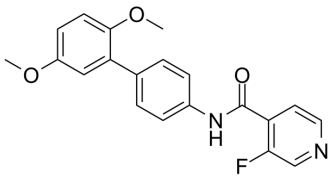
Synta66

Cat. No.:	HY-111325
CAS No.:	835904-51-3
Molecular Formula:	$C_{20}H_{17}FN_2O_3$
Molecular Weight:	352.36
Target:	CRAC Channel
Pathway:	Membrane Transporter/Ion Channel
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 : 77.5 mg/mL (219.95 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8380 mL	14.1900 mL	28.3801 mL
	5 mM	0.5676 mL	2.8380 mL	5.6760 mL
	10 mM	0.2838 mL	1.4190 mL	2.8380 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: 2.58 mg/mL (7.32 mM); Suspended solution; Need ultrasonic and warming
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.58 mg/mL (7.32 mM); Suspended solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.58 mg/mL (7.32 mM); Clear solution; Need warming

BIOLOGICAL ACTIVITY

Description

Synta66 is an inhibitor of store-operated calcium entry channel Orai, which forms the pore of the CRAC channel, and used for the research of neurological disease.

IC_{50} & Target

Orai[1]

In Vitro

Synta66 is an inhibitor of Orai, which forms the pore of the CRAC channel. Synta66 (10 μM) attenuates peak SOCE in Müller glia. Synta66 (10 μM) prevents orai channels mediating the residual SOC current in $Trpc1^{-/-}$ Müller cells[1]. Synta66 (10 μM)

nearly completely blocks the Ca^{2+} entry signal evoked by $CaCl_2$ addition, whereas it moderately reduces Ca^{2+} mobilization from stores with 10% to 30% in platelet. Synta66 (10 μM) suppresses human platelet activation in plasma and whole-blood thrombus formation. Synta66 (10 μM) also inhibits murine platelet responses and thrombus formation[2]. Synta66 (10 μM) inhibits LAD2 human mast cell line. Synta66 (10 μM) significantly inhibits FcεRI stimulated histamine and TNFα secretion, and has differential effects on FcεRI stimulated prostaglandin D2 and cytokine release in human lung mast cells (HLMCs)[3].

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

PROTOCOL

Cell Assay [3]

Human Lung Mast Cell (HLMCs) are cultured in DMEM+Glutamax media containing 1% antibiotic-antimycotic solution, 1% non-essential amino acids, 10% fetal calf serum and supplemented with 100 ng/mL human stem cell factor, 50 ng/mL IL-6 and 10 ng/mL IL-10. For histamine assays mast cells are isolated from human lung tissue and used within 24 h[3].

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

CUSTOMER VALIDATION

- Mater Sci Eng C Mater Biol Appl. 24 October 2021, 112503.
- Cell Signal. 2023 Apr 14;110681.
- Science in Dentistry, University of Pennsylvania. 2019 Jun.

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REFERENCES

[1]. Molnár T, et al. Store-Operated Calcium Entry in Müller Glia Is Controlled by Synergistic Activation of TRPC and Orai Channels. J Neurosci. 2016 Mar 16;36(11):3184-98.

[2]. van Kruchten R, et al. Antithrombotic potential of blockers of store-operated calcium channels in platelets. Arterioscler Thromb Vasc Biol. 2012 Jul;32(7):1717-23.

[3]. Wajdner HE, et al. Orai and TRPC channel characterization in FcεRI-mediated calcium signaling and mediator secretion in human mast cells. Physiol Rep. 2017 Mar;5(5).

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