Small Molecules

β-Nicotinamide Mononucleotide

Intermediate in the biosynthesis of nicotinamide adenine dinucleotide

Catalog #100-1127

100 mg

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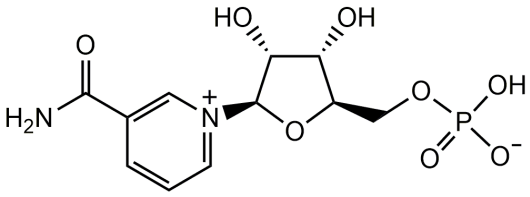
Product Description

β-Nicotinamide Mononucleotide (β-NMN) is an intermediate in the biosynthesis of nicotinamide adenine dinucleotide (NAD+) (Bogan & Brenner). β-NMN is a product of the nicotinamide phosphoribosyltransferase (NAMPT) reaction and is converted to NAD+ by nicotinamide-nucleotide adenylyltransferase (Gallí et al.).

Alternative Names:	β-NMN
CAS Number:	1094-61-7
Chemical Formula:	$C_{11}H_{15}N_2O_8P$
Molecular Weight:	334.2 g/mol
Purity:	≥ 95%
Chemical Name:	3-(aminocarbonyl)-1-(5-O-phosphono-β-D-ribofuranosyl)-pyridinium, inner salt
Structure:	

Properties

Physical Appearance:	A crystalline solid
Storage:	Product stable at -20°C as supplied. As a precaution, STEMCELL recommends storing all small molecules away from direct light. For long-term storage, store with a desiccant. Stable as supplied for 12 months from date of receipt.
Solubility:	• PBS (pH 7.2) ≤ 25 mM For example, to prepare a 10 mM stock solution in PBS (pH 7.2), resuspend 10 mg in 2.99 mL of PBS (pH 7.2). Prepare stock solution fresh before use. Information regarding stability of small molecules in solution has rarely been reported; however, as a general guide we recommend storage in PBS (pH 7.2) at -20°C. Aliquot into working volumes to avoid repeated freeze-thaw cycles. The effect of storage of stock solution on compound performance should be tested for each application. For use as a cell culture supplement, stock solution should be diluted into culture medium immediately before use.

Published Applications

MAINTENANCE AND SELF-RENEWAL

· Delays apoptosis in HL60 cells with MCT1 and MCT4 inhibition by restoring NAD+ levels and ATP depletion (Benjamin et al.).
· Protects MC3T3-E1 cells from aluminum-induced oxidative stress and inhibits thioredoxin-interacting protein (TXNIP)-NLRP3 inflammasome pathway and pro-inflammatory cytokine production (Liang et al.).

DISEASE MODELING

· Reported to treat the pathophysiology of diet- and age-induced type 2 diabetes in mice. β-NMN ameliorates glucose intolerance by restoring NAD+ levels in type 2 diabetic mice models. β-NMN also enhances hepatic insulin sensitivity and restores gene expression related to inflammatory response, oxidative stress and circadian rhythm, partly through SIRT1 (Yoshino et al.).
· Reported to diminish several old age-associated pathologies (decrease body weight gain, enhance energy metabolism, improve insulin sensitivity and eye function) in mouse models of aging (Mills et al.).
· Restores SIRT1 activity and reverse age-related arterial dysfunction by decreasing oxidative stress in mouse models of aging (Picciotto et al.).
· Reported to improve aging-induced cerebrovascular endothelial dysfunction and neurovascular coupling responses in old mice. Restores NAD+ and mitochondrial energetics and reduces mtROS in aged cerebromicrovascular endothelial cells (Tarantini et al.).
· Promotes mesenchymal stromal cell (MSC) expansion in vitro and in aged mice and upregulates SIRT1. Also, it is shown to stimulate osteogenesis of endogenous MSCs and protect bone from aging and irradiation induced damage in mice (Song et al.).

References

Benjamin D et al. (2018) Dual inhibition of the lactate transporters MCT1 and MCT4 is synthetic lethal with metformin due to NAD+ depletion in cancer cells. Cell Rep 25(11): 3047-3058.e4.

Bogan KL & Brenner C. (2008) Nicotinic acid, nicotinamide, and nicotinamide riboside: a molecular evaluation of NAD + precursor vitamins in human nutrition. Annu Rev Nutr 28(1): 115–30.

Gallí M et al. (2010) The nicotinamide phosphoribosyltransferase: a molecular link between metabolism, inflammation, and cancer. Cancer Res 70(1): 8–11.

Liang H et al. (2019) Nicotinamide mononucleotide alleviates aluminum induced bone loss by inhibiting the TXNIP-NLRP3 inflammasome. Toxicol Appl Pharmacol 362: 20–7.

Mills KF et al. (2016) Long-term administration of nicotinamide mononucleotide mitigates age-associated physiological decline in mice. Cell Metab 24(6): 795–806.

Picciotto NE et al. (2016) Nicotinamide mononucleotide supplementation reverses vascular dysfunction and oxidative stress with aging in mice. Aging Cell 15(3): 522–30.

Song J et al. (2019) Nicotinamide mononucleotide promotes osteogenesis and reduces adipogenesis by regulating mesenchymal stromal cells via the SIRT1 pathway in aged bone marrow. Cell Death Dis 10(5): 336.

Tarantini S et al. (2019) Nicotinamide mononucleotide (NMN) supplementation rescues cerebromicrovascular endothelial function and neurovascular coupling responses and improves cognitive function in aged mice. Redox Biol 24: 101192.

Yoshino J et al. (2011) Nicotinamide mononucleotide, a key NAD+ intermediate, treats the pathophysiology of diet- and age-induced diabetes in mice. Cell Metab 14(4): 528–36.

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