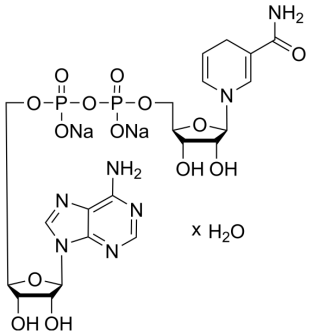
NADH disodium hydrate

Cat. No.:	HY-F0001A
CAS No.:	1949720-50-6
Molecular Formula:	$C_{21}H_{29}N_7O_{14}P_2.xH_2O.2Na$
Target:	Endogenous Metabolite
Pathway:	Metabolic Enzyme/Protease
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

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

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

- Cell Mol Immunol. 2024 Jun;21(6):561-574.
- 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.

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