

Product Data Sheet

Product Name: NADH disodium salt

Cat. No.: GC61830

Chemical Properties

Cas. No. 606-68-8

SMILES NC1=C2C(N([C@H]3[C@H](O)[C@H](O)[C@@H](COP(OP([O-])(OC[C@@H]4[C@@H](O)[C@@H](O)[C@H](N5C=C(C(N)=O)CC=C5)O4)=O)([O-])=O)O3)C=N2)=NC=N1.[Na+].[Na+]

Formula $C_{21}H_{27}N_7Na_2O_{14}P_2$ M.Wt 709.4

Solubility Water : >100 mg/mL (140.96 mM) Storage Store at 2-8°C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

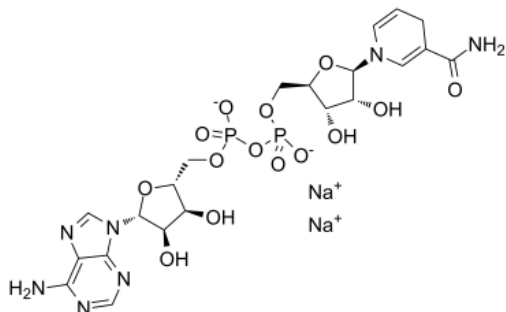
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Protocol

Cell experiment [1]:

Cell lines C2C12, Neuro2a and AML12 cells

Preparation Method C2C12, Neuro2a and AML12 cells were incubated with 1 mM NADH disodium salt or NRH for 24 h.

Reaction Conditions 1mM; 24 h

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Applications NADH disodium salt increases the NAD⁺ concentration to reduce toxicity, and can protect cells in genotoxicity environment.

Animal experiment [2]:

Animal models Male SD rats

Preparation Method NADH disodium salt (10 or 100 mg/kg) was freshly dissolved in sterile 0.9% saline and administered subcutaneously. Rats were decapitated 60 minutes later, and the striatum and midbrain were dissected on ice and homogenized in 1.3 or 0.65 ml of 0.1 N HClO₄, respectively.

Dosage form 10 or 100mg/kg ; s.c.

Applications NADH disodium salt did not influence the tissue content of DA, 5-HT, or their metabolites in the midbrain or striatum.

References:

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

[2] Pearl S M, Antion M D, Stanwood G D, et al. Effects of NADH on dopamine release in rat striatum[J]. Synapse, 2000, 36(2): 95-101.

Background

NADH disodium salt is the sodium salt form of nicotinamide adenine dinucleotide phosphate (NADH), a coenzyme that serves as a regenerative electron donor in cellular energy metabolism, including glycolysis, beta oxidation, and citric acid Loop[1]. NADH can be used for the synthesis and regeneration of tetrahydrobiopterin (BH₄), which is an essential cofactor for the rate-limiting enzyme

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in dopamine (DA) synthesis [2].

In vitro, NADH disodium salt (1mM) treated C2C12, Neuro2a and AML12 cells for 24h, significantly increased the NAD⁺ production of the three cells and protected the cells from genotoxicity [3]. NADH disodium salt (100μM) treated HUVEC and PIEC cells for 30 minutes had no significant effect on intracellular O₂⁻ production [4]. When treatment with NADH disodium salt (100μM) induces rodent β-cells, it cannot enhance depolarization-induced exocytosis [5].

In vivo, subcutaneous injection of NADH disodium salt (10 or 100 mg/kg) in SD rats did not affect the tissue contents of DA, 5-HT and their metabolites in the midbrain or striatum [2]. When treated with intraperitoneal injection of NADH disodium salt (1000mg/kg) in C57BL/6J mice, the NAD⁺ content in the blood, brain, fat and kidney increased several times, and the NAD⁺ content in the liver increased by more than 7 times within 4 hours [3]. Intraperitoneal injection of NADH disodium salt (5μM) into ICR mice significantly increased the urinary excretion of nicotinamide and its metabolites [6].

References:

- [1] Ying W. NAD⁺ and NADH in cellular functions and cell death[J]. Front Biosci, 2006, 11(1): 3129-3148.
- [2] Pearl S M, Antion M D, Stanwood G D, et al. Effects of NADH on dopamine release in rat striatum[J]. Synapse, 2000, 36(2): 95-101.
- [3] Wu K, Li J, Zhou X, et al. NADH and NRH as potential dietary supplements or pharmacological agents for early liver injury caused by acute alcohol exposure[J]. Journal of Functional Foods, 2021, 87: 104852.
- [4] Li, J. Differential NADPH-versus NADH-dependent superoxide production by phagocyte-type endothelial cell NADPH oxidase[J]. Cardiovascular Research, 2001, 52(3):477.
- [5] Ivarsson R, Quintens R, Dejonghe S, et al. Redox control of exocytosis: regulatory role of NADPH, thioredoxin, and glutaredoxin[J]. Diabetes, 2005, 54(7): 2132-2142.
- [6] Kimura N, Fukuwatari T, Sasaki R, et al. Comparison of metabolic fates of nicotinamide, NAD⁺ and NADH administered orally and intraperitoneally; characterization of oral NADH[J]. Journal of nutritional science and vitaminology, 2006, 52(2): 142-148.

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