Ethylenediaminetetraacetic acid disodium dihydrate

Cat. No.: HY-Y0682A

CAS No.: 6381-92-6

Molecular Formula: $C_{10}H_{18}N_2Na_2O_{10}$

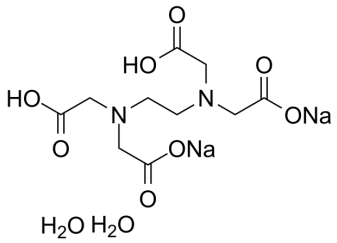
Molecular Weight: 372.24

Target: Biochemical Assay Reagents; Bacterial; SOD

Pathway: Others; Anti-infection; Immunology/Inflammation

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 33.33 mg/mL (89.54 mM; Need ultrasonic)

DMSO : < 1 mg/mL (ultrasonic;warming;heat to 80°C) (insoluble or slightly soluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6864 mL	13.4322 mL	26.8644 mL
5 mM	0.5373 mL	2.6864 mL	5.3729 mL
10 mM	0.2686 mL	1.3432 mL	2.6864 mL

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

BIOLOGICAL ACTIVITY

Description

Ethylenediaminetetraacetic acid (EDTA) disodium dehydrate is a kind of metal chelating agent (binds to bivalent and trivalent metal cations, including calcium). Ethylenediaminetetraacetic acid disodium dehydrate has antibacterial, anti-inflammatory, antioxidant, anti-hypercalcemia and anticoagulant activities. Ethylenediaminetetraacetic acid disodium dehydrate decreases the metal ion-catalyzed oxidative damage to proteins, and allows maintenance of reducing environment during protein purification. Ethylenediaminetetraacetic acid disodium dehydrate can alleviate the liver fibrosis. Ethylenediaminetetraacetic acid disodium dehydrate can be used for coronary artery disease and neural system disease research[1][2][3][4][5][6][7].

In Vitro

Ethylenediaminetetraacetic acid has a strong bactericidal effect on the cell wall of P. aeruginosa and a. faecalis[4].

Ethylenediaminetetraacetic acid (0.005-0.01 M) has good heavy metal extraction in contaminated silty-clay-loam soil columns, can extract Pb, Cd and Zn in a concentration-dependent way with an extraction efficiency sequence of Pb > Cd > Zn [6].

Ethylenediaminetetraacetic acid (1.2 mM) enhances the activity of the CRE driving promoter by activating T-cell death-associated gene 8 (TDAG8) in HEK293T cells, thereby enhancing the production of cAMP in the cells[7].

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

In Vivo

Ethylenediaminetetraacetic acid (60 mg/kg; Intraperitoneal injection; Three times per week for three weeks) can reduce liver fibrosis, lipid peroxidation and liver inflammation in CCl_4 induced liver fibrosis rats, and has antioxidant and anti-inflammatory activities[5].

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

Animal Model:	Male Wistar rat model of cirrhosis induced by CCl_4[5]
Dosage:	60 mg/kg, 120 mg/kg, 240 mg/kg
Administration:	Intraperitoneal injection (i.p.); Three times per week for 3 weeks (during this period, CCl4 administration continued). After CCl_4 and mineral oil mixture treatment (200 μL/mouse; i.p.; Three times per week for eight weeks).
Result:	Kept kept all the rats alive at the 60 mg/kg dose, but died at 120 and 240 mg/kg. Reduced fibrosis of the liver in surviving rats (20%).

Animal Model:	Male Wistar rat model of cirrhosis induced by CCl[5]
Dosage:	60 mg/kg
Administration:	Intraperitoneal injection (i.p.); Three times per week for 3 weeks (during this period, Intraperitoneal injection (i.p.); Three times per week for 11 weeks. (Preventive Ethylenediaminetetraacetic acid (EDTA) group: EDTA and CCl_4 were administered during 11 weeks, three times per week on alternate days). Intraperitoneal injection (i.p.); Three times per week. After CCl_4 treatment (i.p.; Three times per week for eight weeks) (Therapeutic EDTA group: EDTA and CCl_4 were administered for 3 weeks, three times per week on alternate days).
Result:	Increased sod activity by 50% in the preventive EDTA group. (Compared with untreated EDTA group) Increased Cp activity in the preventive EDTA (30%) and therapeutic EDTA (20%) groups. (Compared with the fibrotic group) Decreased mRNA expression of the pro-inflammatory molecule (TNF-α and LI-6) and the profibrogenic molecules (TGF-β and αCOLI) in both the prevention and treatment groups.

CUSTOMER VALIDATION

- Adv Sci (Weinh). 2022 Dec 12;e2204998.
- Int J Biol Macromol. 2022.
- Colloids Surf B Biointerfaces. 2023 Dec 2, 113680.
- Cell Immunol. 2023 Nov 2, 104781.
- bioRxiv. 2023 Sep 8.

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REFERENCES

[1]. Chumanov RS, et al. Artifact-inducing enrichment of ethylenediaminetetraacetic acid and ethyleneglycoltetraacetic acid on anion exchange resins. Anal Biochem. 2011 May 1;412(1):34-9.

[2]. Banfi G, et al. The role of ethylenediamine tetraacetic acid (EDTA) as in vitro anticoagulant for diagnostic purposes. Clin Chem Lab Med. 2007;45(5):565-76.

[3]. Ibad A, et al. Chelation therapy in the treatment of cardiovascular diseases. J Clin Lipidol. 2016 Jan-Feb;10(1):58-62.

[4]. Gray GW, et al. The effect of ethylenediaminetetra-acetic acid on the cell walls of some gram-negative bacteria. J Gen Microbiol. 1965 Jun;39(3):385-99.

[5]. González-Cuevas J, et al. Ethylenediaminetetraacetic acid induces antioxidant and anti-inflammatory activities in experimental liver fibrosis. Redox Rep. 2011;16(2):62-70.

[6]. Naghipour D, et al. Remediation of heavy metals contaminated silty clay loam soil by column extraction with ethylenediaminetetraacetic acid and nitrilo triacetic acid[J]. Journal of Environmental Engineering, 2017, 143(8): 04017026.

[7]. Deai M, et al. Ethylenediaminetetraacetic acid (EDTA) enhances cAMP production in human TDAG8-expressing cells. Biochem Biophys Res Commun. 2022 Oct 20;626:15-20.

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