Trimetazidine

Cat. No.: HY-B0968A

CAS No.: 5011-34-7

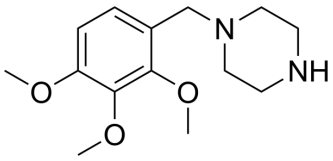
Molecular Formula: $C_{14}H_{22}N_2O_3$

Molecular Weight: 266.34

Target: Autophagy

Pathway: Autophagy

Storage: 4°C, protect from light, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light, stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 125 mg/mL (469.32 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.7546 mL	18.7730 mL	37.5460 mL
	5 mM	0.7509 mL	3.7546 mL	7.5092 mL
	10 mM	0.3755 mL	1.8773 mL	3.7546 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: ≥ 2.08 mg/mL (7.81 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (7.81 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (7.81 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Trimetazidine is a selective long chain 3-ketoyl coenzyme A thiolase inhibitor with an IC_{50} of 75 nM, which can inhibit β-oxidation of free fatty acid (FFA). Trimetazidine is an effective antianginal agent and a cytoprotective agent, has anti-oxidant, anti-inflammatory, antinociceptive and gastroprotective properties. Trimetazidine triggers autophagy. Trimetazidine is also a 3-hydroxyacyl-CoA dehydrogenase (HADHA) inhibitor[1][2][3][4].

IC_{50} & Target

IC50: 75 nM (long chain 3-ketoyl coenzyme A thiolase)[2]
β-oxidation[2]

	Autophagy[3] 3-hydroxyacyl-CoA dehydrogenase (HADHA)[4]
In Vitro	Trimetazidine (1-100 μM; 24 hours; HUVECs) could enhance the viability of the injured HUVECs induced by oxidation in a certain dose-dependent manner[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. Cell Viability Assay[1]

Cell Line:	Human umbilical vein endothelial cells (HUVECs)
Concentration:	1 μM,10 μM,100 μM
Incubation Time:	24 hours
Result:	Enhanced the viability of the injured HUVECs induced by oxidation.

In Vivo

Trimetazidine (5-20 mg/kg; oral administration; 1 hour; Swiss albino male mice) in 10 and 20mg/kg doses significantly raises the seizure-threshold current in the ICES test in the mice[5].

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

Animal Model:	Swiss albino male mice (24-35 g)[4]
Dosage:	5 mg/kg, 10 mg/kg and 20 mg/kg; 10 mL/kg body weight
Administration:	Oral administration ; 1 hour
Result:	In 10 and 20mg/kg doses significantly raised the seizure-threshold current in the ICES test.

CUSTOMER VALIDATION

- Mol Cell. 2020 Oct 1;80(1):43-58.e7.
- Acta Pharmacol Sin. 2022 Feb 25.
- J Pharmaceut Biomed. 2020, 113870.
- Anatol J Cardiol. 2019 Nov;22(5):232-239.

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REFERENCES

[1]. Shenghu He, et al. Protective effects of trimetazidine against vascular endothelial cell injury induced by oxidation. Journal of Geriatric Cardiology, December 2008 , Vol 5 No 4.

[2]. Kantor PF, et al. The antianginal drug trimetazidine shifts cardiac energy metabolism from fatty acid oxidation to glucose oxidation by inhibiting mitochondrial long-chain 3-ketoacyl coenzyme A thiolase. Circ Res. 2000 Mar 17;86(5):580-8.

[3]. Jain S, et al. Trimetazidine exerts protection against increasing current electroshock seizure test in mice. Seizure. 2010 Jun;19(5):300-2.

[4]. Chrusciel P, et al. Defining the role of trimetazidine in the treatment of cardiovascular disorders: some insights on its role in heart failure and peripheral artery disease. Drugs. 2014 Jun;74(9):971-80.

[5]. Hossain F, et al.Inhibition of Fatty Acid Oxidation Modulates Immunosuppressive Functions of Myeloid-Derived Suppressor Cells and Enhances Cancer Therapies. Cancer Immunol Res. 2015 Nov;3(11):1236-47.

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