK 293 cells. Pharmacological Research. Pharmacol Res, 2016 Feb, 104:61-9.

[2]. Jing Z, et al. SKF-96365 activates cytoprotective autophagy to delay apoptosis in colorectal cancer cells through inhibition of the calcium/CaMKIIγ/AKT-mediated pathway. Cancer Lett, 2016 Mar 28, 372(2):226-38.

Caution: Product has not been fully validated for medical applications. For research use only.

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