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NAD+ Biology and Multi-Component Nutraceutical Support for Cellular Energy, Healthy Ageing, Cognition, Cardiovascular Wellness, Stress Adaptation and Fatigue

A Critical Review with Reference to the Biotex NAD+ Formulation

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Abstract: Nicotinamide adenine dinucleotide (NAD+) is a central redox cofactor and signalling substrate that links nutrient oxidation, mitochondrial electron transfer, DNA-damage responses, sirtuin activity and cellular stress adaptation. Interest in NAD+-supportive nutraceuticals has increased in parallel with human trials of nicotinamide riboside and nicotinamide mononucleotide and with preclinical work connecting NAD homeostasis to mitochondrial function and ageing. This critical review evaluates the biological and clinical evidence relevant to a multi-component nutraceutical, Biotex NAD+, containing Terminalia arjuna extract (150 mg), coenzyme Q10/ubiquinol-related material (100 mg), Withania somnifera extract (100 mg), adenosine 5'-monophosphate (50 mg), nicotinamide (14 mg) and riboflavin (2 mg) per capsule. Only peer-reviewed primary research reports were used as numbered evidentiary citations; review articles, systematic reviews, meta-analyses, editorials, protocols and preprints were excluded from the evidence set. Human randomised or controlled studies were prioritised, with mechanistic animal or cellular studies used when clinical data were unavailable. The strongest ingredient-level human support concerns CoQ10/ubiquinol for mitochondrial and fatigue-related outcomes, Withania somnifera for stress adaptation and selected cognitive outcomes, and T. arjuna for cardiovascular functional endpoints. Human NAD-precursor trials confirm that NAD metabolism is modifiable, but recent direct comparison shows sustained circulatory NAD+ elevation with nicotinamide riboside and nicotinamide mononucleotide rather than nicotinamide; therefore, the 14-mg nicotinamide content of the formulation should not be interpreted as proof that the finished product raises systemic NAD+. Evidence for healthy ageing is strongest at the mechanistic and preclinical levels, while human lifespan extension, cellular 'rejuvenation', direct ATP elevation and product-specific cognitive or cardiovascular efficacy remain unproven. Overall, the formulation has a coherent multi-pathway rationale for supporting energy metabolism, fatigue resistance, stress adaptation, cognitive wellness and cardiovascular function, but formulation-specific randomised trials are required to establish

magnitude, reproducibility and dose relevance of these effects.

Keywords: NAD+; nicotinamide; coenzyme Q10; ubiquinol; Withania somnifera; Terminalia arjuna; adenosine 5'-monophosphate; riboflavin; mitochondrial bioenergetics; fatigue; cognition; stress adaptation; cardiovascular wellness; healthy ageing

Abbreviations

Abbreviation	Definition
AMP	Adenosine 5'-monophosphate
AMPK	AMP-activated protein kinase
ATP	Adenosine triphosphate
CoQ10	Coenzyme Q10
FAD	Flavin adenine dinucleotide
FMN	Flavin mononucleotide
HPA	Hypothalamic-pituitary-adrenal
HPMC	Hydroxypropyl methylcellulose
LVEF	Left ventricular ejection fraction
NAD+	Oxidised nicotinamide adenine dinucleotide
NADH	Reduced nicotinamide adenine dinucleotide
Nam	Nicotinamide
NMN	Nicotinamide mononucleotide
NR	Nicotinamide riboside
PARP	Poly(ADP-ribose) polymerase
SIRT	Sirtuin

1. Introduction

Nicotinamide adenine dinucleotide exists in oxidised (NAD+) and reduced (NADH) states and occupies a pivotal position in intermediary metabolism. NAD+-dependent redox reactions permit electron transfer during glycolysis, pyruvate oxidation, beta-oxidation and the tricarboxylic acid cycle, while NADH supplies reducing equivalents to mitochondrial complex I. NAD+ also functions as a consumed substrate for sirtuins, poly(ADP-ribose) polymerases and NADases, creating a direct interface between energy metabolism, stress signalling and cellular maintenance. Human supplementation studies have established that NAD metabolism can be modified nutritionally, although the magnitude and physiological consequences depend strongly on the precursor employed[1-4].

This distinction is central when evaluating products marketed within the NAD+ wellness category. In a 2026 randomised human comparison, 14 days of nicotinamide riboside (NR) or nicotinamide mononucleotide (NMN), but not nicotinamide (Nam), produced sustained increases in circulating NAD+; nicotinamide instead generated a more acute and transient change in the whole-blood NAD metabolome[1]. Earlier controlled studies similarly showed substantial NAD-metabolome responses to NR or NMN at pharmacological supplementation doses[2-7]. Consequently, evidence derived from NR or NMN cannot be transferred uncritically to a formulation containing nutritional-dose nicotinamide.

At the same time, NAD+ biology is broader than precursor pharmacokinetics. Age-related perturbation of NAD homeostasis has been linked experimentally to impaired nuclear-mitochondrial communication, increased CD38-dependent NAD consumption, mitochondrial dysfunction, senescence-associated inflammation and altered stem-cell function[11-16]. These observations provide a mechanistic basis for healthy-ageing research, but they do not demonstrate that an orally consumed multi-ingredient supplement extends human lifespan. Indeed, a primary mouse study found that chronic nicotinamide

favourably affected several age-related physiological measures without increasing overall survival duration[16].

The Biotex NAD+ formulation combines nicotinamide with CoQ10, riboflavin, adenosine 5'-monophosphate (AMP), Withania somnifera and Terminalia arjuna. This design is more appropriately interpreted as a multi-pathway formulation targeting normal energy metabolism, redox support, stress adaptation and cardiovascular wellness than as a direct pharmacological NAD+-replacement intervention. The aim of this review is therefore to evaluate the evidence claim by claim, distinguish mechanistic plausibility from human efficacy, and identify where the product dose is or is not comparable with the interventions tested in primary academic studies.

2. Literature Search and Evidence-Selection Strategy

This manuscript was developed as a critical narrative review. Candidate literature was identified through targeted searches of PubMed and primary publisher platforms, including Nature Portfolio, AAAS/Science, Cell Press/Elsevier, ScienceDirect, Springer Nature, JACC, Diabetes Care, Sleep Medicine and other peer-reviewed academic sources. The search covered literature available through August 2026 and used combinations of NAD+, nicotinamide, nicotinamide riboside, nicotinamide mononucleotide, coenzyme Q10, ubiquinol, Withania somnifera, ashwagandha, Terminalia arjuna, AMP, riboflavin, mitochondrial bioenergetics, fatigue, cognition, attention, stress, cortisol, endothelial function, cardiac function, ageing, CD38, sirtuin and senescence.

The numbered evidence base was deliberately restricted to primary research publications. Human randomised controlled trials, placebo-controlled trials, crossover trials and controlled clinical studies were prioritised. Primary mechanistic studies in animals or cells were retained only where necessary to explain NAD+ biology or healthy-ageing pathways not yet established in humans. Review articles, systematic reviews, meta-analyses, editorials, commentaries, conference abstracts, trial protocols, preprints and non-peer-reviewed commercial reports were excluded as numbered evidence sources. Existence and bibliographic metadata of the core references were cross-checked against PubMed and/or the primary publisher record before inclusion.

The approach is evidence-graded rather than claim confirmatory. A positive study was not considered transferable to the Biotex formulation when the tested ingredient, dose, extract standardisation, population or endpoint differed materially from the product. Negative or null primary studies were retained when they materially changed interpretation; examples include lack of mitochondrial-bioenergetic improvement despite NR-induced changes in the muscle NAD metabolome, lack of cognitive improvement in an NR pilot trial despite increased blood NAD+, a negative CoQ10 fatigue trial in post-polio sequelae, and an ashwagandha trial in which the primary perceived-stress comparison was not superior to placebo[4,10,23,40].

3. NAD+ Biology and Human Translational Evidence

3.1 NAD+ biosynthesis, salvage and consumption

Mammalian NAD+ is synthesised through several interconnected routes. Tryptophan contributes via the de novo pathway; nicotinic acid enters the Preiss-Handler pathway; and nicotinamide is recycled through the salvage pathway, in which nicotinamide phosphoribosyltransferase generates NMN and nicotinamide mononucleotide adenylyltransferases generate NAD+. NR can be phosphorylated to NMN before conversion to NAD+. These pathways coexist with continuous NAD+ consumption by sirtuins, PARPs, CD38 and related enzymes. Thus, tissue NAD status represents a dynamic balance between synthesis, recycling, compartmental exchange and enzymatic consumption rather than the simple intake of a single precursor.

The salvage pathway gives the Biotex nicotinamide component a genuine biochemical relationship to NAD+ metabolism. However, dose and precursor identity matter. The Biotex label provides 14 mg nicotinamide per capsule, a nutritional quantity rather than a dose comparable with most NAD-augmentation trials. Human NR studies have commonly used hundreds of milligrams to 1 g/day, while NMN trials have commonly used 250 mg/day or more[2-7]. The 2026 head-to-head study further shows why

it is scientifically inappropriate to use NR/NMN data as direct proof that oral nicotinamide produces the same sustained circulatory NAD+ response[1].

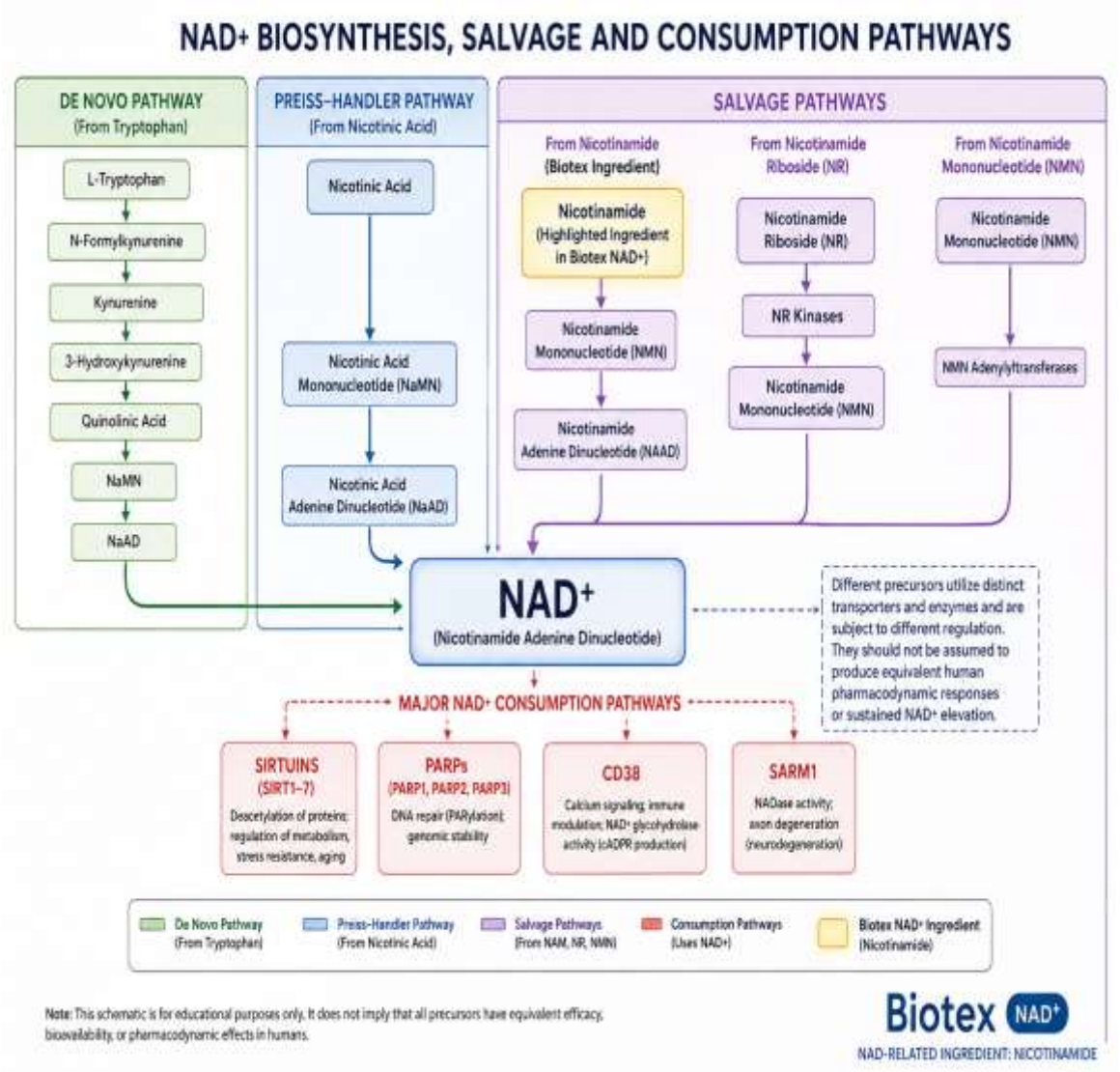

Fig 1: NAD+ biosynthesis, salvage and consumption pathways. The schematic illustrates the de novo pathway from tryptophan, the Preiss–Handler pathway from nicotinic acid, and salvage pathways from nicotinamide, nicotinamide riboside (NR) and nicotinamide mononucleotide (NMN), all converging on cellular NAD+. Major NAD+-consuming enzymes, including sirtuins, poly(ADP-ribose) polymerases (PARPs), CD38 and SARM1, are also shown. Nicotinamide is highlighted as the NAD-related ingredient in Biotex NAD+. The diagram distinguishes the different precursor routes and does not imply equivalent bioavailability, pharmacodynamic effects or sustained NAD+ elevation across nicotinamide, NR and NMN.

3.2 Human NAD-precursor trials: what is established and what is not

Controlled human studies establish proof of principle that the NAD metabolome is nutritionally modifiable. Martens and colleagues reported that six weeks of NR increased NAD-related metabolites in healthy middle-aged and older adults and was well tolerated[2]. Trammell and colleagues demonstrated oral bioavailability and a dose-dependent NAD metabolomic response to NR in humans[3]. In older men, Elhassan and colleagues found that 1 g/day NR for 21 days augmented the skeletal-muscle NAD metabolome, yet did not improve measured mitochondrial bioenergetics, demonstrating that biochemical target engagement does not automatically translate into improved mitochondrial function[4].

NMN trials provide a parallel human evidence stream. In postmenopausal women with prediabetes, NMN improved skeletal-muscle insulin sensitivity in a controlled trial[5]. In older men, 250 mg/day NMN elevated blood NAD-related measures and altered selected muscle-function outcomes[6]. Dose-ranging and older-adult trials have also documented increases in blood NAD and selected functional or sleep-related outcomes, although results across clinical endpoints are not uniformly positive[7-9].

Cognitive translation is similarly unresolved. In older adults with mild cognitive impairment, NR increased blood NAD+ but did not significantly alter cognition over the studied period[10]. This primary negative result is particularly relevant to products positioned for cognitive wellness: an increase in a biochemical NAD endpoint cannot be assumed to improve memory, focus or executive function. For the Biotex product, the cognitive rationale therefore depends more directly on the ashwagandha evidence and on general neuronal-energy biology than on a demonstrated NAD-mediated nootropic effect.

Table 1: Representative primary human NAD-precursor studies and their relevance to the Biotex NAD+ formulation.

Study	Population/design	Intervention	Key primary finding	Interpretation for this review
Christen et al., 2026	Healthy adults; randomised comparison	NR, NMN and nicotinamide; 14 days	NR and NMN produced sustained circulating NAD+ increases; nicotinamide showed a more acute/transient metabolomic response [1]	Critical for avoiding direct transfer of NR/NMN findings to 14 mg nicotinamide
Martens et al., 2018	Healthy middle-aged and older adults	NR; 6 weeks	Raised NAD-related metabolites and was well tolerated [2]	Establishes human target engagement for NR, not direct evidence for Biotex nicotinamide
Trammell et al., 2016	Human pharmacokinetic/metabolomic study	Oral NR; dose-ranging	Demonstrated oral bioavailability and dose-dependent NAD-metabolome responses [3]	Supports precursor pharmacology and route-specific differences
Elhassan et al., 2019	Older men; crossover study	NR 1 g/day; 21 days	Increased skeletal-muscle NAD metabolome but did not improve measured mitochondrial bioenergetics [4]	Shows that biochemical NAD target engagement does not guarantee functional mitochondrial benefit
Yoshino et al., 2021	Postmenopausal women with prediabetes	NMN	Improved skeletal-muscle insulin sensitivity in a controlled human trial [5]	Relevant to human NAD biology; intervention and dose differ from product
Orr et al., 2024	Older adults with mild cognitive impairment	NR	Increased blood NAD+ without significant cognitive improvement [10]	Important negative translational evidence for cognition

4. Confirmed Biotex NAD+ Formulation and Ingredient Rationale

The composition used in this review was taken from the manufacturer-supplied nutrition facts image. The serving size is one capsule (approximately 0.516 g). The label identifies six active ingredients and an HPMC vegetarian capsule shell. Because the botanical extracts and the CoQ10 form are not fully standardised on the supplied panel, the manuscript treats dose comparability conservatively.

Nutritional Facts (Approx. value)

Servings Per Container: Serving Size- 1 Capsule (0.516 g)

Ingredients:	Amount Per Serving	%RDA
Arjuna extract	150 mg	#
CoQ10 (from Non GM source & Ubiquinol acetate)	100 mg	33.3
Ashwagandha extract	100 mg	10.0
Adenosine 5-monophosphate	50 mg	#
Niacin as Nicotinamide	14 mg	100
Vitamin B2 as Riboflavin	2 mg	83.3

Additives: Capsule shell (Hydroxypropyl methyl cellulose).

%RDA values are established as per ICMR 2020 guidelines.
#%RDA value not established.

No Artifical flavors, Sugar, Starch, Wheat or Soy.
Gluten, Dairy, Sodium and Yeast free.

NOT FOR MEDICINAL USE

Fig 2: Biotex NAD+ nutraceutical formulation and labelled composition. Representative image of the Biotex NAD+ product alongside its nutritional information panel showing the per-capsule composition, including Arjuna extract (150 mg), CoQ10 (100 mg), Ashwagandha extract (100 mg), adenosine 5′-monophosphate (50 mg), niacin as nicotinamide (14 mg), and vitamin B2 as riboflavin (2 mg). The product image and label information are presented for formulation identification and contextualisation within the review.

Table 2. Confirmed Biotex NAD+ composition, proposed scientific role and principal evidence limitation for each ingredient.

Ingredient	Amount per capsule	Principal scientific role	Critical standardization/evidence issue
Terminalia arjuna extract	150 mg	Cardiovascular/endothelial support; fatigue/exercise-related outcomes	Plant part, extract ratio, solvent, and marker standardisation not stated on supplied panel
CoQ10 (non-GM source; label also refers to ubiquinol acetate)	100 mg	Mitochondrial electron transfer; fatigue; cardiovascular function	Exact chemical form and proportion of ubiquinone/ubiquinol-related material require clarification
Withania somnifera (Ashwagandha) extract	100 mg	Stress adaptation; cognition/focus; fatigue-related outcomes	Root vs root/leaf, extract ratio and withanolide standardisation require clarification
Adenosine 5'-monophosphate (AMP)	50 mg	Adenine-nucleotide/energy-sensing rationale	Direct oral AMP efficacy at this dose is not established in strong human trials.

Niacin as nicotinamide	14 mg	NAD+ salvage precursor; normal energy metabolism	Nutritional dose; not equivalent to NR/NMN intervention doses
Vitamin B2 as riboflavin	2 mg	FMN/FAD cofactor support for oxidative metabolism	Nutritional-dose metabolic cofactor

5. Natural Energy and Cellular Bioenergetics

5.1 Integrated mitochondrial rationale

The strongest mechanistic rationale of the formulation is not a direct 'NAD boost' but the convergence of several cofactors and electron-transfer components required for normal oxidative metabolism. NADH formed during substrate oxidation donates electrons to complex I. Complex I contains flavin mononucleotide derived from riboflavin and transfers electrons onward to the ubiquinone/ubiquinol pool. CoQ10 then shuttles electrons to complex III, contributing to the proton motive force ultimately used by ATP synthase. In this framework, nicotinamide contributes to NAD salvage, riboflavin supplies precursors for FMN/FAD, and CoQ10 participates directly in respiratory-chain electron transfer.

AMP is biologically important as part of the adenine nucleotide pool and intracellular energy sensing. Rising cellular AMP relative to ATP can activate AMP-activated protein kinase and related adaptive pathways. Nevertheless, the intracellular AMP/ATP ratio is not equivalent to dietary AMP intake, and strong primary human evidence showing that 50 mg oral AMP increases cellular ATP, activates AMPK in target tissues or reduces fatigue was not identified. AMP should therefore remain a mechanistic formulation component rather than an independent clinical evidence pillar.

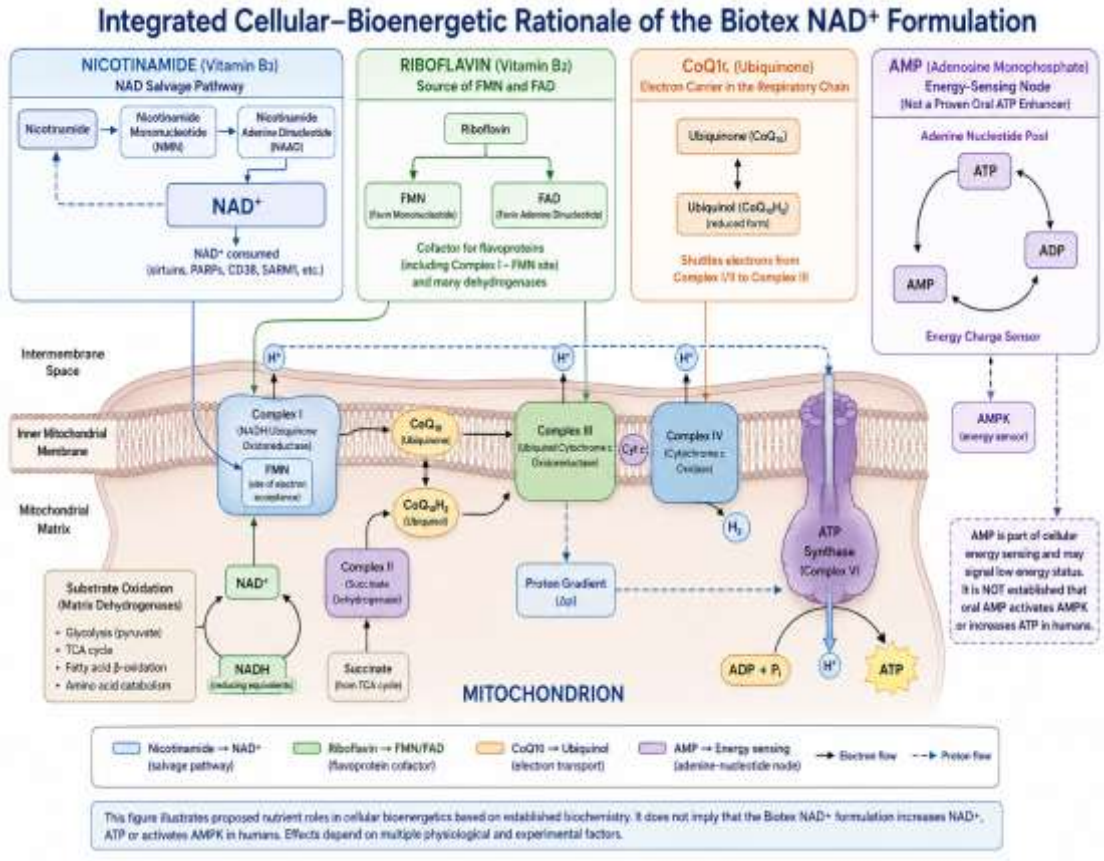

Fig 3: Integrated cellular-bioenergetic rationale of the Biotex NAD+ formulation. The schematic depicts substrate oxidation generating NADH, which transfers reducing equivalents to mitochondrial complex I, where FMN functions as an initial electron-accepting cofactor. Riboflavin is positioned as the precursor of FMN and FAD, supporting flavoprotein-dependent reactions, while CoQ10/ubiquinone and its reduced form ubiquinol mediate electron transfer from complexes I and II to complex III, followed by transfer through cytochrome c to complex IV. Electron transport supports proton translocation across the

inner mitochondrial membrane, generating the electrochemical gradient used by ATP synthase to form ATP from ADP and inorganic phosphate. Nicotinamide is shown as a substrate for NAD+ salvage metabolism. Adenosine monophosphate (AMP) is represented within the adenine-nucleotide energy-sensing network and its relationship with AMPK is shown as a physiological signalling context rather than evidence that oral AMP directly activates AMPK or increases ATP in humans.

5.2 CoQ10 and human fatigue/energy outcomes

CoQ10 provides the most dose-relevant human evidence for the product's energy and fatigue positioning. In a double-blind, placebo-controlled crossover study of healthy volunteers, 100 or 300 mg/day CoQ10 was administered for eight days; the higher dose improved selected measures during fatigue-inducing exercise, while responses were not uniform across all endpoints[17]. A later randomised study of healthy individuals with mild fatigue tested ubiquinol at 100 and 150 mg/day for 12 weeks and reported improvement in fatigue-related outcomes, making it particularly relevant to the 100-mg CoQ10/ubiquinol-related quantity on the Biotex label[18].

The evidence is not universally positive. In individuals with late-onset sequelae of poliomyelitis, 100 mg/day CoQ10 for 60 days did not alleviate fatigue[23]. This heterogeneity suggests that the effect of CoQ10 depends on population, baseline CoQ status, formulation, dose and cause of fatigue. Therefore, the defensible product-level statement is that CoQ10 has ingredient-level human evidence relevant to fatigue and mitochondrial support, not that the finished formulation is clinically proven to increase energy in all users.

Table 3: Primary human evidence for CoQ10/ubiquinol relevant to cellular energy, fatigue and cardiovascular wellness.

Study	Population/design	Intervention	Key finding	Claim domain/interpretation
Mizuno et al., 2008	Healthy volunteers; double-blind crossover	CoQ10 100 or 300 mg/day; 8 days	Higher dose improved selected fatigue/exercise outcomes; effects were not uniform across endpoints [17]	Energy/fatigue
Mizuno et al., 2020	Healthy adults with mild fatigue; randomised	Ubiquinol 100 or 150 mg/day; 12 weeks	Improved fatigue-related outcomes [18]	Most dose-aligned human evidence for the 100-mg CoQ10/ubiquinol-related label quantity
Mortensen et al., 2014 (Q-SYMBIO)	Chronic heart failure; multicenter randomised trial	CoQ10 100 mg three times daily	Improved several long-term clinical outcomes as adjunctive therapy [19]	Shows cardiovascular relevance; disease population and 300-mg/day dose preclude direct product-level inference
Dai et al., 2011	Ischaemic LV systolic dysfunction	CoQ10 supplementation	Improved endothelial function with mitochondrial-function-related changes [20]	Cardiovascular/endothelial mechanism
Hamilton et al., 2009	Statin-treated adults with type 2 diabetes	CoQ10 supplementation	Improved brachial-artery flow-mediated dilation [21]	Endothelial-function evidence
Lee et al.	Coronary artery	CoQ10 150	Lowered	Redox/cardiovascular

	disease	mg/day	oxidative-injury markers while enhancing endogenous antioxidant-enzyme activity [22]	support
Peel et al.	Post-polio fatigue population; randomised	CoQ10 100 mg/day; 60 days	Did not alleviate fatigue [23]	Important negative evidence; fatigue response is population-dependent

6. Healthy Stress Response

The presence of Withania somnifera substantially strengthens the formulation's stress-adaptation rationale. Stress is a multi-level phenomenon involving perception, autonomic balance, hypothalamic-pituitary-adrenal (HPA) signalling, sleep, inflammatory tone and metabolic demand. In a randomised double-blind placebo-controlled trial, a standardised ashwagandha root preparation improved stress and anxiety measures and reduced serum cortisol in stressed adults[24]. Lopresti and colleagues likewise reported stress-related improvements and findings consistent with modulation of the HPA axis in a randomised trial[25]. Salve and colleagues found improvements in perceived stress and cortisol-related outcomes with 250 and 600 mg/day root extract[26].

A balanced interpretation must also include trials with weaker primary outcomes. Smith and colleagues evaluated a standardised ashwagandha root extract in adults with high stress and fatigue; although stress scores improved over time, the primary between-group Perceived Stress Scale comparison was not superior to placebo, while some secondary fatigue-related outcomes were more favourable [40]. Consequently, the manuscript supports 'healthy stress response' more strongly than categorical treatment language for anxiety or stress disorders.

7. Cognitive Health, Attention and Focus

The cognitive claim requires separation of neuronal-energy plausibility from direct psychometric evidence. NAD+/NADH, flavin cofactors and CoQ10 participate in neuronal energy metabolism, but human NAD-precursor trials have not established consistent cognitive enhancement. The NR trial in mild cognitive impairment is especially instructive: blood NAD+ increased, yet cognition did not improve significantly[10]. Thus, the product's direct human cognitive evidence is more appropriately anchored to W. somnifera than to the NAD+ label concept.

Gopukumar and colleagues conducted a randomised double-blind placebo-controlled trial in healthy stressed adults and reported improvements in memory/focus-related outcomes, psychological well-being and sleep with a standardised ashwagandha root extract[29]. Leonard and colleagues evaluated acute and repeated 225-mg/day liposomal ashwagandha in healthy adults and reported improvements in selected measures of memory, attention, vigilance and executive function together with reduced perceptions of tension and fatigue[30]. In people with mild cognitive impairment, Choudhary and colleagues reported favourable changes across several memory measures and selected executive, attentional and information-processing tasks after ashwagandha root extract[31].

These trials support ingredient-level cognitive potential but do not establish that 100 mg of the unspecified Biotex extract produces the same effect. In addition, 'focus' is not interchangeable with broad cognitive health. The strongest focus-relevant evidence comes from attention, vigilance and executive-function tasks in standardised-extract trials, whereas the finished product has not undergone a validated attention battery. A publication-ready claim should therefore use language such as 'supports cognitive wellness and focus-related functions' rather than 'improves memory' or 'boosts brain power'.

Sleep and stress can indirectly influence daytime cognition. A randomised trial of ashwagandha in healthy adults reported improved sleep quality with a standardised extract[32]. Such findings provide a plausible secondary route by which stress-adaptation ingredients may support sustained daytime functioning, but sleep outcomes should not be presented as direct proof of improved attention.

Table 4: Primary human evidence for Withania somnifera relevant to healthy stress response, fatigue, cognition and focus.

Study	Population/design	Intervention	Key finding	Interpretation for Biotex NAD+
Chandrasekhar et al., 2012	Stressed adults; randomised, double-blind, placebo-controlled	Standardised root preparation; 300 mg twice daily	Improved stress/anxiety measures and reduced serum cortisol [24]	Strong stress-response evidence but dose exceeds Biotex 100 mg
Lopresti et al., 2019	Adults with stress; randomised double-blind placebo-controlled	Standardised ashwagandha extract	Improved stress-related outcomes with findings consistent with HPA-axis modulation [25]	Stress/HPA-axis support
Salve et al.	Healthy adults; randomised	Root extract 250 or 600 mg/day	Improved perceived stress and cortisol-related outcomes [26]	Stress support; higher doses than product
Pandit et al.	Chronically stressed adults; randomised	Characterised aqueous extract 125, 250 or 500 mg/day	Reported dose-related stress reduction and HPA-axis effects [27]	125-mg arm is relatively dose-proximate, but standardisation must match
Mishra & Kumar	Adults with stress/anxiety; randomised	Highly standardised low-dose extract 60 or 120 mg/day	Reduced stress/anxiety outcomes [28]	Dose-proximate evidence only if extract standardisation is comparable
Gopukumar et al., 2021	Healthy stressed adults; randomised double-blind placebo-controlled	Standardised root extract	Improved memory/focus-related outcomes, wellbeing and sleep [29]	Cognitive/focus support
Leonard et al., 2024	Healthy adults; acute and repeated randomised supplementation	Liposomal ashwagandha 225 mg/day	Improved selected memory, attention, vigilance and executive-function measures; reduced tension/fatigue perception [30]	Directly relevant to focus-related endpoints; dose/form differs
Choudhary et al., 2017	Mild cognitive impairment; randomised trial	Ashwagandha root extract	Favourable changes across memory and selected executive/attentional performance measures [31]	Supports ingredient-level cognitive potential in a specific population
Smith et al.	Adults with high stress/fatigue	Standardised root extract	Primary between-group perceived-stress endpoint was not superior to	Balanced/negative-primary evidence that limits categorical stress claims

			placebo; some secondary fatigue outcomes were more favourable [40]	

8. Cardiovascular and Heart Wellness

8.1 CoQ10 and cardiac bioenergetics/endothelial function

Cardiac tissue has a high and continuous ATP requirement, making mitochondrial electron transport central to myocardial performance. CoQ10 has both electron-carrier and redox functions, and it has been studied extensively in cardiovascular populations. In Q-SYMBIO, a multicenter randomised double-blind trial in chronic heart failure, 100 mg CoQ10 three times daily as adjunctive therapy improved several long-term clinical outcomes[19]. Because this was a disease-specific trial at 300 mg/day, it supports the clinical relevance of CoQ10 to cardiac physiology but cannot be translated into a claim that a 100-mg nutraceutical treats heart failure.

More mechanistically aligned human studies have examined endothelial and mitochondrial endpoints. In participants with ischaemic left-ventricular dysfunction, CoQ10 supplementation improved vascular reactivity and was accompanied by changes consistent with better mitochondrial performance[20]. Hamilton and colleagues observed improved brachial-artery flow-mediated dilation in statin-treated adults with type 2 diabetes after CoQ10 supplementation[21]. Among participants with coronary artery disease, 150 mg/day CoQ10 lowered several indices of oxidative injury while enhancing endogenous antioxidant-enzyme activity[22]. Together, these primary trials support cardiovascular and redox relevance, while leaving product-specific efficacy untested.

8.2 Terminalia arjuna clinical evidence and dose comparability

Terminalia arjuna provides the second cardiovascular pillar of the formulation. A randomised double-blind placebo-controlled trial of a standardised T. arjuna bark extract (E-OJ-01/Oxyjun) in healthy adults reported an increase in left-ventricular ejection fraction and reduction in fatigue compared with baseline/placebo analyses[33]. A separate randomised study in young exercising adults reported improvement in cardiac-conditioning measures with the standardised extract[34]. These studies are particularly relevant because they investigate functional cardiovascular outcomes in non-disease or broadly healthy populations.

Older clinical studies add endothelial and exercise-related evidence but require cautious interpretation. In chronic smokers, T. arjuna improved impaired endothelium-dependent vasodilation over two weeks[35]. In chronic stable angina, a randomised crossover study reported improvements in treadmill and symptom-related measures compared with placebo[36]. However, these disease-specific studies used substantially larger quantities than the 150 mg extract in Biotex NAD+ and should not be converted into disease-treatment claims.

A rigorous review must also preserve conflicting findings. In a 100-patient double-blind randomised placebo-controlled trial in chronic heart failure, 750 mg T. arjuna extract twice daily did not significantly improve the primary LVEF endpoint at 12 weeks, although some functional, antioxidant and quality-of-life outcomes improved[37]. A randomised placebo-controlled trial of T. arjuna bark powder also reported antioxidant and lipid-related changes in coronary heart disease patients[38]. Collectively, these studies justify cardiovascular-wellness relevance but not a conclusion of established cardioprotective efficacy at 150 mg/day.

An additional healthy-adult study assessed *W. somnifera* and *T. arjuna* in relation to physical performance and cardiorespiratory endurance, reporting improvements in selected performance variables[39]. The study included Ashwagandha-alone, Arjuna-alone, and combined Ashwagandha-Arjuna intervention arms; however, it did not evaluate the complete six-component Biotex NAD+ formulation. It is therefore best interpreted as supporting ingredient-level and two-botanical combination plausibility rather than finished-product synergy.

Table 5: Primary human evidence for Terminalia arjuna relevant to cardiovascular wellness and functional outcomes.

Study	Population/design	Intervention	Key finding	Interpretation for Biotex NAD+
Srivastava et al., 2022	Healthy adults; randomised, double-blind, placebo-controlled	Standardised T. arjuna bark extract (E-OJ-01/Oxyjun)	Reported increased LVEF and reduced fatigue in trial analyses [33]	Most directly relevant healthy-adult cardiovascular/fatigue evidence; exact dose/extract comparability required
Girandola & Srivastava, 2017	Young exercising adults; randomised	Standardised T. arjuna extract	Improved selected cardiac-conditioning measures [34]	Functional cardiovascular evidence in broadly healthy adults
Bharani et al., 2004	Chronic smokers	T. arjuna; 2 weeks	Improved impaired endothelium-dependent vasodilation [35]	Endothelial relevance; population and dosing differ from product
Bharani et al., 2002	Chronic stable angina; randomised crossover	T. arjuna	Improved treadmill/symptom-related measures versus placebo [36]	Disease-specific evidence; cannot be converted to treatment claim
Maulik et al., 2016	Chronic heart failure; 100-patient double-blind randomised placebo-controlled trial	T. arjuna extract 750 mg twice daily	Did not significantly improve primary LVEF endpoint; some functional, antioxidant and quality-of-life outcomes improved [37]	Important conflicting evidence and major dose difference
Gupta et al.	Coronary heart disease; randomised controlled	T. arjuna bark powder	Reported antioxidant and lipid-related changes [38]	Supplementary cardiovascular/redox evidence
Sandhu et al., 2010	Healthy young adults	Ashwagandha alone, Arjuna alone, and combined Ashwagandha + Arjuna intervention arms	Reported improvements in selected physical/cardiorespiratory performance variables [39]	Supports ingredient-level and two-botanical combination plausibility; does not test the complete six-ingredient Biotex NAD+ formulation

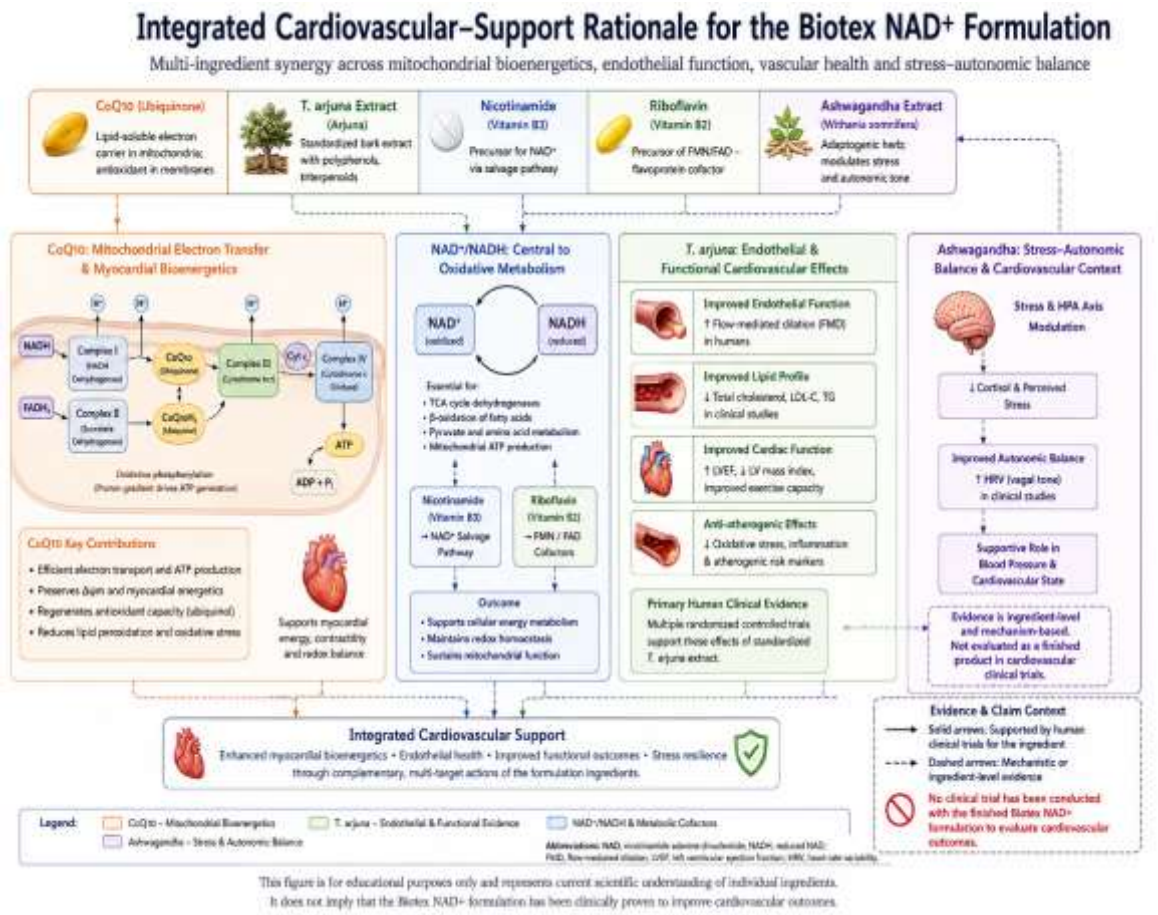

Fig 4: Integrated cardiovascular-support rationale of the Biotex NAD+ formulation. CoQ10 is linked to mitochondrial electron transfer and myocardial bioenergetics, *Terminalia arjuna* to endothelial and cardiovascular function, nicotinamide/riboflavin to metabolic cofactor pathways, and ashwagandha to stress–autonomic regulation. Dashed arrows indicate mechanistic or ingredient-level evidence; no finished-product cardiovascular trial is implied.

9. Cellular Maintenance, Healthy Ageing and Longevity-Related Pathways

NAD+ has become prominent in ageing research because it links metabolic flux with enzymes involved in chromatin regulation, DNA-damage responses, mitochondrial quality control and inflammatory signalling. In primary experimental work, Gomes and colleagues demonstrated that reduced NAD availability in aged mice altered oxygen-sensing pathways and impaired coordination between nuclear and mitochondrial metabolic programmes[11]. Camacho-Pereira and colleagues identified increased CD38 activity as an important contributor to the loss of cellular NAD during ageing, with downstream impairment of mitochondrial function involving SIRT3 signalling[12]. Later studies connected senescent-cell inflammatory signalling with accumulation of CD38-positive macrophages and tissue NAD depletion[15].

Preclinical NAD repletion can improve aspects of physiological ageing. Chronic NMN exposure in mice was associated with preservation of several metabolic and functional parameters that otherwise deteriorated with advancing age[13]. In another mouse model, restoration of NAD availability improved mitochondrial performance and muscle stem-cell activity and was accompanied by increased survival[14]. These studies are high-impact primary research, but the species, doses and interventions differ fundamentally from the Biotex formula. They support a biological rationale for healthy-ageing pathways, not a human longevity claim.

The distinction between healthspan and lifespan is critical. Mitchell and colleagues reported that long-term nicotinamide administration favourably affected several physiological measures of ageing in mice, although overall survival duration remained unchanged[16]. This finding directly argues against equating nicotinamide supplementation with proven longevity. In humans, NR/NMN trials demonstrate

target engagement and selected metabolic or functional signals, but no adequately designed study has shown extension of human lifespan[1-10]. Therefore, the consumer-facing phrase 'cellular rejuvenation and longevity' should be translated in academic prose to 'cellular maintenance and healthy-ageing-related pathways'.

For the Biotex formulation, CoQ10 and riboflavin contribute to mitochondrial redox metabolism, while ashwagandha and T. arjuna add stress and cardiovascular dimensions. These complementary mechanisms may be relevant to maintaining physiological resilience across ageing, but the complete combination has not been tested against validated ageing biomarkers, epigenetic clocks, mitochondrial-respiration endpoints or long-term functional trajectories.

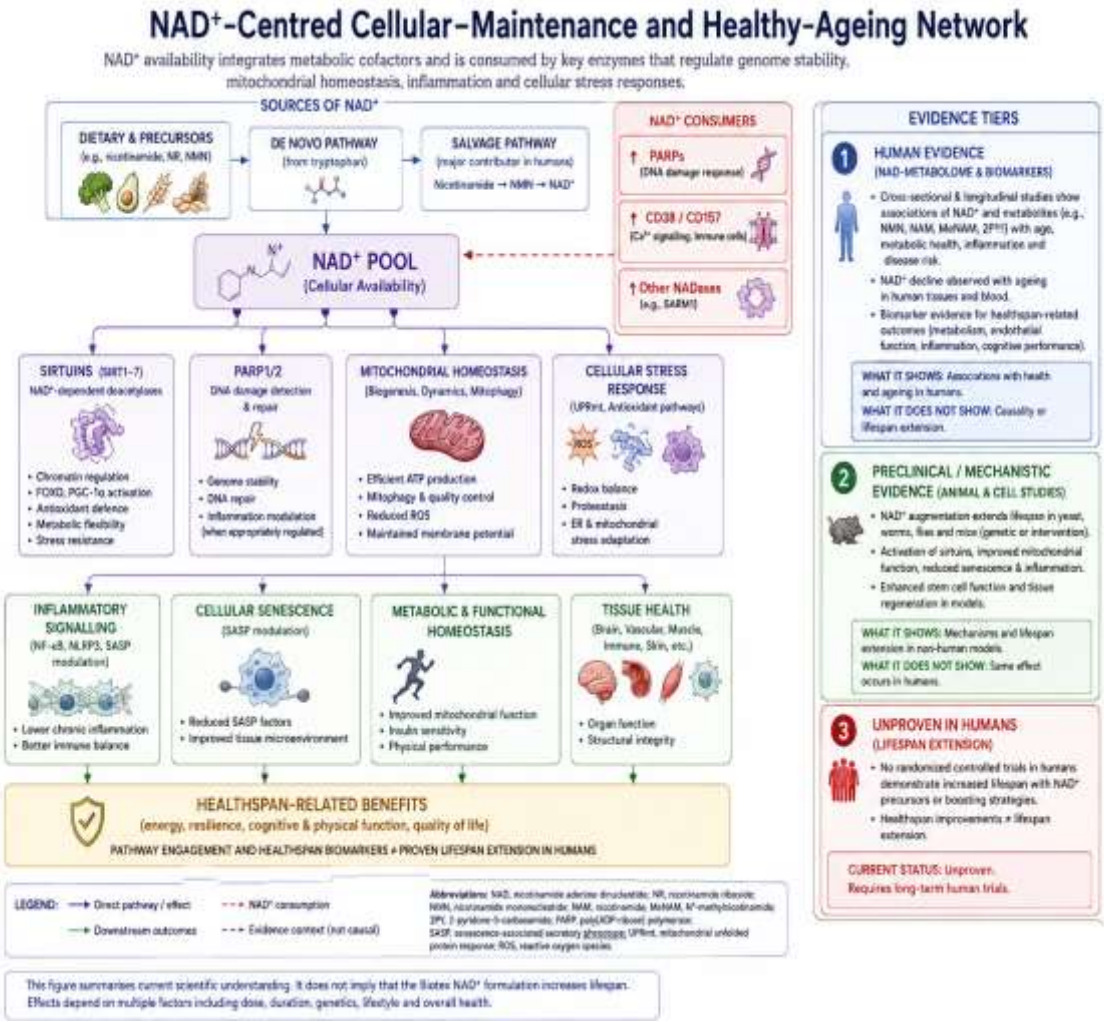

Fig 5: NAD+-centred cellular-maintenance and healthy-ageing network. The schematic links NAD+ availability with sirtuins, PARPs, CD38, mitochondrial homeostasis, cellular stress responses, inflammation and senescence, while distinguishing human biomarker evidence from preclinical ageing mechanisms and unproven human lifespan extension.

10. Integrated Formulation Rationale and Evidence-to-Claim Translation

The formulation is best understood as a convergence of partially independent evidence domains. Nicotinamide and riboflavin are nutritional metabolic cofactors; CoQ10 has human evidence for fatigue and cardiovascular physiology; ashwagandha has human evidence for stress adaptation and selected cognitive outcomes; T. arjuna has human cardiovascular evidence; and AMP provides biochemical plausibility but limited direct oral clinical evidence. These components are mechanistically complementary, yet complementarity is not synonymous with pharmacological synergy.

No primary clinical study was identified that tested the complete Biotex NAD+ formulation, measured circulating or tissue NAD+ after its use, quantified ATP production, or evaluated the exact combination against fatigue, attention, stress, endothelial or healthy-ageing endpoints. Therefore, finished-product efficacy remains an evidence gap. The most academically defensible review architecture is to state the strength of each ingredient-level evidence stream and then explicitly identify the missing formulation-level bridge.

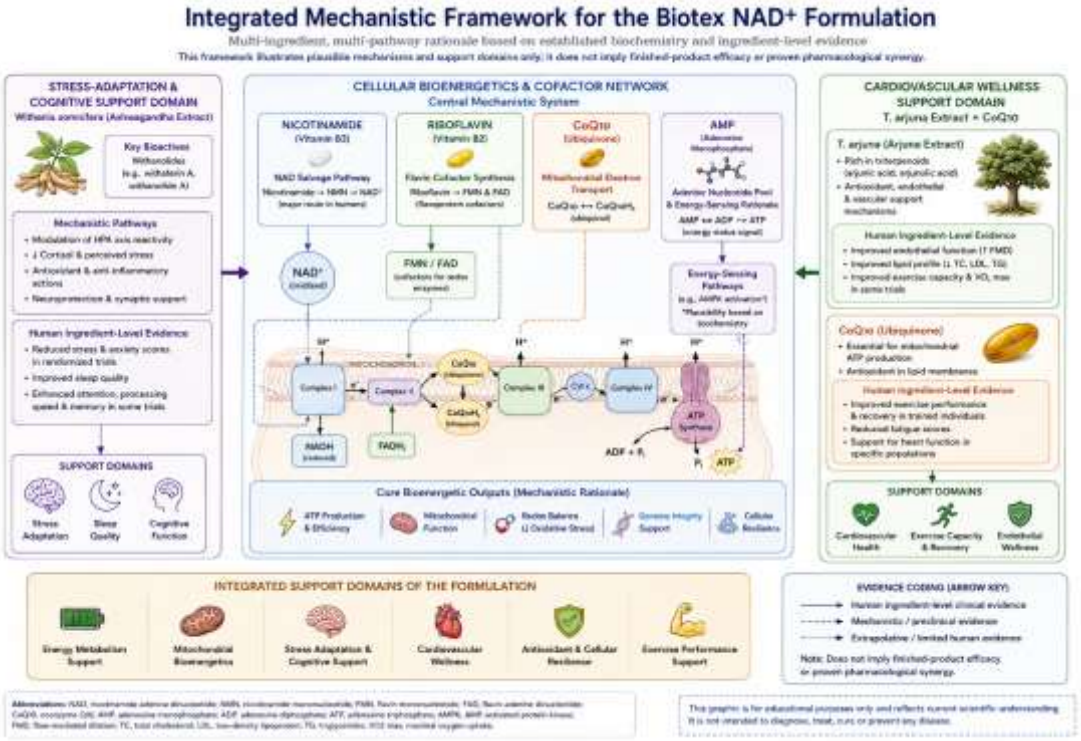

Fig 6: Integrated mechanistic framework of the Biotex NAD+ formulation. The schematic links formulation ingredients to cellular bioenergetics, stress–cognitive support and cardiovascular wellness, with evidence-coded arrows distinguishing human, mechanistic and limited evidence.

Table 6: Evidence-to-claim translation for the confirmed Biotex NAD+ formulation.

Support domain	Principal components	Current evidence strength	Key gap	Publication-safe interpretation
Natural energy / cellular bioenergetics	CoQ10, nicotinamide, riboflavin; AMP mechanistic	Strong mechanistic; moderate human ingredient evidence	No finished-product ATP/NAD study	Support normal energy metabolism / daily vitality
Fatigue reduction	CoQ10; ashwagandha; T. arjuna	Moderate-to-good ingredient-level human evidence; heterogeneous	No finished-product fatigue RCT	May help support reduced feelings of tiredness / fatigue-related wellness
Healthy stress response	Ashwagandha; redox-energy context	Good human evidence for standardised ashwagandha extracts	Biotex extract standardisation unknown	Supports adaptation to everyday stressors
Cognitive health/focus	Ashwagandha; neuronal energy biology	Moderate; attention/vigilance signals in selected trials	No finished-product cognitive battery	Supports cognitive wellness and focus-related functions
Cardiovascular / heart wellness	CoQ10 + T. arjuna	Good ingredient-level evidence, mostly	No finished-product	Supports cardiovascular

		dose/formulation dependent	cardiovascular trial	wellness
Cellular maintenance / healthy ageing	NAD biology, mitochondrial pathways	Strong mechanistic and preclinical; emerging human NAD biomarker evidence	No human lifespan evidence; product not shown to raise NAD+	Supports pathways relevant to healthy ageing
Longevity	NAD-related preclinical ageing studies	Preclinical only for lifespan; no proof for product	Not established in humans	Avoid lifespan-extension claim

11. Standardisation, Dose Comparability, Bioavailability and Safety

11.1 Standardisation and dose comparability

The most important product-development issue exposed by the literature is standardisation. The label lists 100 mg ashwagandha extract, but does not state whether the material is root, root-and-leaf or another plant part, the extraction solvent/ratio, or the withanolide concentration. Because clinically studied extracts differ markedly in withanolide composition, a 60-125 mg highly standardised preparation can be pharmacologically different from 100 mg of an unspecified extract[27,28]. The same principle applies to T. arjuna: bark identity, extract ratio, solvent and marker profile are required before 150 mg can be compared with 400 mg/day standardised Oxyjun or gram-level bark/extract regimens used in older trials[33-38].

The CoQ10 label wording also requires clarification. 'CoQ10 (from non-GM source & ubiquinol acetate) 100 mg' does not unambiguously state whether the entire 100 mg is ubiquinone, ubiquinol, an ester-related material or a defined combination. This matters because pharmacokinetics and dose comparisons depend on chemical form and formulation. The 100-mg ubiquinol trial is encouragingly dose-aligned, but direct equivalence should not be claimed until the product specification is confirmed[18].

Nicotinamide and riboflavin occur at nutritional rather than pharmacological quantities. Their scientific role is therefore best framed as support for normal cofactor availability and metabolic pathways. The AMP content should be treated similarly conservatively because oral bioavailability, conversion and tissue exposure at 50 mg have not been linked to validated clinical endpoints in strong primary human trials.

Table 7. Dose and evidence comparability between the confirmed Biotex NAD+ formulation and the closest relevant primary human studies.

Ingredient	Biotex amount	Closest primary evidence	Study exposure/context	Comparability	Publication-safe interpretation
Nicotinamide	14 mg	Christen et al., 2026 [1]	Direct human precursor comparison; pharmacological intervention context	Low direct comparability	Confirms that nicotinamide behaves differently from NR/NMN; does not establish sustained NAD+ elevation at 14 mg.
CoQ10 / ubiquinol-related material	100 mg	Mizuno et al., 2020 [18]	Ubiquinol 100 or 150 mg/day for 12 weeks	Relatively high dose alignment	Strongest dose-proximate human evidence for fatigue-related outcomes, pending confirmation of

					the exact CoQ10 chemical form in the product.
Withania somnifera extract	100 mg	Pandit et al., 2024 [27]; Mishra & Kumar, 2024 [28]	Characterised extracts at 125 mg/day and 60 or 120 mg/day, respectively	Potentially high if standardisation matches	Dose proximity is encouraging, but equivalence depends on plant part, extraction process and withanolide specification.
Terminalia arjuna extract	150 mg	Srivastava et al., 2022 [33]	Standardised Oxyjun/E-OJ-01 regimen reported in the manuscript as 400 mg/day	Moderate-to-low	Human cardiovascular/fatigue relevance is present, but the product dose is lower and exact extract standardisation is not yet documented.
Adenosine 5'-monophosphate	50 mg	No strong dose-matched primary human efficacy trial identified in the curated evidence set.	Not established	Low / evidence gap	AMP contributes mechanistic adenine-nucleotide plausibility; direct human efficacy at 50 mg should not be claimed.
Riboflavin	2 mg	Nutritional cofactor biology rather than pharmacological-dose intervention evidence	Nutritional exposure	Contextual rather than therapeutic	Best interpreted as support for normal FMN/FAD-dependent energy metabolism, not as an independent pharmacological efficacy driver.

11.2 Safety and interaction considerations

The selected primary clinical trials generally reported acceptable short-term tolerability of NR/NMN, CoQ10, standardised ashwagandha extracts and T. arjuna under study conditions[2,5-7,18,24-28,33,37]. This does not establish long-term safety of the complete combination. Botanical products also require authentication, contaminant testing, pesticide/heavy-metal limits, microbial quality, residual-solvent control and stability testing. For ashwagandha and Arjuna, reproducible phytochemical fingerprints are particularly important.

Potential medicine-supplement interactions should be approached conservatively because the complete formulation has not undergone dedicated interaction studies. Because the product combines botanical extracts with metabolically active nutrients, individuals receiving prescription medicines, those with chronic medical conditions, and those with polypharmacy should seek professional guidance before

use. The finished product is a nutraceutical, and the supplied label states that it is not for medicinal use. Individuals who are pregnant or breastfeeding should also seek professional guidance before use.

Table 8. Safety, tolerability and interaction considerations relevant to the confirmed Biotex NAD+ formulation.

Component	Evidence from selected studies	Interaction/population caution	Interpretation
CoQ10 / ubiquinol-related material	Generally acceptable short-term tolerability in the selected primary clinical studies [18-23].	No dedicated interaction study of the finished formulation was identified; professional guidance is appropriate when used alongside prescription medicines.	Ingredient-level tolerability does not establish long-term safety of the complete combination.
Withania somnifera	Selected randomised trials reported acceptable short-term tolerability under study conditions [24-28,40].	No dedicated interaction study of the finished formulation was identified; professional guidance is appropriate when used alongside prescription medicines or in medically complex individuals.	Safety interpretation depends on extract standardisation and dose; finished-product interaction studies are unavailable.
Terminalia arjuna	Primary cardiovascular trials provide short-term clinical exposure data [33-39].	No dedicated interaction study of the finished formulation was identified; professional guidance is appropriate when used alongside prescription medicines.	Disease-population studies cannot define safety of chronic wellness use at the Biotex dose.
Nicotinamide	Present at a nutritional quantity (14 mg) in the confirmed label.	No product-specific interaction study identified.	Should be interpreted as nutritional cofactor support; finished-formulation long-term safety remains untested.
Riboflavin	Present at a nutritional quantity (2 mg) in the confirmed label.	No product-specific interaction study identified.	Primarily a nutritional cofactor; does not materially resolve combination-safety uncertainty.
Adenosine 5'-monophosphate	Direct strong human efficacy/safety evidence at 50 mg was limited in the curated primary evidence set.	Clinical interaction profile of the finished oral combination has not been established.	Treat conservatively as an evidence-limited component until formulation-specific data are available.
Finished Biotex NAD+ formulation	No published long-term randomised safety trial of the complete combination was identified.	Professional guidance is appropriate for individuals using prescription medicines, those with chronic medical conditions or polypharmacy, and during pregnancy or breastfeeding.	Ingredient-level tolerability cannot substitute for combination-specific safety surveillance.

Table 9. Product specifications required for rigorous evidence translation and dose comparability.

Ingredient	Recommended specification	Why it matters
Ashwagandha 100 mg	Plant part; extract ratio; solvent; total withanolides/withanolide profile	Essential for comparing with 60-125 mg highly standardised trials and 250-600 mg conventional extracts
T. arjuna 150 mg	Confirm T. arjuna; bark/plant part; extract ratio; solvent; marker compounds	Essential for comparing with standardised 400 mg/day and higher-dose clinical studies
CoQ10-related 100 mg	Clarify ubiquinone vs ubiquinol vs "ubiquinol acetate"; assay and purity	Determines dose/formulation comparability with 100-150 mg ubiquinol and higher-dose CoQ10 studies
AMP 50 mg	Identity, assay, purity, stability	Human efficacy evidence is currently weak; quality specification still required.
Nicotinamide 14 mg	Assay, stability	Supports nutritional NAD-salvage rationale; does not establish systemic NAD+ augmentation
Riboflavin 2 mg	Assay, stability, light protection	Supports FMN/FAD cofactor biology at nutritional dose

12. Limitations and Research Priorities

This review has several deliberate boundaries. It is a critical narrative synthesis rather than a formal systematic review, and the evidence set was curated for relevance to the confirmed formulation and support domains. Only primary publications were used as numbered scientific references. This improves traceability to original experiments but means that pooled effect estimates from meta-analyses are intentionally not presented.

The major formulation-level limitations are clear: (1) no published randomized trial of the finished Biotex NAD+ combination; (2) no direct demonstration that one capsule increases circulating or tissue NAD+; (3) substantial dose and precursor differences between human NR/NMN studies and 14 mg nicotinamide; (4) incomplete ashwagandha and T. arjuna extract standardization; (5) ambiguous CoQ10 chemical-form wording; (6) limited direct human evidence for oral AMP 50 mg; (7) no product-specific attention/focus or fatigue endpoint; and (8) no evidence that the product extends human lifespan or produces clinical 'cellular rejuvenation'.

The most informative development sequence would begin with analytical characterisation and a short pharmacodynamic study measuring whole-blood NAD+, NADH and related metabolites after repeated dosing. A randomised placebo-controlled fatigue/energy study could then use validated fatigue instruments and, where feasible, objective exercise or mitochondrial endpoints. A stress study should include a validated perceived-stress instrument plus morning cortisol or other prespecified HPA-related biomarkers. Cognitive testing should prespecify attention, vigilance, working memory and executive-function outcomes. Cardiovascular studies in generally healthy adults could focus on endothelial function, resting hemodynamics and exercise capacity rather than disease-treatment endpoints.

For healthy-ageing claims, the appropriate initial targets are mechanistically anchored biomarkers rather than lifespan language. Longitudinal measures of NAD metabolites, mitochondrial function, inflammatory markers, physical performance and potentially validated biological-age measures could be explored, but no single biomarker should be interpreted as proof of slowed human ageing.

Table 10: Priority studies and endpoints for formulation-specific validation of Biotex NAD+.

Research stage	Suggested design	Primary measurements	Interpretive purpose
Analytical characterization	Confirm identity, assay and stability of all six actives; define CoQ10 chemical form; characterise withanolides and T.	Release specifications, chromatographic fingerprints, contaminant/stability profile	Required before meaningful dose comparison with published studies

	arjuna markers.		
NAD pharmacodynamics	Randomised placebo-controlled repeated-dose study	Whole-blood NAD+, NADH, NAD metabolome; time course	Tests whether the finished formulation alters NAD biology at the labelled dose
Energy/fatigue	Randomised placebo-controlled efficacy study in adults with mild fatigue	Validated fatigue scale; optional exercise capacity/mitochondrial markers	Directly tests the strongest consumer-facing energy/fatigue domain
Healthy stress response	Randomised placebo-controlled study	Validated perceived-stress score; morning cortisol or prespecified HPA-related biomarkers	Links the product, not just ashwagandha, to stress-adaptation outcomes
Cognition/focus	Prespecified cognitive-battery study	Attention, vigilance, working memory, executive function; avoid composite post hoc claims	Needed before direct focus/cognitive-performance claims
Cardiovascular wellness	Generally healthy or risk-factor population	Endothelial function, resting hemodynamics, exercise capacity	Tests product-level cardiovascular support without disease-treatment positioning
Healthy-ageing biology	Longer-duration exploratory study	NAD metabolites, physical function, mitochondrial/redox/inflammatory biomarkers	Biomarkers may indicate pathway engagement but should not be equated with lifespan extension.

13. Conclusion

The confirmed Biotex NAD+ formulation has a coherent multi-component scientific rationale, but its strongest justification is broader than direct NAD+ augmentation. CoQ10/ubiquinol-related material provides human evidence relevant to mitochondrial function, fatigue and cardiovascular physiology; W. somnifera provides the principal human evidence for stress adaptation and selected cognitive/focus outcomes; T. arjuna contributes cardiovascular and functional evidence; nicotinamide and riboflavin support normal cofactor pathways; and AMP adds a biochemical energy-sensing rationale with comparatively limited direct oral clinical evidence.

Human NR and NMN studies confirm that NAD metabolism can be modified, while the 2026 direct comparison of NAD precursors shows that nicotinamide should not be assumed to produce the same sustained circulatory NAD+ response[1]. Therefore, the finished formulation should not be described as clinically proven to raise NAD+, increase ATP, rejuvenate cells or extend lifespan. The strongest publication-ready interpretation is that the ingredients collectively target biological pathways relevant to cellular energy, daily vitality, fatigue resistance, stress adaptation, cognitive wellness, cardiovascular function and healthy ageing. Product-specific randomised trials remain necessary to determine whether these complementary ingredient-level effects translate into clinically meaningful outcomes from the complete formulation.

Declarations

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Competing interests: All authors are employees of Biotex Life Solutions Pvt. Ltd., the company that developed and markets the Biotex NAD+ formulation discussed in this review. This employment and institutional relationship is disclosed as a potential competing interest. The manuscript is a literature-based

scientific review and does not imply product-specific clinical efficacy in the absence of direct clinical trials. The authors declare no other competing interests.

Author contributions: All authors contributed to the conception and scope of the review, literature identification, evidence appraisal, interpretation of the published data, and development of the manuscript. All authors participated in drafting and critical revision of the article, reviewed the final manuscript, approved the version submitted for publication, and agree to be accountable for the integrity of the work. Sahithi Polineni coordinated manuscript preparation and correspondence with the journal.

Data availability: All scientific data discussed in this review are derived from the cited published primary studies. Product composition was verified from the manufacturer-supplied nutrition facts image reproduced in the manuscript. No new datasets were generated or analysed for this review.

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Use of artificial-intelligence tools: Generative artificial-intelligence tools were used during manuscript preparation for language organisation, structural formatting and document preparation. Scientific interpretation, literature selection, reference verification, factual accuracy and responsibility for the final manuscript remained with the authors. All authors reviewed and approved the final content.

Disclaimer: This article is a scientific review of published evidence and is not intended to diagnose, treat, cure or prevent disease. Ingredient-level evidence should not be interpreted as proof of efficacy of the finished formulation.

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