10,12-Tricosadiynoic acid

Cat. No.: HY-135425

CAS No.: 66990-30-5

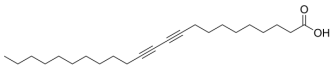
Molecular Formula: $C_{23}H_{38}O_2$

Molecular Weight: 346.55

Target: Acyltransferase

Pathway: Metabolic Enzyme/Protease

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 : 100 mg/mL (288.56 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8856 mL	14.4279 mL	28.8559 mL
	5 mM	0.5771 mL	2.8856 mL	5.7712 mL
	10 mM	0.2886 mL	1.4428 mL	2.8856 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: 5 mg/mL (14.43 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 5 mg/mL (14.43 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 5 mg/mL (14.43 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

10,12-Tricosadiynoic acid is a highly specific, selective, high affinity and orally active acyl-CoA oxidase-1 (ACOX1) inhibitor. 10,12-Tricosadiynoic acid can treat high fat diet- or obesity-induced metabolic diseases by improving mitochondrial lipid and ROS metabolism[1]. 10,12-Tricosadiynoic acid is a click chemistry reagent, it contains an Alkyne group and can undergo copper-catalyzed azide-alkyne cycloaddition (CuAAc) with molecules containing Azide groups.

IC_{50} & Target

Acyl-CoA oxidase-1 (ACOX1)[1].

In Vitro

10,12-Tricosadiynoic acid-CoA rapidly inhibits ACOX1 activity in a time- and concentration-dependent manner. The activity

of ACOX1 decreases by nearly 95% after 5 min of incubation with 10 eq of 10,12-Tricosadiynoic acid-CoA. ACOX1 activity is inhibited only if free 10,12-Tricosadiynoic Acid is activated as the CoA thioester, the substrate form. Inhibition of ACOX1 by 10,12-Tricosadiynoic acid-CoA is irreversible. And the kinetics parameters KI and kinact are calculated to be 680 nm and 3.18 min^{1}, respectively[1].

10,12-Tricosadiynoic acid is the precursor of 10,12-Tricosadiynoic acid-CoA and is transformed into 10,12-Tricosadiynoic acid-CoA by peroxisomal very long chain acyl-CoA synthetase (VLACS) after entering into cells, and it inhibits ACOX1 in vivo[1].

10,12-Tricosadiynoic acid (500 nM) inhibits acyl-CoA oxidase-1 (ACOX1) activity. 10,12-Tricosadiynoic acid treatment abrogates the protective effect by Sirt5 siRNA[2].

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

In Vivo

10,12-Tricosadiynoic acid (100 μg/kg; oral gavage; daily; for 8 weeks; male Wistar rats) treatment increases hepatic mitochondrial fatty acid oxidation (FAO) via activation of the SIRT1-AMPK (adenosine 5′-monophosphate-activated protein kinase) pathway and proliferator activator receptor α and reduces hydrogen peroxide accumulation in high fat diet-fed rats, which significantly decreases hepatic lipid and ROS contents, reduces body weight gain, and decreases serum triglyceride and insulin levels[1].

10,12-Tricosadiynoic acid (0 mg/kg, 37.5 mg/kg, 75 mg/kg, and 150 mg/kg diet) treatment does not affect weight gain, but significantly decreases peroxisomal β-oxidation in the liver, and increased body fat accumulation in Nile tilapia. The fish with impaired peroxisomal β-oxidation exhibited higher contents of serum lipid and peroxidation products, and alanine aminotransferase activity, and significantly lowered hepatic activities of superoxide dismutase and catalase[3].

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

Animal Model:	Male Wistar rats (210-230 g) fed with high fat diet[1]
Dosage:	100 μg/kg
Administration:	Oral gavage; daily; for 8 weeks
Result:	Reduced hydrogen peroxide accumulation in high fat diet-fed rats, which significantly decreased hepatic lipid and ROS contents, reduced body weight gain, and decreased serum triglyceride and insulin levels.

CUSTOMER VALIDATION

- Environ Int. 2023 Aug 8;178:108138.
- Phytomedicine. 2023 Nov 3, 155183.
- Dev Comp Immunol. 2022 Aug 9;104501.

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REFERENCES

[1]. Zeng J, et al. Specific Inhibition of Acyl-CoA Oxidase-1 by an Acetylenic Acid Improves Hepatic Lipid and Reactive Oxygen Species (ROS) Metabolism in Rats Fed a High Fat Diet. J Biol Chem. 2017 Mar 3;292(9):3800-3809.

[2]. Takuto Chiba, et al. Sirtuin 5 Regulates Proximal Tubule Fatty Acid Oxidation to Protect against AKI. J Am Soc Nephrol. 2019 Dec;30(12):2384-2398.

[3]. Yan Liu, et al. Impaired peroxisomal fat oxidation induces hepatic lipid accumulation and oxidative damage in Nile tilapia. Fish Physiol Biochem. 2020 Aug;46(4):1229-1242.

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

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