# 10,12-Tricosadiynoic acid

**Cat. No.:** HY-135425

**CAS No.:** 66990-30-5

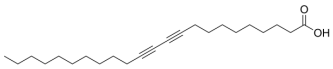
**Molecular Formula:** $C_{23}H_{38}O_2$

**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 | Concentration / Solvent / Mass | 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.

See more customer validations on www.MedChemExpress.com

## 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.**

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