

This Small Molecule Reverses Alzheimer's Disease Progression, Study Shows

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STORY AT-A-GLANCE

- Nicotinamide adenine dinucleotide (NAD⁺) is central to cellular energy and mitochondrial health, driving redox reactions that produce ATP. Declining levels are linked to metabolic disorders, sarcopenia, and diabetes
- Alzheimer's disease has strongly associated with disrupted NAD⁺ balance, and research suggests restoring intake can reverse cognitive decline rather than merely slowing disease progression
- Animal studies show restoring NAD⁺ fully reversed advanced Alzheimer's features, including memory loss, inflammation, tau pathology, oxidative stress, and DNA damage, even after severe disease was established
- NAD⁺ functions as an upstream regulator of brain resilience, coordinating energy production, DNA repair, inflammation control, and protein processing, reframing Alzheimer's as a systems-level energy failure
- Practical strategies emphasize testing NAD⁺ status and supporting production safely via niacinamide alongside adequate intake of other B vitamins

Nicotinamide adenine dinucleotide (NAD⁺) may be one of the most overlooked factors when it comes to optimizing cellular health. It is a cellular coenzyme that plays a role in many metabolic and signaling reactions.

For example, it partakes in redox reactions — **chemical exchanges that transfer energy between molecules** — which lead to the production of adenosine triphosphate (ATP), your body's energy currency.¹ In fact, research shows that a deficiency is linked to an array of conditions, such as sarcopenia and diabetes.²

But that's not all — Alzheimer's disease, the most common form of dementia,³ has now been linked to declining NAD⁺ levels. Following this line of thought, emerging research shows that boosting NAD⁺ intake can reverse the progression of Alzheimer's disease. This discovery could be one of the biggest breakthroughs in recent times, as most people believe that Alzheimer's only worsens the longer it goes,⁴ and treatment focuses on slowing decline rather than reversing it.

Restoring Brain Energy Reversed Advanced Alzheimer's in Animal Models

A study published in Cell Reports Medicine set out to discover how Alzheimer's disease can be reversed by boosting NAD⁺ levels. For the experiment, the researchers used multiple mouse models of Alzheimer's disease that already showed severe cognitive impairment, brain inflammation, tau pathology, and structural brain damage.⁵

Mice were administered P7C3-A20 at a dosage of 10 milligrams (mg) per kilogram (kg) of weight each day. Analysis involved observing changes across behavior, brain chemistry, and physical brain structure. For context, P7C3-A20 is a carbazole compound that can readily cross the blood-brain barrier. It works by binding to NAMPT (an enzyme that controls how much NAD⁺ is made from niacinamide) to enhance NAD⁺ production⁶ at safe levels.

- **One striking finding is the rate of improvement** — The authors reported that in treated mice, cognitive function recovered fully, meaning their memory performance returned to levels seen in healthy animals. These mice performed just as well as non-diseased controls on learning and memory tests.

- **What changed inside the brain samples** — Multiple hallmarks of Alzheimer's disease improved at the same time. Tau pathology, which refers to tangled protein structures that disrupt neuron function, decreased after NAD⁺ restoration. Neuroinflammation markers dropped, indicating a calmer immune environment in the brain. Signals of oxidative stress and DNA damage — both signs of energy failure inside cells — also declined.
- **Results were observed right away** — The intervention occurred after the disease had fully developed in these animals. Again, this directly challenges the long-standing belief that Alzheimer's damage becomes permanent once it crosses a certain threshold.
- **Other disease models were used to solidify the findings** — The researchers tested the same approach in two different forms of Alzheimer's pathology. In amyloid-driven mice and in tau-driven PS19 mice, restoring NAD⁺ reversed advanced disease features. That distinction matters because amyloid and tau represent different biological drivers of Alzheimer's. Seeing improvement in both strengthens the argument that NAD⁺ disruption sits upstream of these visible brain lesions.
- **Blood biomarkers also benefited** — Treated animals showed reduced levels of phosphorylated tau 217, a biomarker now used clinically to track Alzheimer's severity. This helps bridge the gap between animal research and its implications for Alzheimer's disease in humans.
- **At the center of all the changes is NAD⁺ homeostasis** — NAD⁺ is required for cells to convert nutrients into usable energy and to repair daily damage to proteins and DNA. That said, the study found that Alzheimer's disease severity correlated with how disrupted NAD⁺ balance became in the brain. In other words, as energy systems failed, disease features worsened and restoring that balance reversed the cascade.

The researchers described this as a "resilience" model rather than a single-target approach. Instead of attacking amyloid alone or tau alone, restoring NAD⁺ stabilized multiple systems at once — energy production, inflammation control, blood-brain barrier integrity, and cellular repair. Thus, the findings reframe Alzheimer's as a system-level energy failure rather than a mystery buildup of toxic debris in the brain.

- **Human relevance strengthened the findings further** — Using human brain samples and sophisticated molecular analysis techniques, the authors reported that NAD⁺ disruption also tracked with Alzheimer's severity in people. They identified overlapping biological nodes between mice and humans that responded to restored NAD⁺ balance.
- **Mechanistic explanation of the benefits** — The paper explained that NAD⁺ acts as a central coordinator for enzymes involved in DNA repair, mitochondrial function, and stress resistance. When NAD⁺ levels fall, these systems stall. Neurons, which require constant energy, suffer first. Restoring NAD⁺ reactivated these pathways simultaneously.

The study also highlighted why focusing solely on plaques has delivered limited success. Amyloid and tau accumulation appeared downstream of NAD⁺ disruption rather than as isolated causes. Once energy systems failed, the brain lost its ability to manage protein turnover, immune balance, and structural integrity. Fixing the upstream energy deficit corrected multiple downstream failures at once.

From a practical standpoint, the findings support the idea that improving **cellular energy** changes the trajectory of Alzheimer's disease rather than simply slowing damage. It shows that neurons under metabolic stress can recover when you address cellular energy production at its root.

NAD⁺ Restores Memory by Rewriting Neuronal Instructions

In a related study published in Science Advances, researchers examined how restoring NAD⁺ reverses Alzheimer's features inside brain cells from a genetic perspective. Specifically, the researchers focused on gene regulation, which influences how neurons read and process instructions that control memory and brain resilience.⁷

- **Core findings of the analysis** — Increasing NAD⁺ corrected widespread errors in gene instruction processing and restored memory performance, but only when a specific control protein, EVA1C, remained intact. When this was suppressed, the memory benefit disappeared, even with NAD⁺ restoration.

Another important improvement that was observed is memory retention. Animals receiving NAD⁺ showed clear restoration of learning and recall ability, measured through standardized behavioral tests used in neuroscience research. When researchers interfered with EVA1C expression in the hippocampus, those gains vanished, even though NAD⁺ levels rose.

- **A deeper look into the mechanism at play** — The study showed that NAD⁺ corrected abnormal alternative splicing events across many genes. For context, alternative splicing refers to how cells assemble genetic instructions before building proteins.

Think of the process as editing a recipe. If the editing goes wrong, the cell produces dysfunctional proteins. In Alzheimer's models, these editing errors appeared widespread. NAD⁺ restored normal editing patterns, but only through EVA1C.

- **The largest benefits appeared in hippocampal neurons** — This is especially observed within the CA1 region. For context, the hippocampus is the brain's memory hub, and CA1 neurons act as a relay station for forming and retrieving memories. When EVA1C levels dropped in this region, NAD⁺ no longer improved memory performance.
- **Comparisons between test variables** — NAD⁺ alone improved memory only when EVA1C function remained intact. Meanwhile, EVA1C suppression alone worsened memory outcomes even when energy levels improved. This shows that NAD⁺ and

EVA1C did not work independently — they functioned as a linked system, with EVA1C acting as the gatekeeper for the cognitive benefits of NAD⁺.

The study also included human data. Researchers reported that EVA1C expression was reduced in the hippocampus of participants with Alzheimer's disease compared to cognitively normal controls.

- **A closer analysis of the mechanisms involved** — Ribonucleic acid (RNA) splicing determines which protein versions neurons produce. In Alzheimer's disease, incorrect splicing led to dysfunctional proteins that weaken synapses and disrupt communication between brain cells. Now, NAD⁺ restored normal splicing patterns by regulating EVA1C activity, which stabilized protein production inside neurons.

Again, the researchers emphasized that this process represented a form of resilience. Neurons did not simply slow deterioration — they regained the ability to produce functional proteins required for learning and memory.

Before Boosting Levels, It's Important to Get a Baseline

Based on the findings, boosting NAD⁺ has enormous potential when it comes to managing Alzheimer's disease. Hence, testing your current levels is important, as it would be wise not to take any supplement without proper direction or planning.

- **A new test will be launched in the future** — I'm excited to introduce the upcoming Mitochondrial Wellness Test Kit, which is designed to offer you a current snapshot of your mitochondrial function. While this provides an overview, additional targeted testing may still be needed to fully understand the more intricate nuances of your health.
- **Existing NAD⁺ tests fall short** — NAD⁺ is highly unstable once it's outside the cells and degrades quickly, making reliable measurement difficult. To maintain accuracy, it requires immediate processing and advanced laboratory methods.

In practice, this means blood samples need to be collected and analyzed rapidly within the same research facility, which is not possible at most clinics. Moreover, transporting samples between labs further compromises integrity. Despite these obstacles, my team and I have remained committed to advancing practical health testing for everyone.

- **A higher standard for NAD⁺ assessment** — Mercola Labs is developing a novel solution that avoids the pitfalls of measuring NAD⁺. Instead, we assess NAD⁺ levels by analyzing redox balance among these essential biomarkers — acetoacetate and beta-hydroxybutyrate, lactate and pyruvate, and the oxidized and reduced forms of glutathione. Additional details will be shared closer to release.

Niacinamide Supports NAD⁺ Production

Taking niacinamide is a convenient way of boosting your NAD⁺ levels. However, this approach calls for precision and balance — the reason why I encourage proper testing. While high doses have shown benefits in clinical settings, smaller and consistent amounts are far more appropriate for everyday use. This approach supports mitochondrial and metabolic function without placing unnecessary stress on the body, since excessive intake can disrupt methylation pathways and raise the risk of adverse events over time.

- **Take small, evenly distributed daily doses** — For daily support, take 50 milligrams of niacinamide three times per day. This modest dose supports NAD⁺ production without the risks associated with high-dose vitamin B3 supplementation. You can even divide it into four servings per day. Take one dose upon waking, one before bed, and space the remaining doses evenly throughout the day.
- **Excessive B3 intake can be counterproductive** — Taking too much vitamin B3, whether as niacin or niacinamide, will lead to negative outcomes. Research cited by the Cleveland Clinic indicates that high doses can increase cardiovascular risk.⁸ Although both compounds are forms of vitamin B3, niacin does not activate NAMPT the way niacinamide does, making niacinamide the preferred option.

- **Don't forget the other B vitamins** — Adequate intake of other B vitamins is essential for overall health and mitochondrial function, particularly niacin, riboflavin, and folate. Suboptimal mitochondrial health is often linked to B-vitamin deficiencies,⁹ which can typically be corrected with a low-dose, high-quality B-complex supplement.

When it comes to food sources, vitamin B3 is abundant in grass fed beef and mushrooms.¹⁰ Vitamin B6 is found in grass fed beef, potatoes, and bananas.¹¹ Folate (vitamin B9) is plentiful in spinach, broccoli, and asparagus,¹² while vitamin B12 is concentrated in foods such as grass fed beef liver, wild rainbow trout, and wild sockeye salmon.

Frequently Asked Questions (FAQs) About NAD⁺ and Its Link to Alzheimer's Disease

Q: What is NAD⁺ and why is it essential for cellular and brain health?

A: NAD⁺ is a core cellular coenzyme required for energy production, mitochondrial function, DNA repair, and metabolic signaling. Low NAD⁺ levels impair cellular energy and are linked to aging, metabolic disease, and neurodegeneration.

Q: How is NAD⁺ connected to Alzheimer's disease progression?

A: Research shows Alzheimer's disease severity correlates with disrupted NAD⁺ balance. Declining NAD⁺ levels impairs neuronal energy, repair, and resilience, suggesting the condition is driven by upstream energy failure rather than plaque buildup alone.

Q: Can restoring NAD⁺ reverse Alzheimer's-related damage?

A: In advanced animal models, restoring NAD⁺ led to full cognitive recovery, reduced inflammation, improved tau pathology, and lower blood biomarkers, even after severe disease was established, challenging the idea of irreversible damage.

Q: How does NAD⁺ improve memory at a genetic and cellular level?

A: NAD⁺ restores proper gene instruction processing through EVA1C-dependent RNA splicing, particularly in hippocampal neurons. This allows neurons to rebuild functional proteins required for learning and memory, promoting true neuronal recovery.

Q: What is the safest way to support NAD⁺ levels?

A: Modest, consistent niacinamide dosing, combined with adequate B vitamins, supports NAD⁺ production safely without disrupting methylation or increasing health risks.

Sources and References

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