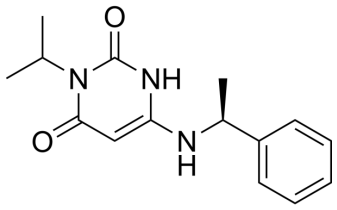
Mavacamten

Cat. No.:	HY-109037
CAS No.:	1642288-47-8
Molecular Formula:	$C_{15}H_{19}N_3O_2$
Molecular Weight:	273.33
Target:	Myosin
Pathway:	Cytoskeleton
Storage:	Powder -20°C 3 years In solvent -80°C 1 year -20°C 6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 83.33 mg/mL (304.87 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.6586 mL	18.2929 mL	36.5858 mL
	5 mM	0.7317 mL	3.6586 mL	7.3172 mL
	10 mM	0.3659 mL	1.8293 mL	3.6586 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 (9.15 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (7.61 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Mavacamten (MYK461) is an orally active modulator of cardiac myosin, with IC_{50}s of 490, 711 nM for bovine cardiac and human cardiac, respectively.

IC_{50} & Target

IC50: 490 nM (bovine cardiac), 711 nM (human cardiac)[1].

In Vitro

Mavacamten is found to have an IC_{50} value of 490 nM in the bovine system, 711 nM in the human system, and 2140 nM in the rabbit system, indicating selectivity of >4-fold for cardiac myosin[1].

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

In Vivo

Treatment with Mavacamten reduces FS from 52±3% to 38±7%. Treatment with Mavacamten reduces FS from 81±7% to 60±13%, corresponding to a relative reduction of 25%. Across all measurements there is a linear correlation between FS and

Mavacamten plasma concentrations with each 100 ng/mL increase in Mavacamten concentration lowering FS by 4.9%[2]. Mavacamten reduces contractility by decreasing the adenosine triphosphatase activity of the cardiac myosin heavy chain. Chronic administration of Mavacamten suppresses the development of ventricular hypertrophy, cardiomyocyte disarray, and myocardial fibrosis and attenuates hypertrophic and profibrotic gene expression in mice harboring heterozygous human mutations in the myosin heavy chain[3].

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PROTOCOL

Animal Administration [2]

Cats[2]

Five cats are selected for study. At the completion of imaging, a tenminute intravenous infusion of Mavacamten (MYK-461 (n=5)) at 0.3 mg/kg/hr IV is started. Focused echocardiography is performed after five minutes. After ten minutes, the Mavacamten infusion rate is lowered to 0.12 mg/kg/hr IV, a blood sample is drawn and an echocardiogram performed. If ventricular function remains hypercontractile or within normal limits by visual inspection, another blood sample is obtained and the Mavacamten infusion rate is increased to 0.36 mg/kg/hr IV for ten minutes. Focused echocardiography is performed after five minutes. After ten minutes, the Mavacamten infusion rate is lowered to 0.15 mg/kg/hr IV, a blood sample is drawn and an echocardiogram performed[2].

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

CUSTOMER VALIDATION

- J Gen Physiol. 2023 Mar 6;155(3):e202113054.
- bioRxiv. 2023 Apr 14.

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REFERENCES

[1]. Kawas RF, et al. A small-molecule modulator of cardiac myosin acts on multiple stages of the myosin chemomechanical cycle. J Biol Chem. 2017 Oct 6;292(40):16571-16577.

[2]. Stern JA, et al. A Small Molecule Inhibitor of Sarcomere Contractility Acutely Relieves Left Ventricular Outflow Tract Obstruction in Feline Hypertrophic Cardiomyopathy. PLoS One. 2016 Dec 14;11(12):e0168407.

[3]. Green EM, et al. A small-molecule inhibitor of sarcomere contractility suppresses hypertrophic cardiomyopathy in mice. Science. 2016 Feb 5;351(6273):617-21.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA