BIOMOD LABS · RESEARCH PEPTIDE CO.
RESEARCH COMPOUND MONOGRAPH · SERIES 1 · NO. 009

NAD⁺ (Nicotinamide Adenine Dinucleotide)

An endogenous dinucleotide cofactor essential for cellular redox chemistry and the substrate for sirtuin and PARP enzymes — supplied as research-grade NAD⁺ for laboratory and cell-culture studies of cellular metabolism, sirtuin biology, and DNA-damage-response pathways.

CAS REGISTRY	CATALOG REFERENCE	CLASS	DATE OF ISSUE
53-84-9	BM-LYO-009	Dinucleotide cofactor · non-peptide	May 2026

Nicotinamide adenine dinucleotide (NAD⁺) is an endogenous dinucleotide cofactor essential for cellular redox biology and metabolic enzymology. The molecule consists of two nucleotides — adenosine 5'-monophosphate and nicotinamide mononucleotide — joined by a pyrophosphate linkage between their 5'-phosphate groups. NAD⁺ serves four principal cellular functions: (i) as the redox cofactor in hundreds of dehydrogenase and oxidoreductase enzymatic reactions, cycling between the oxidised NAD⁺ and reduced NADH forms; (ii) as the substrate for sirtuin deacetylase enzymes (SIRT1-SIRT7), which couple NAD⁺ hydrolysis to protein deacetylation in lifespan-modulating chromatin and metabolic-regulation pathways; (iii) as the substrate for poly-ADP-ribose polymerases (PARP1, PARP2 etc.), which engage NAD⁺ in DNA-damage-response signalling; and (iv) as a precursor for additional signalling nucleotides including cyclic ADP-ribose. Plasma and intracellular NAD⁺ levels decline with age, and the chemistry restoration of NAD⁺ pools through precursor supplementation or direct administration has emerged as a substantial area of preclinical and translational research. ***This monograph summarises published cellular biology findings for laboratory research reference only.***

01 Compound Profile

COMMON DESIGNATION	NAD^+ · Nicotinamide adenine dinucleotide · DPN (legacy)
CHEMICAL STRUCTURE	Adenosine 5'-monophosphate joined through pyrophosphate to nicotinamide mononucleotide; oxidised form (NAD^+) carries a positive charge on the nicotinamide ring
CAS REGISTRY	53-84-9
MOLECULAR FORMULA	$C_{21}H_{27}N_7O_{14}P_2$ (free acid)
MOLECULAR MASS	663.43 g · mol^{-1}
PRINCIPAL CELLULAR FUNCTIONS	Redox cofactor (NAD^+/NADH cycling); sirtuin (SIRT1-SIRT7) substrate; PARP substrate; signalling nucleotide precursor[1]
PHYSICAL FORM	White to pale yellow lyophilised solid
SOLUBILITY (LAB RECONSTITUTION)	Water-soluble in sterile water for injection at neutral to slightly basic pH
CRITICAL CHEMISTRY NOTE	**NAD^+ MUST NOT be reconstituted with bacteriostatic water containing benzyl alcohol**. Benzyl alcohol degrades NAD^+ in solution. Sterile water for injection is the appropriate reconstitution vehicle
STORAGE (RESEARCH HANDLING)	Lyophilised solid: −18 °C, desiccated; reconstituted solution refrigerated 2–8 °C and used promptly; long-term frozen aliquots at −18 °C; the molecule is moisture- and pH-sensitive — strongly acidic or strongly basic conditions accelerate hydrolytic degradation
ANALYTICAL SPECIFICATION	≥ 98 % purity by HPLC (BIOMOD Labs internal release specification)

02 Chemistry and Biochemistry

NAD^+ IS ONE OF THE MOST FUNDAMENTAL COFACTORS IN CELLULAR BIOCHEMISTRY. THE OXIDISED FORM (NAD^+) carries a positive charge on the nicotinamide ring; the reduced form (NADH) has accepted a hydride and is electrically neutral. Cellular NAD^+/NADH ratios — typically ~700:1 in cytoplasm and ~7:1 in mitochondria — establish the principal redox state of cellular metabolism. The molecule was identified by Harden and Young in 1906 as a heat-stable cofactor essential for yeast fermentation, and the modern understanding of its central role in cellular energy metabolism developed through the foundational biochemistry of Otto Warburg, Hans Krebs, and others in the early 20th century.[1]

The 21st-century renaissance of NAD⁺ research emerged from the discovery that the sirtuin deacetylase enzymes (mammalian SIRT1-SIRT7, yeast Sir2) consume NAD⁺ stoichiometrically as a substrate, coupling NAD⁺ hydrolysis to protein deacetylation. The Sinclair, Imai, Verdin, and Guarente laboratories established sirtuin biology as a principal axis of metabolic and longevity regulation, with NAD⁺ availability — declining with age in tissues — emerging as a rate-limiting factor for sirtuin function. Subsequent characterisation of the NAD⁺-consuming PARP enzymes added the DNA-damage-response axis to the picture.[2]

03 Proposed Mechanisms in Preclinical Models

Research applications of NAD⁺ in cellular and animal preparations operate through multiple parallel mechanisms. **Redox cofactor restoration** — replenishment of NAD⁺ pools depleted by mitochondrial dysfunction, oxidative stress, or PARP hyperactivation supports continued electron transport chain function and ATP synthesis. **Sirtuin activation** — elevated NAD⁺ availability supports increased sirtuin enzymatic activity, with downstream protein deacetylation effects on transcription factors (FOXO, p53, PGC-1α), metabolic enzymes, and chromatin modifications. **PARP substrate provision** — sustained NAD⁺ availability supports PARP-mediated DNA-damage-response signalling without depleting cellular NAD⁺ pools to deleterious levels. **NADPH precursor** — NAD⁺ is phosphorylated by NAD kinase to NADP⁺, which is reduced to NADPH for reductive biosynthesis and glutathione regeneration.[3] IN VITRO

04 Preclinical Findings

SYSTEM	ANIMAL MODEL / PREPARATION	REPORTED OBSERVATION	REF.
Cellular NAD⁺ restoration	Cell-culture metabolic-stress preparations	Restoration of intracellular NAD⁺ pools; recovery of mitochondrial function	1
Sirtuin enzymology	Cell-free and cell-culture SIRT1 assays	NAD⁺ as essential substrate; sirtuin activity rate-limited by NAD⁺ availability	2
Aging biology	Aged rodent tissue preparations	Tissue NAD⁺ levels decline with age; precursor supplementation restores levels in animal models	4
Mitochondrial bioenergetics	Rodent skeletal muscle & cardiac preparations	NAD⁺ restoration supports mitochondrial function in aged or stress preparations	3
DNA-damage-response biology	Cell-culture genotoxic-stress preparations	PARP-mediated DNA repair supported by adequate NAD⁺ availability	5

05 Research Synthesis & Limitations

METHODOLOGICAL NOTES

NAD^+ is one of the most extensively characterised molecules in biology, with foundational biochemistry spanning over a century and contemporary research focused on its declining levels with age and its role as a sirtuin and PARP substrate. For researchers working with the molecule, the principal handling consideration is the chemistry sensitivity of the lyophilised compound and reconstituted solutions. **Bacteriostatic water containing benzyl alcohol must not be used for reconstitution** — benzyl alcohol degrades NAD^+. Sterile water for injection at neutral pH is appropriate. Reconstituted solutions are best used promptly. Strongly acidic or strongly basic conditions accelerate hydrolytic degradation of the pyrophosphate linkage and ring-opening of the nicotinamide moiety.

06 Laboratory Handling, Reconstitution, and Storage

Lyophilised NAD^+ is supplied under research-use specifications. **Reconstitute with sterile water for injection at neutral pH only — never with bacteriostatic water containing benzyl alcohol, which degrades NAD^+.** Lyophilised storage at −18 °C, desiccated; reconstituted solutions held refrigerated 2–8 °C and used promptly; long-term frozen aliquots at −18 °C with strict minimisation of freeze–thaw. Strongly acidic (pH < 4) or strongly basic (pH > 9) conditions should be avoided. Working concentrations are determined by the investigator's experimental design.

07 References

1. Cantó C, Menzies KJ, Auwerx J. NAD^+ Metabolism and the Control of Energy Homeostasis: A Balancing Act between Mitochondria and the Nucleus. *Cell Metab.* 2015;22(1):31–53. pubmed.ncbi.nlm.nih.gov/26118927
2. Imai S, Armstrong CM, Kaeberlein M, Guarente L. Transcriptional silencing and longevity protein Sir2 is an NAD-dependent histone deacetylase. *Nature.* 2000;403(6771):795–800. pubmed.ncbi.nlm.nih.gov/10693811
3. Verdin E. NAD^+ in aging, metabolism, and neurodegeneration. *Science.* 2015;350(6265):1208–1213. pubmed.ncbi.nlm.nih.gov/26785480
4. Yoshino J, Mills KF, Yoon MJ, Imai S. Nicotinamide mononucleotide, a key NAD(+) intermediate, treats the pathophysiology of diet- and age-induced diabetes in mice. *Cell Metab.* 2011;14(4):528–536. pubmed.ncbi.nlm.nih.gov/21982712
5. Fang EF, Lautrup S, Hou Y, Demarest TG, Croteau DL, Mattson MP, Bohr VA. NAD^+ in Aging: Molecular Mechanisms and Translational Implications. *Trends Mol Med.* 2017;23(10):899–916. pubmed.ncbi.nlm.nih.gov/28899755