α-Lipoic Acid

Cat. No.: HY-N0492

CAS No.: 1077-28-7

Molecular Formula: $C_8H_{14}O_2S_2$

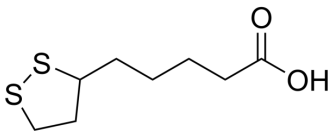
Molecular Weight: 206.33

Target: NF-κB; HIV; Mitochondrial Metabolism; Endogenous Metabolite; Apoptosis

Pathway: NF-κB; Anti-infection; Metabolic Enzyme/Protease; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (484.66 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic) (insoluble)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.8466 mL	24.2330 mL	48.4660 mL
	5 mM	0.9693 mL	4.8466 mL	9.6932 mL
	10 mM	0.4847 mL	2.4233 mL	4.8466 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 10 mg/mL (48.47 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (12.12 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (12.12 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (12.12 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

α-Lipoic Acid (Thioctic acid) is an antioxidant, which is an essential cofactor of mitochondrial enzyme complexes. α-Lipoic Acid inhibits NF-κB-dependent HIV-1 LTR activation[1][2][3]. α-Lipoic Acid induces endoplasmic reticulum (ER) stress-mediated apoptosis in hepatoma cells[4]. α-Lipoic Acid can be used with CPUL1 (HY-151802) to construct the self-assembled nanoaggregate CPUL1-LA NA, which has improved antitumor efficacy than CPUL1[5].

IC_{50} & Target	Human Endogenous Metabolite	NF-κB	Mitochondrial bioenergetics	HIV-1

In Vitro

The long terminal repeat (LTR) of HIV-1 is the target of cellular transcription factors such as NF-κB, and serves as the promoter-enhancer for the viral genome when integrated in host DNA[1]. α-Lipoic Acid (Alpha-Lipoic acid, ALA), a naturally occurring dithiol compound, plays an essential role in mitochondrial bioenergetics. α-Lipoic Acid reduces lipid accumulation in the liver by regulating the transcriptional factors SREBP-1, FoxO1, and Nrf2, and their downstream lipogenic targets via the activation of the SIRT1/LKB1/AMPK pathway. Treatment of cells with α-Lipoic Acid (250, 500 and 1000 μM) significantly increases the NAD^+/NADH ratio in HepG2 cells ($P<0.05$ or $P<0.01$). Treatment with α-Lipoic Acid (50, 125, 250 and 500 μM) increases SIRT1 activity in HepG2 cells. α-Lipoic Acid (50, 125, 250, 500 and 1000 μM) increases phosphorylation of AMPK and acetyl-CoA carboxylase (ACC) in HepG2 cells in a dose-dependent fashion[1].

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

In Vivo

C57BL/6J mice, divided into four groups, are fed an high-fat diet (HFD) for 24 weeks to induce nonalcoholic fatty liver disease (NAFLD) followed by daily administration of α-Lipoic Acid. Then, the effects of α-Lipoic Acid on hepatic lipid accumulation in long-term HFD-fed mice are assessed. Administration of α-Lipoic Acid (100 mg/kg or 200 mg/kg) markedly reduces visceral fat mass in mice. In addition, α-Lipoic Acid (100 mg/kg or 200 mg/kg) treatment inhibits the appetite and causes a dramatic weight loss (all $P<0.05$)[1].

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

PROTOCOL

Cell Assay [1]

The human hepatocellular carcinoma (HepG2) cell line is cultured in Dulbecco's modified Eagle's medium containing 10% fetal bovine serum at 37°C and 5% CO_2. HepG2 cells are treated with AMPK inhibitor (CC, 20 μM, 0.5 h), SIRT1 inhibitor (NA, 10 mM, 12 or 24 h), and AMPK activator (AICAR, 2 mM, 1 h), Palmitate (PA, 125 μM, 12 h) and α-Lipoic Acid (250 μM, 6 or 12 h) [1].

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Animal Administration [1]

Mice[1]

Male C57BL/6J mice (6-week-old; body weight: 22-24 g) are allowed ad libitum access to normal diet and water for 2 weeks before dividing into four groups (n=8): normal diet (ND) (10% energy from fat), high-fat diet (HFD) (60% energy from fat) and HFD plus α-Lipoic Acid (100 mg/kg or 200 mg/kg). After 24 weeks of treatment, blood samples are collected after the eyeballs of the mice are extracted for serum preparation by centrifugation at 2000×g for 10 min at 4°C. The liver tissues are harvested in liquid nitrogen and stored at -80°C.

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

CUSTOMER VALIDATION

- J Nanostructure Chem. 13 May 2022.
- Virol Sin. 2021 Sep 12;1-12.
- J Biochem Mol Toxicol. 2023 Sep 15;e23542.
- Oxid Med Cell Longev. 2021 Jun 4.
- Oncotarget. 2018 Jan 30;9(15):12137-12153.

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REFERENCES

[1]. Liu J, et al. Nanoaggregates of Disulfide-Decorated TrxR Inhibitor Promote Cellular Uptake, Selective Targeting, and Antitumor Efficacy. Langmuir, 2022.

[2]. Xiao L, et al. Activity of the dietary antioxidant ergothioneine in a virus gene-based assay for inhibitors of HIV transcription. Biofactors. 2006;27(1-4):157-65.

[3]. Yang Y, et al. Alpha-lipoic acid improves high-fat diet-induced hepatic steatosis by modulating the transcription factors SREBP-1, FoxO1 and Nrf2 via the SIRT1/LKB1/AMPK pathway. J Nutr Biochem. 2014 Nov;25(11):1207-1217.

[4]. Lei D, et al. Synergistic neuroprotective effect of rasagiline and idebenone against retinal ischemia-reperfusion injury via the Lin28-let-7-Dicer pathway. Oncotarget. 2018 Jan 30;9(15):12137-12153.

[5]. Pibiri M, et al. α-Lipoic acid induces Endoplasmic Reticulum stress-mediated apoptosis in hepatoma cells. Sci Rep. 2020 Apr 28;10(1):7139.

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