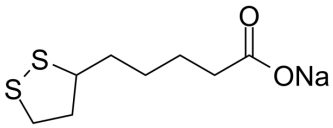
α-Lipoic Acid sodium

Cat. No.:	HY-N0492A
CAS No.:	2319-84-8
Molecular Formula:	$C_8H_{13}NaO_2S_2$
Molecular Weight:	228.31
Target:	NF-κB; HIV; Mitochondrial Metabolism; Endogenous Metabolite; Apoptosis
Pathway:	NF-κB; Anti-infection; Metabolic Enzyme/Protease; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	α-Lipoic Acid (Thioctic acid) sodium is an antioxidant, which is an essential cofactor of mitochondrial enzyme complexes. α-Lipoic Acid sodium inhibits NF-κB-dependent HIV-1 LTR activation[1][2][3]. α-Lipoic Acid sodium induces endoplasmic reticulum (ER) stress-mediated apoptosis in hepatoma cells[4]. α-Lipoic Acid sodium 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				
	HIV-1	NF-κB	Human Endogenous Metabolite	Microbial Metabolite

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].

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]. 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.

[2]. 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.

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

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

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