4-Hydroxynonenal

Cat. No.: HY-113466

CAS No.: 75899-68-2

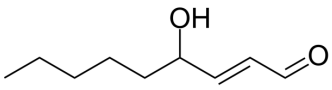
Molecular Formula: $C_9H_{16}O_2$

Molecular Weight: 156.22

Target: Aldehyde Dehydrogenase (ALDH); Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (640.12 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	6.4012 mL	32.0061 mL	64.0123 mL
	5 mM	1.2802 mL	6.4012 mL	12.8025 mL
	10 mM	0.6401 mL	3.2006 mL	6.4012 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (16.00 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (13.31 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (13.31 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

4-Hydroxynonenal (4-HNE) is an α,β unsaturated hydroxyalkenal and an oxidative/nitrosative stress biomarker. 4-Hydroxynonenal is a substrate and an inhibitor of acetaldehyde dehydrogenase 2 (ALDH2). 4-Hydroxynonenal can modulate a number of signaling processes mainly through forming covalent adducts with nucleophilic functional groups in proteins, nucleic acids, and membrane lipids. 4-Hydroxynonenal plays an important role in cancer through mitochondria[1][2][3].

IC_{50} & Target		
	ALDH2	Human Endogenous Metabolite

In Vitro

4-Hydroxynonenal is both a substrate and an inhibitor of ALDH2; inhibition of ALDH2 by 4-Hydroxynonenal is reversible at

	low concentration and become irreversible when the concentration of 4-HNE reaches 10 μM[1]. 4-Hydroxynonenal can induce antioxidant defense mechanisms to restrain its own production and to enhance the cellular protection against oxidative stress[1]. 4-Hydroxynonenal, the product of lipid peroxidation, is mutagenic and genotoxic in viruses, bacteria and mammalian cells. It reacts with all four DNA bases but with different efficiency: G >C > A >T. 4-Hydroxynonenal-dG represents the best biomarker of the genotoxic effects of 4-Hydroxynonenal and these adducts are primarily found in nuclear DNA. A classic example of etiological relevance of 4-Hydroxynonenal-dG in human cancers is 4-Hydroxynonenal-dG induced p53 mutation. 4-Hydroxynonenal-dG adducts were preferentially formed at the third base of codon 249 in the p53 gene, causing gene mutation and affecting diverse biological processes including cell cycle arrest, apoptosis, DNA repair, and differentiation[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Following 24 h after fluid percussion injury (FPI), the mouse brain tissue is analyzed for the expression level of NADPH oxidase 1 (NOX1), inducible nitric oxide synthase (iNOS), 4-Hydroxynonenal (4-HNE. Both wild-type ($Nrf2^{+/+}$) and Nrf2-deficient mice ($Nrf2^{-/-}$) results in increased expression of 4-Hydroxynonenal following 15 psi injury (moderate injury) when compared to uninjured $Nrf2^{+/+}$ and $Nrf2^{-/-}$ mice. Similar to iNOS result, in $Nrf2^{-/-}$ KO mice, the expression level of 4-Hydroxynonenal is significantly high when compared to corresponding injured and uninjured $Nrf2^{+/+}$ WT animals[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Mol Cell. 2023 Nov 16:S1097-2765(23)00913-9.
- Redox Biol. 20 August 2022, 102437.
- Antioxidants (Basel). 2022 Apr 7;11(4):728.
- Research Square Print. 2023 Feb 14.
- Research Square Preprint. 2021 Dec.

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REFERENCES

[1]. Zhong H, et al. Role of lipid peroxidation derived 4-hydroxynonenal (4-HNE) in cancer: focusing on mitochondria. Redox Biol. 2015;4:193-9.

[2]. Csala M, et al. On the role of 4-hydroxynonenal in health and disease. Biochim Biophys Acta. 2015 May;1852(5):826-38.

[3]. Bhowmick S, et al. Traumatic brain injury-induced downregulation of Nrf2 activates inflammatory response and apoptotic cell death. J Mol Med (Berl). 2019 Nov 22.

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