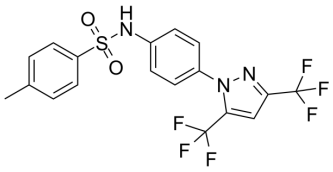
Pyr10

Cat. No.:	HY-19408
CAS No.:	1315323-00-2
Molecular Formula:	$C_{18}H_{13}F_6N_3O_2S$
Molecular Weight:	449.37
Target:	TRP Channel
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling
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 : 100 mg/mL (222.53 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2253 mL	11.1267 mL	22.2534 mL
5 mM	0.4451 mL	2.2253 mL	4.4507 mL
10 mM	0.2225 mL	1.1127 mL	2.2253 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 (5.56 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.56 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pyr10 is a pyrazole derivative and a selective TRP cation 3 (TRPC3) inhibitor. Pyr10 inhibits Ca^{2+} influx in carbachol-stimulated TRPC3-transfected HEK293 cells with an IC_{50} of 0.72 μM (IC_{50} of 13.08 μM for store operated Ca^{2+} entry in BRL-2H3 cells). Pyr10 has the ability to distinguish between receptor-operated TRPC3 and native stromal interaction molecule 1 (STIM1)/Orai1 channels[1].

IC_{50} & Target

TRPC3
0.72 μM (IC_{50})

In Vitro

Pyr10 has the ability to discriminate between the classical Orai-mediated, highly Ca^{2+} selective signalling pathway and the phospholipase C-dependent Ca^{2+} entry-mediated by TRPC channels, specifically by TRPC3. Pyr10 (3 μM) completely

	eliminates TRPC3 currents as well as Ca^{2+} entry while exerting modest effects on Orai-mediated responses. The selective block of TRPC3 channels by Pyr10 barely affected mast cell activation[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Genetic deletion ($TRPC3^{-/-}$) and pharmacological channel blockade with Pyr10 blunts ventricular cardiac fibroblast activation and myocardial fibrosis in N(ω)-nitro-l-arginine methyl ester (l-NAME) hypertensive mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Cell Mol Med. 2020 Jan;24(1):488-510.

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REFERENCES

[1]. Schleifer H, et al. Novel pyrazole compounds for pharmacological discrimination between receptor-operated and store-operated Ca(2+) entry pathways. Br J Pharmacol. 2012 Dec;167(8):1712-1722.

[2]. Saliba Y, et al. Transient Receptor Potential Canonical 3 and Nuclear Factor of Activated T Cells C3 Signaling Pathway Critically Regulates Myocardial Fibrosis. Antioxid Redox Signal. 2019 Jun 1;30(16):1851-1879.

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