NADH disodium salt

Cat. No.: HY-F0001

CAS No.: 606-68-8

Molecular Formula: $C_{21}H_{27}N_7Na_2O_{14}P_2$

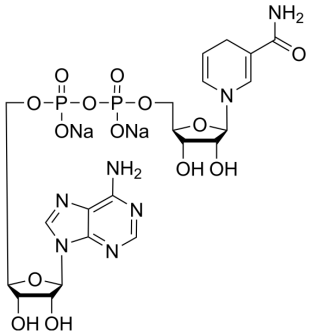
Molecular Weight: 709.4

Target: Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease

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 : ≥ 100 mg/mL (140.96 mM)

DMSO : 100 mg/mL (140.96 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	1.4096 mL	7.0482 mL	14.0964 mL
5 mM	0.2819 mL	1.4096 mL	2.8193 mL
10 mM	0.1410 mL	0.7048 mL	1.4096 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (140.96 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 (3.52 mM); Clear solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (3.52 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

NADH disodium salt (Disodium NADH) is an orally active reduced coenzyme. NADH disodium salt is a donor of ADP-ribose units in ADP-ribosylaton reactions and a precursor of cyclic ADP-ribose. NADH disodium salt plays a role as a regenerative electron donor in cellular energy metabolism, including glycolysis, β-oxidation and the tricarboxylic acid (TCA) cycle[1].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

NADH is unstable under acidic conditions but it is stable under alkaline conditions[2].

NADH (0-1 mM; 0-12 h) increases NAD^+ levels in various mammalian cell lines[3].
NADH (1 mM; 24 h) causes low toxicity and protects cells from genotoxicity[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

NADH (5 μmol/mouse; i.p.; once) increases urinary excretion of nicotinamide and its metabolites in mice[2].
NADH (500 mg/kg; i.g.; once) promotes alcohol metabolism and prevents or ameliorates early liver injury caused by acute alcohol exposure in ethanol-loaded mice[3].
NADH (1000 mg/kg; i.p.; once) enhances tissue NAD^+ levels in male C57BL/6J mice[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male ICR mice[2]
Dosage:	5 μmol/mouse
Administration:	Intraperitoneal injection or oral administration, once
Result:	Produced significant increases in urinary excretions of nicotinamide (Nam) with intraperitoneal injection. Oral administration did not produce any increases in Nam or its metabolites.

Animal Model:	Male C57BL/6J mice[3]
Dosage:	500 mg/kg
Administration:	Intragastric administration, 15 min before ethanol administration
Result:	Significantly increased blood acetaldehyde levels in mice administered with alcohol between 30 min and two hours. Significantly reduced the acetaldehyde in the blood after two hours. Inhibited the decrease of NAD^+/NADH redox ratio in hepatocytes.

CUSTOMER VALIDATION

- Food Chem. 2023 May 5;423:136274.
- Biochemistry. 2023 Nov 10.
- Research Square Preprint. 2023 Sep 15.

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REFERENCES

[1]. Ying W. NAD+ and NADH in cellular functions and cell death. Front Biosci. 2006 Sep 1;11:3129-48.

[2]. Kimura N, et al. Comparison of metabolic fates of nicotinamide, NAD+ and NADH administered orally and intraperitoneally; characterization of oral NADH. J Nutr Sci Vitaminol (Tokyo). 2006 Apr;52(2):142-8.

[3]. Wu K, et al. NADH and NRH as potential dietary supplements or pharmacological agents for early liver injury caused by acute alcohol exposure. Journal of Functional Foods, 2021, 87: 104852.

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

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