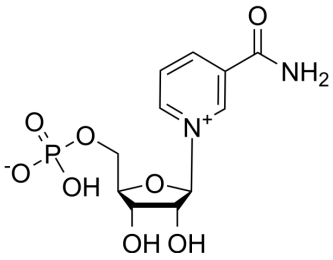
β-Nicotinamide mononucleotide

Cat. No.:	HY-F0004
CAS No.:	1094-61-7
Molecular Formula:	$C_{11}H_{15}N_2O_8P$
Molecular Weight:	334.22
Target:	Endogenous Metabolite
Pathway:	Metabolic Enzyme/Protease
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 83.33 mg/mL (249.33 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.9920 mL	14.9602 mL	29.9204 mL
	5 mM	0.5984 mL	2.9920 mL	5.9841 mL
	10 mM	0.2992 mL	1.4960 mL	2.9920 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 (299.20 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

β-nicotinamide mononucleotide (β-NM) is a product of the nicotinamide phosphoribosyltransferase (NAMPT) reaction and a key NAD^+ intermediate. The pharmacological activities of β-nicotinamide mononucleotide include its role in cellular biochemical functions, cardioprotection, diabetes, Alzheimer's disease, and complications associated with obesity[1].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

β-nicotinamide mononucleotide has several beneficial pharmacological activities. Mostly mediated by its involvement in NAD^+ biosynthesis, the pharmacological activities of NMN include its role in cellular biochemical functions, cardioprotection, diabetes, Alzheimer's disease, and complications associated with obesity[1].

The intracellular NAD^+ levels are significantly decreased by knockdown or knockout of Nampt (Nampt KD or Nampt KO) or treatment with Nampt inhibitor FK866, whereas NAD^+ levels are dramatically increased by supplement of NAD^+ precursors NAM or NMN (0.5-1 mM). NAD^+ precursor NMN treatment inhibited $CD8^+$ T cells activation and function[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

β-Nicotinamide mononucleotide (500 mg/kg; i.p.; 3 times per week for 7-10 week) prevents mtDNA damage and Dox-induced cardiac dysfunction[3].

Nampt KO markedly inhibits tumor progression, whereas Nampt metabolite β-Nicotinamide mononucleotide (300 mg/kg body weight; i.p.; once every two days for 2 weeks) significantly promotes tumor growth in C57BL/6 mice (bearing wildtype Hepa1-6 cells). The reduction and increase in NAD^+ level of respective Nampt KO and β-Nicotinamide mononucleotide-treated tumors are confirmed[2].

β-nicotinamide mononucleotide ameliorates glucose intolerance by restoring NAD^+ levels in HFD-induced T2D mice. β-nicotinamide mononucleotide also enhances hepatic insulin sensitivity and restores gene expression related to oxidative stress, inflammatory response, and circadian rhythm, partly through SIRT1 activation[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL6 mice ($p53^{-/-}$ mice)[3]
Dosage:	500 mg/kg
Administration:	I.p.; 3 times per week for 7-10 week
Result:	Prevented the significant decline in cardiac function of Dox-treated $p53^{-/-}$ mice (study week 7 versus 10) along with rescuing the decreased mitochondrial respiration and tissue ATP depletion caused by Doxorubicin (Dox).

CUSTOMER VALIDATION

- Cell Metab. 2021 Jan 5;33(1):110-127.e5.
- Nat Commun. 2023 Jan 16;14(1):240.
- Nat Commun. 2022 Aug 6;13(1):4583.
- Hepatology. 2022 Jul 11.
- Proc Natl Acad Sci U S A. 2019 Sep 24;116(39):19626-19634.

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REFERENCES

[1]. Poddar SK, et al. Nicotinamide Mononucleotide: Exploration of Diverse Therapeutic Applications of a Potential Molecule. Biomolecules. 2019;9(1):34. Published 2019 Jan 21.

[2]. Lv H, et al. NAD+ Metabolism Maintains Inducible PD-L1 Expression to Drive Tumor Immune Evasion [published online ahead of print, 2020 Nov 3]. Cell Metab. 2020;S1550-4131(20)30554-4.

[3]. Li J, et al. p53 prevents doxorubicin cardiotoxicity independently of its prototypical tumor suppressor activities. Proc Natl Acad Sci U S A. 2019;116(39):19626-19634.

[4]. Yoshino J, et al Nicotinamide mononucleotide, a key NAD(+) intermediate, treats the pathophysiology of diet- and age-induced diabetes in mice. Cell Metab. 2011;14(4):528-536.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA