β -Nicotinamide mononucleotide-d₄

MedChemExpress

Cat. No.:	HY-F0004S		
Molecular Formula:	$C_{11}H_{11}D_4N_2O_8P$		
Molecular Weight:	338.24		
Target:	Endogenous Metabolite		
Pathway:	Metabolic Enzyme/Protease		
Storage:	Powder	-20°C	3 years
	In solvent	-80°C	6 months
		-20°C	1 month

SOLVENT & SOLUBILITY

In Vitro

PBS (pH 7.2) : ≥ 10 mg/mL (29.56 mM) * "≥" means soluble, but saturation unknown.

Solvent Mass Concentration	1 mg	5 mg	10 mg
1 mM	2.9565 mL	14.7824 mL	29.5648 mL
5 mM	0.5913 mL	2.9565 mL	5.9130 mL
10 mM	0.2956 mL	1.4782 mL	2.9565 mL
	Solvent Mass Concentration 1 mM 5 mM 10 mM	Mass Solvent1 mgConcentration1 mM1 mM2.9565 mL5 mM0.5913 mL10 mM0.2956 mL	Solvent Mass 1 mg 5 mg Concentration 1 mM 2.9565 mL 14.7824 mL 1 mM 0.5913 mL 2.9565 mL 10 mM 0.2956 mL 1.4782 mL

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

BIOLOGICAL ACTIVITY				
Description	β-Nicotinamide mononucleotide-d ₄ is the deuterium labeled β-Nicotinamide mononucleotide. 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].			
In Vitro	Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.			

REFERENCES

[1]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019;53(2):211-216.

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

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 $D H_2N$

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[3]. 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.

[4]. 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.

[5]. 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.

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

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