thetrutides

NAD+

The cellular energy coenzyme — real biology, contested delivery

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NAD+ is not a peptide or a drug but a coenzyme every cell in your body already depends on. The underlying biology is textbook and beyond dispute. What is genuinely contested is whether administering it directly accomplishes anything.

In Plain English

NAD+ is the molecule your mitochondria use to convert food into usable energy, and it is also required for DNA repair. Levels drop as you age, which is well documented. The catch is that NAD+ is a fairly large, charged molecule, and there is real scientific debate about whether it can get into cells intact — or whether your body simply takes it apart and rebuilds it, in which case supplying the smaller building blocks might work just as well.

What people use it for. Energy, mental clarity, recovery and general anti-ageing. It is one of the most popular compounds in the longevity category.

What to know before you buy. The biology is real — NAD+ genuinely is essential to how your cells make energy, and levels genuinely do fall with age. The open question is whether injecting the finished molecule is the right way to address that, because NAD+ may be too large to enter cells intact and may simply be broken down and rebuilt from smaller pieces. Most of the better human research is on those smaller precursor molecules instead. Also: administer it slowly. Rapid administration commonly causes flushing, chest tightness and nausea, and that is a speed problem rather than a dose problem.

What It's Used For

GOAL	WHAT THAT RESTS ON
Cellular energy	NAD+ is essential to mitochondrial energy production — settled, textbook biology
DNA repair	Required by PARP enzymes for DNA repair and by sirtuins for their signalling roles
Replacing age-related decline	Tissue NAD+ falls substantially with age across multiple species
Honest caveat	Whether injected NAD+ raises levels inside cells is genuinely debated. Most stronger human research is on precursors

The left column lists goals people commonly pursue with this compound; the right column states what that rests on. These are described uses and research findings, not proven outcomes or treatment claims. Individual results vary and no outcome is promised.

Pricing & Sizes

PRICING

500mg vial (buffered)	$50
1000mg vial (buffered)	$80

Bulk pricing: 2-3 vials −5% | 4-6 vials −10% | 7-9 vials −15% | 10+ vials −20%

Ships sealed and lyophilized — stable for years at room temperature, reconstitute before use, or ask and it will be reconstituted free at the volume shown below. **Free shipping on every order, no minimum.** Third-party COA available for every batch — HPLC purity and mass spec verification.

Reconstitution — bacteriostatic water: 500mg vial at 2.5mL = 200mg/mL, so 10 units on a 100-unit syringe draws 20mg; **1000mg vial** at 5mL = 200mg/mL, so 10 units on a 100-unit syringe draws 20mg. Concentrations are arithmetic from vial content and diluent volume, not a dosing recommendation.

At a Glance

CLASS	Nicotinamide adenine dinucleotide — an essential coenzyme, not a drug
FUNCTION	Central to mitochondrial energy production, DNA repair via PARP enzymes, and sirtuin signalling
AGE DECLINE	Tissue NAD+ levels fall substantially with age, which is the basis for the entire supplementation category
THE OPEN QUESTION	Whether administered NAD+ enters cells intact, or is broken down and reassembled from precursors
EVIDENCE	Strong for the biology; limited and mixed for direct NAD+ administration in humans
REGULATORY (US)	Not FDA-approved as a drug. **Not** on the July 2026 PCAC agenda
FORMAT	500mg and 1000mg buffered vials

Essential COENZYME IN ALL CELLS	Sirtuins + PARP DOWNSTREAM PATHWAYS	Declines WITH AGE	Contested DELIVERY ROUTE

EVIDENCE STATUS

The importance of NAD+ in cellular metabolism is settled science. What is **not** settled is whether administering NAD+ directly raises intracellular NAD+ meaningfully, or whether the molecule is degraded extracellularly and reassembled from precursors such as nicotinamide riboside or nicotinamide mononucleotide. Most of the higher-quality human supplementation research concerns those precursors rather than NAD+ itself. Do not treat evidence for NR or NMN as evidence for injected NAD+. Not on the July 2026 PCAC agenda.

The Delivery Question

This is the crux, and it is worth understanding before spending money on any format. Cell membranes do not readily admit intact NAD+. The prevailing view in the research literature is that extracellular NAD+ is largely degraded to precursors, which are then transported in and resynthesised. If that is correct, the practical value of administering NAD+ itself over cheaper precursors is unclear.

Injectable and IV administration bypass gut degradation, which is a real advantage over oral NAD+ — oral NAD+ is broadly regarded as poorly bioavailable. But bypassing the gut does not resolve the cell membrane question, which is the more fundamental issue.

What the Research Supports

Well established

- NAD+ is essential to mitochondrial energy metabolism
- NAD+ is required by PARP enzymes for DNA repair and by sirtuins for their signalling functions
- Tissue NAD+ declines with age across multiple species

Not established

- That administered NAD+ meaningfully raises intracellular NAD+ in humans
- That raising NAD+ produces the anti-ageing outcomes commonly claimed for it
- That injected NAD+ outperforms oral precursors on any hard endpoint

Reported subjective effects during and after IV or injectable NAD+ — and the flushing, chest tightness and nausea that frequently accompany rapid administration — are widely described. Slow administration is the standard mitigation for those effects.

THE BOTTOM LINE

Unimpeachable underlying biology attached to an unresolved delivery question. The age-related decline is real and the pathways matter; whether injecting the finished coenzyme is the right way to address it remains genuinely open, and most of the better human research is on precursors instead.

Also in the Range

Compare with **5-Amino-1MQ**, which targets NAD+ from the other direction — blocking the enzyme that depletes it rather than supplying the molecule itself.

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