Article

Nicotinamide Adenine Dinucleotide (NAD+): Molecular mechanisms, clinical translation, and industrial application

WenJun Zhang
School of Life Sciences, Sun Yat-sen University, Guangzhou, China
E-mail: zhwj@mail.sysu.edu.cn, wjzhang@iaees.org

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Abstract
Nicotinamide adenine dinucleotide (NAD+) is one of the most functionally core small molecules in all living organisms. Early studies have confirmed that it acts as an electron carrier in redox reactions, and participates in core energy metabolism processes including glycolysis, tricarboxylic acid cycle, and fatty acid oxidation, thus serving as a key molecule for maintaining cellular energy homeostasis. In recent years, in-depth studies have further revealed that NAD+ is also widely involved in multiple critical biological processes such as DNA damage repair, protein translational modification, cellular stress response, and aging signaling pathway regulation, acting as a core regulatory node that connects cellular metabolism, organismal aging and disease pathogenesis. A large number of epidemiological and basic studies have confirmed that NAD+ levels in various mammalian tissues decrease progressively with age, and this reduction has a clear causal association with the occurrence and development of a variety of aging-related diseases, including metabolic diseases, neurodegenerative diseases, cardiovascular dysfunction, and tumors. Boosting cellular NAD+ levels via supplementation with NAD+ and its precursors has become a research hotspot in gerontology and translational medicine. This review systematically summarizes the research progress and current status of the NAD+ field from three dimensions: molecular mechanisms, clinical translation and industrial applications, and sorts out the existing controversies and unresolved problems in current research, aiming to provide a comprehensive reference framework for basic research, clinical drug development and health industry application related to NAD+.

Keywords Nicotinamide Adenine Dinucleotide (NAD+); NAD+ precursors; aging-related diseases; clinical translation; industrial application.

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1 Introduction

1.1 Historical Background and Biological Significance

Nicotinamide adenine dinucleotide (NAD+) was first identified as an essential cofactor for fermentation over 110 years ago, and for decades, research focused exclusively on its canonical role as an electron carrier in core

cellular metabolic pathways (Chen et al., 2019; Zheng et al., 2023). In recent decades, modern research has expanded our understanding of NAD+, revealing it is a master regulator of multiple cellular signaling pathways, positioned at the intersection of aging biology and chronic disease development (Li et al., 2024; Chen et al., 2025). Multiple preclinical and clinical studies consistently confirm that cellular NAD+ levels decline progressively with age across all mammalian tissues, and this depletion is causally linked to the onset and progression of most aging-related diseases, including metabolic disorders, neurodegeneration, cardiovascular dysfunction, and cancer (Huang and Zhang, 2012; Li and Zhang, 2013; Zhang 2016, 2018; Li et al., 2024; Chen et al., 2025; Zhang et al., 2025). As a result, strategies to augment cellular NAD+ levels have become one of the most actively investigated areas in geroscience and translational medicine, attracting widespread attention from the scientific community, industry, and consumers (Damgaard and Treebak, 2023; Hebe, 2023; Yi, 2023).

This comprehensive review systematically synthesizes current progress in NAD+ research, drawing on peer-reviewed published studies and open-access resources to ensure accuracy. It covers molecular mechanisms of NAD+ homeostasis regulation, disease-related research advances, completed and ongoing clinical trials, industrialization and consumer market status, and future challenges and perspectives.

1.2 Objectives and Scope

The core objectives of this review are: (1) clarify the molecular basis of NAD+ biosynthesis and degradation homeostasis; (2) summarize the biological functions of NAD+ and its role in different types of diseases; (3) objectively evaluate results from existing human clinical trials of NAD+ augmentation strategies; (4) analyze the current status of industrialization and consumer market of NAD+-related products; and (5) propose key directions for future research and regulation (Zheng et al., 2023; Shi, 2025).

2 Molecular Mechanisms and Regulation of NAD+ Homeostasis

2.1 Biosynthesis Pathways of NAD+ in Mammalian Cells

Mammalian cells maintain stable NAD+ levels through three interconnected biosynthesis pathways, with contributions varying by cell type and nutrient availability (Sun et al., 2021; Zheng et al., 2023; Fig. 1).

(1) **De novo biosynthesis**: This pathway starts from dietary tryptophan, which is processed through multiple enzymatic steps to form nicotinic acid mononucleotide (NAMN), then converted to nicotinic acid adenine dinucleotide (NAAD), and finally amidated to generate mature NAD+. It contributes approximately 10-15% of total NAD+ production in most somatic tissues, with highest activity in the liver (Li et al., 2024).

(2) **Preiss-Handler pathway**: This pathway uses dietary nicotinic acid (a form of vitamin B3) as a substrate, which is converted to NAMN by nicotinic acid phosphoribosyltransferase, then follows the same downstream steps as the de novo pathway to produce NAD+ (Sun et al., 2021; Li et al., 2024).

(3) **Salvage pathway**: This is the dominant pathway for NAD+ maintenance in adult mammals, contributing more than 80% of total NAD+ production. It recycles nicotinamide (NAM), the byproduct of NAD+ consumption by signaling enzymes, to regenerate NAD+. The rate-limiting step is conversion of NAM to nicotinamide mononucleotide (NMN) by nicotinamide phosphoribosyltransferase (NAMPT). NMN is then converted to NAD+ by NMN adenylyltransferases (NMNATs). Another common precursor, nicotinamide riboside (NR), is also processed through this pathway, converted to NMN by NR kinases before final synthesis of NAD+ (Dang and Cai, 2022; Shi, 2025).

The salvage pathway is particularly critical because most NAD+ consumed in signaling reactions is recycled through this route. Multiple studies confirm that NAMPT activity declines with age, driven by

chronic inflammation and accumulated DNA damage, leading to reduced NAD+ production and net age-related NAD+ depletion (Zheng et al., 2023; Li et al., 2024; Zhang et al., 2025).

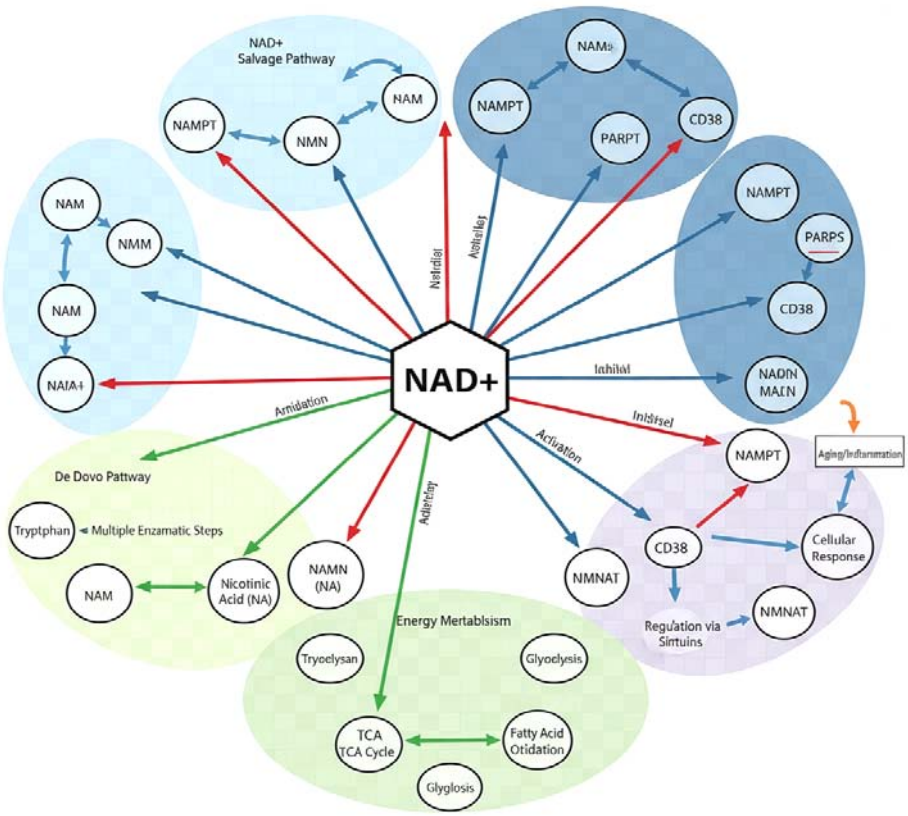

Fig. 1 A simplified signaling pathway of NAD+ metabolism and regulation.

2.2 Degradation and Homeostasis Regulation

NAD+ is consumed as a co-substrate by three major classes of regulatory enzymes in mammalian cells, which all cleave NAD+ to release NAM and ADP-ribose derivatives for downstream signaling (Chen et al., 2019; Wang et al., 2020; Fig. 1):

(1) **Poly (ADP-ribose) polymerases (PARPs)**: PARPs are key mediators of DNA damage repair. Accumulated DNA damage with age leads to persistent PARP activation, which increases NAD+ consumption significantly (Wang et al., 2020; Zheng et al., 2023).

(2) **Sirtuin (SIRT) deacetylases**: Sirtuins are NAD+-dependent enzymes that regulate mitochondrial function, metabolism, stress response, and aging. For example, mitochondrial SIRT3, a key regulator of cardiac function, relies entirely on available NAD+ to exert its biological activity (Sun, 2025).

(3) **CD38/CD157**: CD38 is the major NAD+ hydrolase in most mammalian tissues, expressed highly on immune and senescent cells. Aging-related accumulation of senescent cells and chronic inflammation upregulates CD38 activity, which is now recognized as one of the main drivers of age-related NAD+ decline (Zheng et al., 2023; Li et al., 2024).

Under normal physiological conditions, intracellular NAD+ concentrations are maintained between 100-400 μM (Sun et al., 2021; Zheng et al., 2023). Aging, obesity, chronic inflammation, and DNA damage

disrupt this balance, leading to NAD+ depletion that drives disease development (Wang et al.., 2020; Li et al., 2024).

3 Disease-Relevant Research Advances of NAD+

3.1 Aging and Age-Related Metabolic Diseases

Progressive NAD+ decline is a universal hallmark of mammalian aging (Li et al., 2024; Zhang et al., 2025). Preclinical studies in aging mouse models consistently show that supplementation with NAD+ precursors (NMN, NR) increases tissue NAD+ levels, improves metabolic health, extends median lifespan, and reduces age-related functional decline (Shi, 2025; Sun, 2025).

In metabolic diseases such as type 2 diabetes (T2D) and obesity, multiple studies confirm that NAD+ levels are significantly reduced in adipose tissue, liver, and skeletal muscle of patients and animal models (Shi, 2025). NAD+ depletion impairs insulin signaling, reduces mitochondrial function, and increases oxidative stress, which exacerbates insulin resistance and hyperglycemia. NMN supplementation improves insulin sensitivity, reduces hepatic steatosis, and restores glucose homeostasis in obese mouse models, via activation of SIRT1 and SIRT3, improved mitochondrial function, and reduced inflammation (Shi, 2025; Sun, 2025).

Diabetic cardiomyopathy (DCM), a common severe complication of T2D, is strongly linked to NAD+ depletion. Studies show that NAD+ levels and SIRT3 activity are significantly reduced in the myocardium of DCM patients. SIRT3, an NAD+-dependent mitochondrial deacetylase, regulates mitochondrial respiration, reactive oxygen species scavenging, and cardiomyocyte survival. Reduced SIRT3 activity due to NAD+ depletion drives pathological cardiac remodeling and heart failure in DCM. Preclinical studies confirm that increasing NAD+ levels restores SIRT3 activity and improves cardiac function in DCM models, providing a strong theoretical basis for clinical translation (Sun, 2025).

3.2 Neurodegenerative Diseases

NAD+ depletion is detected in multiple common neurodegenerative diseases including Alzheimer’s disease (AD), Parkinson’s disease (PD), and cerebral ischemia, and NAD+ exerts neuroprotective effects via multiple mechanisms: improving mitochondrial function, reducing oxidative stress, inhibiting neuronal apoptosis, promoting DNA repair, and regulating neuroinflammation (Chen et al., 2019; Chen et al., 2024).

In AD, amyloid-beta accumulation and tau hyperphosphorylation increase DNA damage and neuroinflammation, leading to PARP and CD38 activation and subsequent NAD+ depletion, which exacerbates neuronal damage. Preclinical studies in AD mouse models show that NMN supplementation reduces amyloid-beta plaque load, improves cognitive function, and reduces neuronal loss (Chen et al;., 2019; Zheng et al., 2023). In PD, alpha-synuclein aggregation causes mitochondrial dysfunction and NAD+ depletion, and NAD+ augmentation protects dopaminergic neurons from degeneration. The reduced form of NAD+, NADH, has also shown therapeutic potential for neurodegenerative diseases characterized by mitochondrial dysfunction, improving mitochondrial respiration and reducing oxidative damage (Chen et al., 2024). In acute ischemic stroke, NAD+ levels drop rapidly in the ischemic penumbra, and exogenous NAD+ supplementation reduces infarct volume and improves neurological outcomes in animal models (Chen et al., 2019). Multiple studies confirm that NAD+ is a promising therapeutic target for neurodegenerative diseases and acute brain injury (Chen et al., 2019; Chen et al., 2024).

3.3 Cardiovascular Diseases

Aging is the primary risk factor for cardiovascular disease, and age-related NAD+ decline contributes to pathologies including hypertension, atherosclerosis, vascular dysfunction, and heart failure (Zheng et al., 2023; Sun, 2025). NAD+ augmentation improves endothelial function, reduces vascular stiffness, reduces atherosclerotic plaque formation, and improves cardiac function in preclinical models. For example, NMN

supplementation improves endothelium-dependent vasodilation and reduces blood pressure in aging mice, via SIRT1 activation and increased nitric oxide production in endothelial cells (Zheng et al., 2023).

3.4 Cancer

NAD+ metabolism plays a dual role in cancer: cancer cells require high levels of NAD+ to support rapid proliferation, nucleotide biosynthesis, and DNA repair, so most cancers undergo NAD+ metabolic remodeling to maintain high intracellular NAD+ levels. On the other hand, NAD+ augmentation improves DNA repair and immune function in normal cells, which may reduce cancer incidence in aging populations (Zhang et al., 2015; Che et al., 2025).

Glioblastoma multiforme (GBM), the most aggressive primary malignant brain tumor in adults, provides a well-studied example of NAD+ metabolic remodeling. GBM relies on dysregulated NAD+ metabolism to maintain high intracellular NAD+ levels to support its high proliferation, invasion, and therapeutic resistance. Key NAD+ biosynthesis enzymes such as NAMPT are significantly upregulated in GBM cells, which increases NAD+ production and correlates with poor patient prognosis. Targeting NAD+ metabolism (e.g., NAMPT inhibition to deplete tumor NAD+) has shown significant anti-tumor activity in preclinical GBM models, and multiple clinical trials of this strategy are ongoing (Che et al., 2025). NAD+ depletion also sensitizes cancer cells to chemotherapy and radiotherapy by inhibiting DNA repair, so combining NAD+ targeting with traditional cancer therapy improves treatment efficacy (Zhang et al., 2015; Che et al., 2025).

3.5 Other Chronic Diseases

NAD+ has also been implicated in the pathogenesis of kidney disease, chronic obstructive pulmonary disease, and osteoporosis. Multiple preclinical studies show that NAD+ levels are significantly reduced in renal tissue in both acute kidney injury and chronic kidney disease, and NAD+ augmentation reduces renal damage and improves renal function. Currently, multiple clinical trials of NAD+ precursors for kidney disease are registered in public databases (Yang et al., 2018).

4 Clinical Trials of NAD+ Augmentation Strategies

As of 2026, dozens of clinical trials of NAD+ precursors (mainly NMN and NR) have been completed, with many more ongoing, investigating safety, pharmacokinetics, and therapeutic efficacy for multiple indications (Damgaard and Treebak, 2023; Hebe, 2023; Yang et al., 2018).

4.1 Safety and Pharmacokinetics

Multiple completed trials consistently show that oral NMN and NR at doses up to 1200-2000 mg per day are generally safe and well-tolerated in healthy adults, with no serious adverse events reported in short-term (up to 12 weeks) trials (Dang and Cai, 2022; Hebe, 2023; Shi, 2025). For example, a phase 1 trial of single oral NMN doses (100-1200 mg) in 10 healthy men found NMN was rapidly absorbed, significantly increased circulating NAD+ levels, and caused no adverse effects (Dang and Cai, 2022). Long-term safety data (over 1 year of supplementation) remains limited, but no major adverse events have been reported in existing long-term follow-up studies (Zheng et al., 2023; Shi, 2025).

Most studies confirm that NMN has higher bioavailability than NR, as it is one step closer to NAD+ in the biosynthesis pathway and does not require extra phosphorylation by NR kinases (Shi, 2025). However, there is significant inter-individual variability in response to supplementation, related to differences in baseline NAD+ levels, age, and body composition (Hebe, 2023; Li et al., 2024).

4.2 Clinical Efficacy

The clinical efficacy of NAD+ precursor supplementation remains controversial, with inconsistent results across trials (Damgaard and Treebak, 2023; Hebe, 2023).

A 2023 systematic review that included 25 completed clinical trials of NR supplementation found that oral

NR produced only minimal increases in circulating NAD+ levels in most populations, and did not produce any clinically significant improvements in metabolic health, physical performance, or other measured outcomes (Damgaard and Treebak, 2023; Yi, 2023). This analysis sparked widespread debate about the efficacy of NAD+ precursors and questioned exaggerated marketing claims from commercial brands (Damgaard and Treebak, 2023; Yi, 2023).

However, systematic reviews of NMN trials have found that NMN produces significantly greater increases in circulating and tissue NAD+ levels than NR, with some studies reporting up to a 6-fold increase in blood NAD+ levels after 8-12 weeks of supplementation (Hebe, 2023; Li et al., 2024). Clinical trials of NMN have shown that 1000-1200 mg per day for 10-12 weeks improves insulin sensitivity in overweight prediabetic women, improves endothelial function and vascular compliance in middle-aged and older adults, and improves physical performance in older adults with mild impairment (Hebe, 2023; Zheng et al., 2023; Shi, 2025). Small clinical trials of NADH for neurodegenerative diseases show it improves cognitive function, mood, and motor symptoms in AD and PD patients, consistent with preclinical findings (Zheng et al., 2023; Chen et al., 2024).

As of 2026, more than 14 clinical trials of NMN and NR are ongoing, investigating efficacy for indications including metabolic dysfunction, aging-related cognitive decline, cardiovascular disease, and kidney disease (Yang et al., 2018). Results from these large, long-term randomized controlled trials (RCTs) will provide definitive evidence about the clinical efficacy of NAD+ augmentation (Hebe, 2023; Zheng et al., 2023).

5 Industrialization and Consumer Market Status

NAD+ precursor supplements (mainly NMN and NR) have become one of the fastest-growing categories in the global anti-aging health product market over the past decade (Damgaard and Treebak, 2023; Yi, 2023; Zhang et al., 2025).

5.1 Industrialization Development

Early commercialization focused on NR, which was first launched as a dietary supplement around 2015, gaining widespread attention after high-profile investment from business leaders including KS Li (Yi, 2023). In the past 5 years, NMN has become the dominant product in the market, due to its higher bioavailability and stronger evidence of NAD+ elevation in clinical trials (Shi, 2025).

Early large-scale production of NMN was very expensive, but advances in biocatalytic production technology have significantly reduced production costs, making NMN affordable for mass consumers (Zhang et al., 2025). Currently, companies have developed multiple advanced formulations to improve bioavailability, including liposomal NMN, enteric-coated NMN, and sublingual NMN, which claim to increase absorption and reduce gastric degradation (Damgaard and Treebak, 2023; Yi, 2023; Shi, 2025).

Regulation of NMN varies by region: in the United States, NMN was reclassified as a legal dietary supplement in 2023, allowing over-the-counter sales; in China, NMN was approved as a new food ingredient in 2023, permitting legal consumer sales (Yi, 2023; Zhang et al., 2025).

5.2 Current Market Status and Controversies

The global market for NAD+ precursor supplements was valued at over 500 million in 2025, and is projected to exceed 500 million in 2025, and is projected to exceed 500 million in 2025,and isprojectedto exceed 3 billion by 2030, driven by growing consumer demand for anti-aging and preventive health products (Yi, 2023; Zhang et al., 2025). However, the market faces significant controversies and quality issues (Yi, 2023).

On one hand, preclinical and early clinical evidence supports the potential benefits of NAD+ augmentation for metabolic and age-related health, so millions of consumers use NMN supplements to improve energy, slow aging, and prevent chronic disease (Hebe, 2023; Shi, 2025). On the other hand, many commercial brands make exaggerated unproven claims, advertising NMN as a "cure for aging" or "elixir of life", leading to criticism

that many products are overpriced and benefits are overstated. The 2023 analysis of 25 NR trials that found no clinically significant benefits reinforced this criticism, with some commentators describing commercial NAD+ supplements as a consumer scam (Yi, 2023).

Another major issue is inconsistent product quality: many low-quality products contain less active ingredient than labeled, or contain harmful impurities, posing potential health risks to consumers. Long-term safety of lifelong supplementation also remains unconfirmed (Zheng et al., 2023; Shi, 2025).

6 Future Perspectives and Challenges

NAD+ research has advanced dramatically over the past two decades, moving from basic discovery of its role in aging to early clinical translation and commercialization, but multiple key challenges remain to be addressed (Zheng et al., 2023; Shi, 2025).

6.1 Basic Research Challenges

First, more human data is needed to clarify tissue-specific regulation of NAD+ homeostasis, and the relative contribution of different mechanisms (NAMPT decline, increased CD38 activity, increased PARP activation) to age-related NAD+ decline; most current data comes from mouse models, and needs confirmation in humans (Li et al., 2024; Zhang et al., 2025). Second, the long-term effect of NAD+ augmentation on cancer risk remains unclear: while NAD+ augmentation reduces genomic instability and inflammation in normal cells (which may reduce cancer risk), there is concern that high NAD+ levels could support growth of pre-existing microscopic tumors. Long-term observational studies are needed to address this question (Che et al., 2025; Zhang et al., 2015). Third, more effective NAD+ augmentation strategies need to be developed, such as CD38 inhibitors that reduce NAD+ consumption, which may be more effective than precursor supplementation for older individuals with high CD38 activity (Zheng et al., 2023).

6.2 Clinical Research Challenges

The most pressing need is large, long-term, randomized placebo-controlled trials to confirm clinical efficacy of NAD+ precursor supplementation for different indications, and assess long-term safety (Hebe, 2023; Damgaard and Treebak, 2023; Shi, 2025). Most existing trials are small, short-term, and underpowered to detect clinically significant outcomes. Future trials should also compare efficacy of different precursors, doses, and formulations to identify optimal strategies for different populations (Hebe, 2023; Shi, 2025). For cancer, more clinical trials are needed to test the efficacy of targeted NAD+ metabolism inhibition, especially in combination with chemotherapy, radiotherapy, and immunotherapy (Zhang et al., 2015; Che et al., 2025).

6.3 Market and Regulation Challenges

The rapid growth of the consumer market requires stricter regulation to protect consumers (Yi, 2023). Regulators need to establish clear quality standards for NMN and NR products, require accurate labeling, and crack down on false and exaggerated marketing. Public health education is also needed to help consumers understand the current state of evidence: NAD+ augmentation has shown promising preclinical and early clinical results, but many claimed benefits are not yet confirmed by definitive RCTs (Damgaard and Treebak, 2023; Yi, 2023).

7 Conclusion

NAD+ is a fundamental cellular molecule that plays core roles in energy metabolism, signaling regulation, aging, and disease. Age-related NAD+ decline is a key driver of multiple aging-related chronic diseases, and NAD+ augmentation has shown significant beneficial effects in preclinical models of most age-related diseases (Zheng et al., 2023; Li et al., 2024). Early clinical trials confirm that short-term NAD+ precursor supplementation is safe and effectively increases circulating NAD+ levels, with promising early evidence of

benefits for metabolic health, cardiovascular function, and neurodegenerative diseases (Hebe, 2023; Shi, 2025). However, large long-term RCTs are still needed to confirm long-term safety and clinical efficacy, and the consumer market requires stricter regulation to address exaggerated marketing and product quality issues (Damgaard and Treebak, 2023). Future NAD+ research is expected to deliver new therapeutic strategies for aging-related diseases and promote healthy aging globally (Zheng et al., 2023; Zhang et al., 2025).

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