NAD+ 500 mg

Nicotinamide Adenine Dinucleotide

Cellular Energy and Repair Cofactor | Physician Reference

Overview

NAD+ (nicotinamide adenine dinucleotide) is an essential pyridine dinucleotide coenzyme present in every cell. It is not a peptide but a cofactor, and it is included here because it is frequently formulated and administered alongside peptide protocols. NAD+ is central both to energy metabolism, as a redox carrier, and to cellular repair signaling, as a substrate for regulatory enzymes.

Intracellular NAD+ declines with age and metabolic stress. Repletion strategies, whether by direct NAD+ administration or by precursor supplementation, aim to restore cofactor availability for mitochondrial function, DNA repair, and longevity-associated signaling. The clinical evidence base is uneven and many claimed benefits remain investigational.

Mechanism at a Glance

Attribute	Description
Molecular class	Pyridine dinucleotide coenzyme (not a peptide)
Redox role	Electron carrier across glycolysis, the TCA cycle, and oxidative phosphorylation (NAD+/NADH)
Signaling role	Substrate for sirtuins (SIRT1-7), PARPs, and CD38
Downstream effects	Mitochondrial biogenesis, DNA repair, circadian and metabolic regulation
Biosynthesis	Salvage pathway from nicotinamide, NMN, and NR precursors
Administration	Slow intravenous infusion or subcutaneous injection; oral precursors as an alternative
Regulatory status	Not FDA-approved as a therapeutic; sold as a supplement; not DEA-scheduled

Mechanism of Action

NAD+ (Nicotinamide Adenine Dinucleotide)

As a redox cofactor, NAD+ shuttles electrons between metabolic reactions, cycling between its oxidized (NAD+) and reduced (NADH) forms to support ATP generation. Adequate NAD+ availability is rate-relevant to mitochondrial energy production, which is the basis for its use in fatigue and metabolic decline.

As a signaling substrate, NAD+ is consumed by sirtuins, which deacylate target proteins to drive mitochondrial biogenesis, DNA-damage response, and circadian regulation, and by PARP enzymes involved in DNA repair. Because these enzymes consume NAD+, repletion is proposed to sustain repair and longevity signaling that would otherwise be constrained by falling cofactor levels.

Clinical Application Matrix

The table summarizes the role NAD+ repletion plays across common clinical goals. Primary reflects a core, mechanistically direct role; Supporting or Investigational reflect earlier-stage or less consistent human evidence.

Clinical Application	Role	Mechanistic Basis
Cellular energy and mitochondrial function	Primary	Redox cofactor for ATP-generating pathways
Longevity and repair signaling	Primary	Sirtuin and PARP substrate for DNA repair and biogenesis
Neurocognitive support	Supporting	Neuronal energetics and oxidative-stress modulation
Metabolic support	Supporting	Influence on glucose and lipid handling
Addiction and recovery adjunct	Investigational	Proposed neuro-restorative role; limited controlled data

Ideal Patient Candidate Profile

NAD+ repletion is most appropriate for consideration in patients presenting with one or more of the following:

- Persistent fatigue or low energy with a metabolic or age-related component
- Interest in age-associated decline of cellular repair and mitochondrial function
- Cognitive support goals as an adjunct to broader care
- Structured recovery protocols where cofactor repletion is a defined objective
- Tolerance for infusion-based administration and its rate-dependent side effects

Rapid administration commonly produces chest tightness, nausea, flushing, and cramping; slow titration of the infusion or injection is essential for tolerability. Human trial evidence supports some but not all marketed indications; claims should be framed accordingly.

Clinical and Compounding Context

- **Formulation** Compounded intravenous infusion (administered slowly) and subcutaneous injection are the primary parenteral routes; oral NMN or NR precursors are an alternative delivery strategy.
- **Quality assurance** Physician should verify USP compliance and sterility testing at the sourcing compounding pharmacy.
- **Informed consent** Recommended for all patients; documentation should reflect investigational or off-label status, the available level of human trial data, and an individual risk-benefit discussion.
- **Monitoring** No universally established clinical monitoring protocol; practitioner discretion based on patient profile and treatment goals.

- **Regulatory** Not FDA-approved as a therapeutic and not DEA-scheduled; NAD+ and its precursors are marketed as supplements, which carry quality, purity, and dosing variability.
- **Tolerability** Titrate infusion rate to limit flushing, nausea, and chest tightness; monitor the patient during administration.

Giovane Medical works with physicians to facilitate access to properly sourced, compounded peptide formulations and to support evidence-based protocol development.

For prescribing or physician partnership programs, contact the Giovane Medical clinical team.

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