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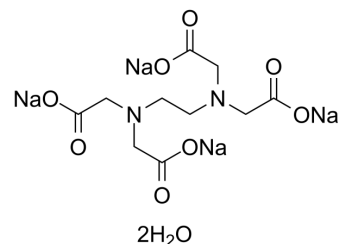
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Ethylenediaminetetraacetic acid sodium hydrate

Cat. No.:	HY-W105700		
CAS No.:	10378-23-1		
Molecular Formula:	C ₁₀ H ₁₆ N ₂ Na ₄ O ₁₀		
Molecular Weight:	416.2		
Target:	Biochemical Assay Reagents; Bacterial; SOD		
Pathway:	Others; Anti-infection; Immunology/Inflammation		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



BIOLOGICAL ACTIVITY

Description	Ethylenediaminetetraacetic acid (EDTA) sodium hydrate is a kind of metal chelating agent (binds to bivalent and trivalent metal cations, including calcium). Ethylenediaminetetraacetic acid sodium hydrate has antibacterial, anti-inflammatory, antioxidant, anti-hypercalcemia and anticoagulant activities. Ethylenediaminetetraacetic acid sodium hydrate decreases the metal ion-catalyzed oxidative damage to proteins, and allows maintenance of reducing environment during protein purification. Ethylenediaminetetraacetic acid sodium hydrate can alleviate the liver fibrosis. Ethylenediaminetetraacetic acid sodium hydrate 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 <i>P. aeruginosa</i> and <i>a. faecalis</i> ^[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.								
	<table border="1"> <tr> <td>Animal Model:</td> <td>Male Wistar rat model of cirrhosis induced by CCl₄^[5]</td> </tr> <tr> <td>Dosage:</td> <td>60 mg/kg, 120 mg/kg, 240 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>Intraperitoneal injection (i.p.); Three times per week for 3 weeks (during this period, CCl₄ administration continued). After CCl₄ and mineral oil mixture treatment (200 µL/mouse; i.p.; Three times per week for eight weeks).</td> </tr> <tr> <td>Result:</td> <td>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%).</td> </tr> </table>	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, CCl ₄ 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%).
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Animal Model:	Male Wistar rat model of cirrhosis induced by CCl ₄ ^[5]
Dosage:	60 mg/kg
Administration:	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 α COLI) 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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- [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.

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

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