PEPTIDE RESEARCH OVERVIEW

NAD+

Nicotinamide Adenine Dinucleotide · Investigational Research Compound · Cellular Coenzyme

A plain-language guide to what NAD+ is, how its cellular-energy signaling is designed to work, and what published research has found so far — written for newcomers to peptide science, not just specialists.

COMPOSITION
NAD+
1,000mg

STRUCTURE
Dinucleotide coenzyme built from adenine and nicotinamide riboside units

STUDIED HALF-LIFE
Short plasma half-life (minutes); intracellular pools decline steadily with age

1 What Is NAD+?

Nicotinamide Adenine Dinucleotide (NAD+) is a coenzyme found in every living cell, essential to hundreds of metabolic reactions including the electron transport chain that produces cellular energy. Unlike a peptide or hormone, it is a small dinucleotide molecule, but it is grouped with research compounds here because it is studied and administered similarly, often via IV or subcutaneous routes, for cellular and metabolic research.

NAD+ serves as a required cofactor for sirtuins (proteins linked to cellular aging regulation) and PARP enzymes (involved in DNA repair), both of which consume NAD+ during their activity. Because intracellular NAD+ concentrations decline substantially with age, most research interest centers on restoring youthful NAD+ levels and their downstream effects on mitochondrial function and cellular repair capacity.

NEW TO THIS? KEY TERMS

Coenzyme — a small molecule required by enzymes to carry out their biochemical reactions.

Sirtuins — a family of proteins (e.g., SIRT1) that regulate cellular aging processes and require NAD+ to function.

PARP enzymes — DNA-repair enzymes that consume large amounts of NAD+ during activation.

Electron transport chain — the mitochondrial system that produces cellular energy (ATP); NAD+ is a required electron carrier.

2 How It's Designed to Work

NAD+'s research profile rests on three linked steps — a natural age-related decline that research aims to counteract, a coenzyme signal that powers sirtuin and repair pathways, and a downstream cascade of mitochondrial and cellular effects:

NATURAL DECLINE

AGE-RELATED REDUCTION

Intracellular NAD+ levels fall substantially with age, a decline studied alongside reduced mitochondrial function and the rationale for exogenous research administration.

COENZYME SIGNAL

SIRTUIN & PARP ACTIVATION

Restored NAD+ availability is studied as fueling sirtuin activity (linked to cellular aging regulation) and PARP-mediated DNA repair, both NAD+-dependent processes.

DOWNSTREAM CASCADE

MITOCHONDRIAL & CELLULAR EFFECTS

Downstream effects studied include improved mitochondrial electron transport chain efficiency, ATP production, and DNA repair capacity.

The decline, coenzyme signaling, and cellular cascade are studied together as one mechanism — a fundamental metabolic cofactor whose research administration aims to restore youthful cellular energy capacity.

3 What the Research Shows

NAD+ has a long biochemical research history alongside a newer wave of clinical trials on precursor supplementation and direct administration. Below is a plain-language summary of frequently cited findings.

Age-Related NAD+ Decline Massudi et al., PLOS ONE, 2012
Studies of human tissue reported NAD+ levels decline significantly with age, correlating with reduced sirtuin activity and establishing the foundational rationale for NAD+ research.

Mitochondrial Function Restoration Gomes et al., Cell, 2013
Preclinical research reported restoring NAD+ levels in aged mice reversed mitochondrial dysfunction within days, reported as a rapid and reversible age-related metabolic decline.

Human Safety & Tolerability Trials Airhart et al., PLOS ONE, 2017
A Phase 1 trial of intravenous NAD+ infusion in healthy adults reported it was safe and well-tolerated at studied doses, with transient infusion-related discomfort as the primary adverse event.

4 Effects Observed in Research

Across the studies above, three effects recur most often. These are findings from controlled research settings, not guarantees or claims about any individual outcome.

Improved Mitochondrial Function

Restored electron transport chain efficiency and ATP production reported in aged preclinical models.

DNA Repair & Cellular Resilience

Enhanced PARP-mediated DNA repair capacity studied as dependent on adequate NAD+ availability.

Sirtuin Activation & Metabolic Regulation

Restored sirtuin activity reported alongside replenished NAD+ pools, tied to cellular aging and metabolic regulation research.

5 Storage & Reconstitution Guide

NAD+ is supplied as a lyophilized (freeze-dried) powder that must be reconstituted before use in the lab. The steps below reflect standard handling practice — always defer to the certificate of analysis (COA) that ships with each vial.

BEFORE RECONSTITUTION

Keep the lyophilized vial refrigerated (2–8°C) for short-term storage, or frozen (–20°C) for longer-term storage, protected from light at all times. The unreconstituted powder is stable for the longest window — reconstitute only what will be used in the near term.

RECONSTITUTING WITH BACTERIOSTATIC WATER

Bring the vial to room temperature. Using a sterile syringe, draw bacteriostatic water and inject it slowly down the inside wall of the vial — not directly onto the powder — to avoid agitating and denaturing the compound. Gently swirl to dissolve; never shake.

RESULTING CONCENTRATION FOR A 1,000MG VIAL

Bacteriostatic Water Added	Resulting Concentration
5 mL	200 mg/mL
10 mL	100 mg/mL
15 mL	66.7 mg/mL
20 mL	50 mg/mL

AFTER RECONSTITUTION

Store the reconstituted solution refrigerated at 2–8°C and protected from light. Reconstituted NAD+ solutions are generally most stable when used within a few weeks — check the COA for the exact window for each lot. Avoid repeated freeze–thaw cycles, label vials with the reconstitution date, and discard any solution that appears cloudy, discolored, or has been left at room temperature for an extended period.

SOURCES

Massudi, H. et al. Age-associated changes in oxidative stress and NAD+ metabolism in human tissue · PLOS ONE, 2012 · Gomes, A.P. et al. Declining NAD+ induces a pseudohypoxic state disrupting nuclear-mitochondrial communication during aging · Cell, 2013 · Airhart, S.E. et al. An open-label, non-randomized study of the pharmacokinetics of the nutritional supplement nicotinamide adenine dinucleotide (NAD+) · PLOS ONE, 2017.