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Selling Longevity: A Narrative Review of the Gap Between the Evidence of 'Anti-ageing' and Commercial Claims

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Abstract

Longevity medicine aims to extend healthspan by targeting the biology of ageing rather than individual diseases. The field rests on credible geroscience, but it has grown alongside a commercial and social media ecosystem that often presents early findings as established clinical fact. This narrative review examines four areas central to both the science and its marketing: senolytic drugs, nicotinamide adenine dinucleotide (NAD+) precursors, caloric restriction, and biological age testing with epigenetic clocks. For each, we compare the human evidence with the claims commonly made to consumers, distinguishing throughout between biomarkers, which show a biological change, and clinical outcomes, which show whether people live longer or healthier lives.

Senolytics have shown biological signals in small, mostly uncontrolled trials, but clinical efficacy has not been established. NAD+ precursors reliably raise blood NAD+ and appear safe in the short term, yet no trial has shown benefit on function, disease or survival. Caloric restriction has the strongest randomised evidence, with the CALERIE trial showing modest favourable effects on ageing biomarkers, though effect sizes are small and long-term adherence is difficult. Epigenetic clocks are valuable population-level research tools but do not yet meet the reliability standards needed for individual clinical decisions.

Across all four areas, commercial messaging runs ahead of the data in consistent ways: extrapolation from animal models, substitution of surrogate biomarkers for outcomes, rebranding of anti-ageing claims as "longevity", and direct-to-consumer promotion by influencers with financial interests. Regulatory and legal actions against named products and clinics, including young-plasma infusions, telomerase supplements, NAD+ supplement advertising and unapproved stem-cell products, illustrate these patterns. Regulatory frameworks that treat many products as food supplements or wellness services enable these patterns. We offer practical recommendations for clinicians counselling patients, for researchers and journals communicating findings, and for regulators and platforms.

Categories: Other, Preventive Medicine, Epidemiology/Public Health
Keywords: biomarkers, epigenetic biomarker, longevity medicine, nicotinamide adenine dinucleotide (nad+), senolytics

Introduction And Background

Ageing is the greatest risk factor for most chronic diseases, including cardiovascular disease, most cancers, neurodegeneration and metabolic disease [1-3]. Geroscience starts from the observation that interventions extending lifespan in model organisms often delay or prevent many of these diseases at once [2], and proposes that ageing raises disease risk through a shared set of biological processes, the "hallmarks of ageing", such as accumulation of senescent cells, DNA damage, altered nutrient sensing, mitochondrial dysfunction and changes to the epigenome [1]. If these processes could be slowed, several diseases might be delayed together rather than treated one at a time [2]. This idea has moved from the laboratory into clinical research, and "longevity medicine" is increasingly used to describe its application to patients.

The same idea has also become a large consumer market. Supplements, private longevity clinics, biological age tests and subscription health-optimisation programmes are now widely marketed, often through social media. An industry analysis put global investment in longevity companies at around $8.5 billion in 2024 [4]; such estimates depend on which companies are counted and have not been independently verified.

The tension between the science and its marketing is not new. In 2002, more than 50 scientists working on ageing published a position statement separating the genuine science of ageing from what they described as a pseudoscientific anti-ageing industry, warning that products claiming to slow, stop or reverse human ageing had little or no scientific basis [5,6]. They also warned that entrepreneurs were drawing customers to "longevity" clinics and claiming a scientific basis for the anti-ageing products they recommended and often sold [6]. Two decades later, the science has advanced considerably, but the gap between what the evidence shows and what consumers are told appears to have persisted. Health misinformation is common on social

media, although estimates vary widely with topic, platform and definition: a systematic review found it in up to 87% of posts in some studies and in around 40% of posts on vaccines and chronic disease, and these figures do not refer to longevity content specifically [7]. False information also tends to spread further and faster online than accurate information [8].

The gap matters clinically, because patients increasingly arrive at consultations with biological age results, supplement regimens or protocols promoted online, and clinicians need a clear, balanced account of what is supported, what is plausible but unproven, and what is overstated.

Terms and scope

In this review, geroscience refers to the research field that studies the biology of ageing in order to delay age-related disease, and longevity medicine to the clinical application of that research to extend healthspan. "Anti-ageing" is used, as in the 2002 position statement [5], for products and services marketed to consumers as slowing, stopping or reversing ageing, whatever their evidence base. Wellness refers more broadly to products and services sold to support general health rather than to treat a diagnosed disease. Established preventive measures with outcome evidence, such as stopping smoking, regular physical activity and treatment of raised blood pressure, are outside the scope of this review.

This narrative review has three objectives. First, to summarise the human evidence in four areas prominent in both geroscience and longevity marketing: senolytics, nicotinamide adenine dinucleotide (NAD+) precursors, caloric restriction and epigenetic clocks. Second, to describe recurring ways in which commercial claims about them run ahead of that evidence, illustrated by cases with formal regulatory or legal findings. Third, to suggest how clinicians, researchers and regulators might respond. The review sets out to identify and illustrate a risk, not to measure it: it does not estimate how common overstated claims are, quantify the harm they cause, or argue for specific legal reform.

Why a narrative review

We chose a narrative rather than a systematic review for three reasons [9,10]. First, the central construct has no objective definition: there is no agreed or validated measure of “overselling” or of the gap between evidence and marketing claims, so it cannot be scored consistently across products, studies or promoters, and research on health misinformation faces the same problem of varying definitions and a lack of objective indicators [7].

Second, the evidence cannot be pooled: it spans randomised trials, observational cohorts, regulatory decisions, court judgments, advertising rulings and journalism, and no trials compare marketing claims with patient outcomes, so a meta-analysis is neither possible nor meaningful; even studies of misinformation alone are hard to compare because their definitions and methods differ [7]. Third, the aim is interpretive, in that the review links credible biomedical evidence to the commercial and social practices that draw on it, and narrative and critical reviews suit this kind of cross-disciplinary synthesis, where the goal is to build and test an argument rather than to estimate a single effect size [10].

Sources were identified through targeted, non-systematic literature and web searches carried out using an AI tool (Claude, Anthropic, San Francisco, CA, USA), rather than a database search with a predefined strategy. Regulatory records, court judgments and selected grey literature were used for the commercial cases. The author checked each cited source against the original publication or, where the full text was not accessible, its abstract, and statements were cited only where the source supported them. Selection was purposive. In reporting the evidence, randomised trials and systematic reviews are presented where they exist, early-phase and uncontrolled studies are identified as such, animal and mechanistic studies are used only as background, and named commercial cases are limited to those with a formal regulatory, legal or self-regulatory finding or a documented public retraction. No formal quality appraisal was performed; instead, the design, size and control group of each human study are reported so that readers can judge the strength of the evidence. This review should, therefore, be read as a reasoned synthesis rather than an exhaustive one; the resulting risk of selection bias is discussed under Limitations. The author scored the final manuscript against the SANRA scale for narrative reviews (Table *1*) [11]. This is a self-assessment, not an independent appraisal, and should be read as such. Scale for the Assessment of Narrative Review Articles (SANRA) has no established quality cut-offs: its developers suggest that a score of 4 or below indicates very poor quality, and in their validation sample of 30 submitted narrative reviews the mean score was 6.0 of 12, with accepted manuscripts averaging 6.6 and rejected ones 5.1 [11]. The scale assesses how transparently a review is reported rather than how its sources were selected, so a high score does not offset the selection bias discussed under Limitations.

SANRA item	Self-assessed score (0-2)	Basis
1. Justification of the article's importance for the readership	2	The Introduction sets out the clinical problem: patients arriving with longevity products, protocols and test results
2. Statement of concrete aims or formulation of questions	2	Three objectives and the boundaries of the review stated at the end of the Introduction
3. Description of the literature search	1	Search approach (non-systematic, AI-assisted) and source types described, but no database search, search terms or date limits
4. Referencing	1	Key statements referenced and each reference checked against its source or abstract, but some general statements remain uncited
5. Scientific reasoning (e.g., appropriate evidence level)	2	Design, size and control group stated for each human study; biomarkers distinguished from clinical outcomes throughout
6. Appropriate presentation of relevant endpoint data	2	Effect sizes and null results reported for key trials and summarised in tables
Total	10/12	

TABLE 1: Self-assessed SANRA score for this review

Scored by the author using the SANRA scale [11]; each item is scored 0-2, with a maximum of 12.

SANRA: Scale for the Assessment of Narrative Review Articles.

Key terms

Table *2* defines the technical terms used in this review in plain language.

Term	Plain-language meaning
Geroscience	The study of how the biology of ageing causes disease, aiming to delay many age-related diseases at once
Lifespan/healthspan	How long a person lives/how long they live in good health, free of serious disease or disability
Cellular senescence	A state in which a damaged cell stops dividing permanently but does not die
Senescent cell	A cell in that state; these accumulate with age and release inflammatory signals
SASP	Senescence-associated secretory phenotype: the mix of inflammatory and tissue-damaging molecules senescent cells release
Senolytic	A drug designed to kill senescent cells selectively, such as dasatinib plus quercetin
NAD+	Nicotinamide adenine dinucleotide, a molecule every cell needs for energy production and DNA repair; levels fall with age
NAD+ precursor (NR, NMN)	Supplements the body converts into NAD+: nicotinamide riboside and nicotinamide mononucleotide
Caloric restriction	Eating fewer calories than usual while still meeting nutritional needs
DNA methylation	Small chemical tags on DNA that switch genes on or off; the pattern changes with age
Epigenetic clock	A computer model that estimates age from DNA methylation patterns in a blood or saliva sample
Biological age	An estimate of how "old" the body appears on a given test, as opposed to years since birth
Biomarker	Something measured in the body that reflects a biological process, such as a blood level or a clock score
Surrogate endpoint	A biomarker used in place of a real health outcome because it is expected to predict that outcome
Clinical outcome	A direct measure of how a person feels, functions or survives, such as walking speed, disease or death
Randomised controlled trial	A study that assigns people by chance to a treatment or a comparison group, the most reliable test of whether a treatment works
Off-label prescribing	Prescribing a licensed drug for a purpose it is not licensed for, such as metformin for ageing
Structure/function claim	A US marketing claim about how a supplement affects the body (for example, "supports cellular energy") that does not need prior regulatory approval

TABLE 2: Plain-language glossary of key terms

Definitions of biomarker, surrogate endpoint and clinical outcome follow the FDA-NIH BEST glossary [12].

NAD+: nicotinamide adenine dinucleotide; NR: nicotinamide riboside; NMN: nicotinamide mononucleotide; SASP: senescence-associated secretory phenotype.

Review

Because the distinction between biomarkers and clinical outcomes underpins the whole review, we define the terms first.

Biomarkers versus clinical outcomes

A biomarker shows that something has changed in the body; a clinical outcome shows whether the person is better off. We use the definitions of the FDA-NIH BEST glossary, which states that a biomarker "is not a measure of how an individual feels, functions, or survives" [12].

The distinction matters because a biomarker can move in the right direction while patients do worse. In the Cardiac Arrhythmia Suppression Trial, patients whose ventricular ectopy after myocardial infarction had been suppressed with encainide or flecainide were more than twice as likely to die or have a cardiac arrest as those given placebo (relative risk 2.38) [13]. Torcetrapib raised high-density lipoprotein cholesterol by 72%, yet increased cardiovascular events by 25% and deaths by 58% [14], possibly through an off-target rise in blood pressure, a reminder that a drug can affect outcomes through pathways a surrogate does not capture [14,15].

A biomarker becomes a trustworthy surrogate only when trials show that changing it reliably changes the

outcome, and few have met this standard [15]. Biomarkers are useful in early-phase trials to show that a treatment is biologically active, but definitive trials need clinical outcomes [15]; most senolytic and NAD+ trials are still at this early stage. In longevity research, almost all human results sit at the biomarker level: higher NAD+, fewer senescence markers, a slower epigenetic clock. No ageing biomarker has yet been validated as a surrogate for healthy ageing. In this review, an intervention is described as beneficial only when a controlled human trial has shown an effect on a clinical outcome. Where no such effect has been shown, we distinguish interventions that have not yet been adequately tested from those that have been tested and showed no benefit; the first is an absence of evidence, not evidence that an intervention is ineffective.

Senolytics

Cellular senescence is a state in which a cell permanently stops dividing but resists dying. It is triggered by stresses such as DNA damage, dysfunctional telomeres or activation of cancer-promoting genes, and it protects against cancer by halting damaged cells before they become malignant [16]. Senescent cells secrete inflammatory cytokines, growth factors and tissue-degrading enzymes, known as the senescence-associated secretory phenotype (SASP). They accumulate with age and at sites of age-related disease such as osteoarthritis and atherosclerosis, for reasons that are not fully established, and their secretions are thought to contribute to tissue dysfunction [16]. Senescent cells are reported to be two to 20 times more common in people over 65 than in people under 35 [1], although no marker is exclusive to senescent cells and estimates of their abundance in aged tissue range from under 1% to over 15% [16].

Senolytics are drugs that selectively kill senescent cells. In mice, transplanting senescent cells causes physical dysfunction, and clearing them with dasatinib plus quercetin (D+Q) improves physical function and extends remaining lifespan [17], findings that prompted rapid translation into humans.

Early human studies are encouraging but small and uncontrolled. In 14 patients with idiopathic pulmonary fibrosis, three weeks of intermittent D+Q improved six-minute walk distance by 21.5 m, but lung function and quality of life were unchanged; the authors cautioned that without a control group the results must be interpreted with caution [18]. In nine patients with diabetic kidney disease, a three-day course reduced senescent cell markers in fat tissue by 17% to 62% [19]. In five patients with early Alzheimer's disease, dasatinib reached the cerebrospinal fluid at under 1% of blood levels, quercetin was undetectable there, cognition did not change, and two cerebrospinal fluid markers of inflammation and astrocyte activation (IL-6 and GFAP) rose rather than fell [20].

A 2025 summary counted only nine published clinical trials of senolytics, just two with a control group, against 58 registered ongoing studies; it concluded that the trials suggest possible improvements in biomarkers of healthspan, with no significant safety concerns [21]. A commentary by the same group noted that clear evidence of efficacy in humans is still lacking and argued for a more personalised approach to future trials [22].

Two points matter for clinicians. First, dasatinib is a prescription tyrosine kinase inhibitor used in leukaemia; patients with leukaemia take it daily for years with tolerable side effects, and serious effects usually reverse when it is stopped [19], but it has known toxicities with continuous use, including pulmonary oedema, and the researchers who first reported D+Q in old mice stressed that toxicity studies were needed before trials in relatively healthy humans [17]. Second, quercetin and fisetin are freely sold as supplements, so the trial regimens can be partly reproduced without medical oversight.

NAD+ precursors

NAD+ is an essential coenzyme in energy metabolism and DNA repair, and tissue levels appear to fall with age. In animal models, restoring NAD+ with precursors such as nicotinamide riboside (NR) and nicotinamide mononucleotide (NMN) improves several features of ageing [23].

Human trials consistently show that oral NR and NMN raise blood NAD+ levels and are well tolerated in the short term [24,25]. A randomised trial in 30 healthy adults found NMN safe over 12 weeks and effective at raising blood NAD+ [25]. In a small randomised trial in postmenopausal women with prediabetes, NMN improved muscle insulin sensitivity [26]. Trials of NR at up to 2,000 mg/day for six to 12 weeks also raised NAD+ metabolism without serious adverse events [27,28].

What has not been shown is equally important: most human trials have been short, many focused only on bioavailability and safety, and any effect on human healthspan remains unestablished [23]. Table *3* summarises trials that measured physiological or functional effects beyond NAD+ levels.

Trial	Population, dose and duration	Physiological and functional outcomes measured	Result
Martens et al., 2018 [27]	Healthy adults aged 55-79; NR 1,000 mg/day for six weeks (crossover)	Blood pressure, arterial stiffness, physical function	No change in physical function or exercise capacity; non-significant trends towards lower systolic blood pressure (about -4 mmHg) and arterial stiffness, mainly in those with raised baseline blood pressure; 24 of 30 completed
Dollerup et al., 2018 [28]	Forty men with obesity and insulin resistance; NR 2,000 mg/day for 12 weeks	Insulin sensitivity, body composition, liver fat	No improvement on any measure
Yoshino et al., 2021 [26]	Twenty-five postmenopausal women with prediabetes; NMN 250 mg/day for 10 weeks	Muscle insulin sensitivity, body composition, blood pressure, lipids	Muscle insulin sensitivity rose 25% (a biomarker); no change in muscle NAD+, muscle strength or fatigue, fasting glucose, liver fat, body composition or blood pressure
Igarashi et al., 2022 [29]	Forty-two healthy men aged 65 and over; NMN 250 mg/day for 12 weeks; 12-week data for only 20 after a dispensing error	Muscle mass (primary), gait speed, grip strength, cognition, metabolic markers	Muscle mass unchanged; nominal improvements in gait speed and left-hand grip only, uncorrected for multiple testing; no effect on cognition, insulin sensitivity or liver fat
Brakedal et al., 2022 (NADPARK) [30]	Thirty people with newly diagnosed Parkinson's disease; NR 1,000 mg/day for 30 days	Brain NAD+ metabolism, disease severity score	Brain NAD+ rose in most participants; no significant change in disease severity score; a non-significant trend towards improvement only in the subgroup whose brain NAD+ rose
Prokopidis et al., 2025 (meta-analysis) [31]	10 randomised trials (six NMN, four NR) in adults with mean age 61-83	Muscle mass, grip strength, gait speed, chair stands	No significant effect of NMN on any pooled measure; NR trials too few to pool, with mixed results

TABLE 3: Human trials of NAD+ precursors that measured effects beyond blood NAD+ levels

All five trials were randomised and placebo-controlled; the final row summarises a meta-analysis rather than a single trial.

NAD+: nicotinamide adenine dinucleotide; NR: nicotinamide riboside; NMN: nicotinamide mononucleotide.

Across these trials, functional benefits have been small, inconsistent or absent. Most were designed around safety and NAD+ levels, with functional measures as secondary or exploratory endpoints. Even the most positive single result, improved muscle insulin sensitivity with NMN, is a biomarker change: muscle strength and fatigue, the trial's functional measures, did not change [26], and insulin sensitivity did not improve with NR in a larger trial [28]. Much of this early evidence comes from trials sponsored by, or run with employees of, the companies that make the products [24,25,29]; such sponsorship does not invalidate the findings, but these trials mainly measured safety and NAD+ levels, while an investigator-initiated trial found no metabolic benefit [28]. A 2025 meta-analysis concluded that current evidence does not support NMN or NR for preserving muscle mass or function in older adults [31]. Larger trials with functional primary outcomes are underway, including a one-year placebo-controlled trial of NR 2,000 mg/day in 100 frail adults aged 75 and over, with gait speed as its primary outcome and primary completion expected in 2027 [32].

Caloric restriction

Caloric restriction, eating fewer calories than usual while still meeting nutritional needs, extends lifespan in many, though not all, animal models, and is also the most rigorously tested longevity intervention in humans: CALERIE phase 2 is the largest and most systematic randomised trial of prolonged caloric restriction in healthy, non-obese adults [33,34]. Most trials of supplements and senolytics, by contrast, have lasted weeks, enrolled tens of people and often lacked a control group.

CALERIE randomised 218 adults aged 21 to 50 to two years of caloric restriction or their usual diet [33]. Participants aimed for a 25% reduction in energy intake but achieved about 12% on average. Caloric restriction improved several cardiometabolic risk factors, including blood pressure, low-density lipoprotein (LDL) cholesterol and insulin sensitivity, without adverse effects on psychological outcomes [33,34]. It was not free of harm: restriction caused small losses of bone mineral density and some cases of anaemia, and the

investigators advise clinical monitoring for anyone practising it [33]. The trial's primary outcomes, changes in resting metabolic rate and core body temperature, were largely not met [33].

Secondary analyses suggest effects on ageing biology itself. In a post hoc analysis, caloric restriction slowed the pace of ageing by roughly 2% to 3% as measured by the DunedinPACE epigenetic algorithm, a small effect, with no change on other clocks such as PhenoAge or GrimAge [35]. After 12 months, caloric restriction also lowered nine of 28 circulating proteins associated with cellular senescence, although, as the authors note, none of these markers specifically identifies senescent cells [36].

These are the strongest randomised data in the field, but they show changes in risk factors, not in outcomes. The trial could not have shown fewer heart attacks, cancers or deaths: its participants were young, healthy and at low absolute risk, so such events would have been rare in both groups over two years. The investigators themselves wrote that whether caloric restriction extends human lifespan "will probably never be determined in randomized clinical trials", and that what the risk-factor changes mean for disease incidence in healthy people has not yet been determined [33]. Lower blood pressure and LDL cholesterol are established markers of lower cardiovascular risk, so the findings are encouraging for cardiovascular health, but they do not show that caloric restriction slows ageing as a whole. Caloric restriction also has costs: in humans it has not improved muscle or bone function, and in mice it can impair immunity and wound healing [3]. Sustaining substantial restriction for decades is unrealistic for most people, and some researchers remain sceptical that moderate caloric restriction would meaningfully change human ageing over a feasible timeframe.

Biological age and epigenetic clocks

Epigenetic clocks are machine-learning models that estimate age from DNA methylation patterns [37]. Later-generation measures were developed to track health rather than chronological age. DunedinPACE, for example, was trained on the rate of decline in 19 biomarkers measured over two decades, and its associations with morbidity, disability and mortality are similar to those of GrimAge and stronger than those of first-generation clocks [38]. At the population level, these tools have been valuable for studying how environment, behaviour and interventions relate to ageing. The field is young: as recently as 2014 it was unclear whether methylation markers tracked chronological or biological age [2], and in 2002 ageing scientists warned that claims to measure a person's "biological age" should be regarded as entertainment, not science [5].

A clock result is produced in five steps. A sample is collected, usually blood, although consumer tests often use saliva, a finger-prick blood spot or a cheek swab posted to a laboratory. DNA is then extracted and treated with bisulfite, which converts unmethylated cytosines so that methylated and unmethylated sites can be told apart. A methylation microarray reads hundreds of thousands of specific sites in the genome (CpG sites), giving each a value from 0 (unmethylated) to 1 (fully methylated). A statistical model trained on data from thousands of people combines a selected subset of these sites, 353 in Horvath’s original clock, into an age estimate or a pace-of-ageing score [37]. Finally, the estimate is compared with chronological age, and a higher estimate is reported as an “older” biological age.

Why clocks work for populations but not individuals. Every step adds error: technical noise between repeat measurements of the same sample can shift some clock estimates by up to nine years [39], and results also vary with the tissue sampled and with the proportions of cell types in it, and adjusting for cell-type proportions weakens some clocks’ associations with mortality, particularly those built from smaller training sets [40]. In a study of thousands of people, these random errors largely cancel out, so small average differences between groups become visible: smokers and people living with socioeconomic disadvantage, for example, tend on average to have older clock estimates. In one person, however, the error does not cancel, and a single result carries a margin of error larger than the effect of any intervention tested so far: caloric restriction in CALERIE changed DunedinPACE by about 2% to 3% on average, and the two groups' trajectories overlapped by about 90% [35]. The situation resembles blood pressure: population averages reliably predict stroke rates, but a single reading in one person says much less. Clocks are also trained to predict population averages, not individual futures, so even a perfectly precise measurement would carry wide uncertainty about one person's health. An expert review of the field listed a clock's accuracy within an individual as unknown [40].

Different clocks also often disagree for the same person; in CALERIE, caloric restriction moved one clock but not two others [35]. A 2025 analysis, whose authors declared no commercial interests, concluded that clocks fail common standards for clinical utility, that using them for individual decisions can be uninformative and potentially harmful, and that they should not currently be used for such decisions even if the technical problems were solved [41]. Consensus work on biomarkers of ageing has called for validation against outcomes before such measures are used as surrogate endpoints [42]. Validated surrogates exist in other fields, such as blood pressure reduction for stroke, but no ageing biomarker of any category has been approved by US regulators for clinical use [42]. The same work notes that a high biological age could reflect faster ageing earlier in life rather than now, so a single result cannot tell someone whether their current habits are working [42].

Summary of the evidence

Table 4 summarises the human evidence across the four areas, separating what has been shown, mostly at the biomarker level, from what has not, and sets out how far typical commercial claims are supported.

	Senolytics	NAD+ precursors	Caloric restriction	Epigenetic clocks
Proposed mechanism	Clear senescent cells and their inflammatory secretions [16,17]	Restore NAD+ levels that fall with age [23]	Reduced energy intake acting on nutrient-sensing pathways [1]	Not an intervention; estimates age from DNA methylation [37]
Best human evidence	Small pilot and phase 1 trials; 2 of 9 controlled [18-21]	Small RCTs, mostly 4-12 weeks [24-30]	CALERIE 2 RCT, 218 adults, 2 years [33]	Large cohort studies [38]
Shown in humans (mostly biomarkers)	Short-term tolerability; biomarker changes [18-21]	Raise blood NAD+; short-term safety [24,25,27,28]	Cardiometabolic improvements; modest biomarker slowing [33-36]	Population-level association with morbidity and mortality [38]
Not yet shown in humans (clinical outcomes)	Clinical efficacy; any healthspan or lifespan effect [21,22]	Functional or disease benefit; healthspan or lifespan effect [23,31]	Disease or survival benefit; long-term feasibility [33]	Reliability for individual decisions; validity as a trial surrogate [39-42]
Principal limitations	Small samples; few controlled trials; short follow-up [21]	Short, small trials, several industry-sponsored [23-25,29]	Young, healthy volunteers; 12% restriction achieved against a 25% target [33]	Technical noise; clocks disagree; unknown accuracy in individuals [35,39,40]
Support for anti-ageing claims	Claims of clinical benefit not supported: no clinical efficacy shown [21,22]	Claims to reverse metabolic ageing [26] not supported: no functional benefit in pooled trials [31]	Claims of slower ageing partly supported: risk factors improve, but no evidence of slower whole-body ageing [33,35]	Claims to measure a person's "real age" [5] not supported for individual use [41]

TABLE 4: Summary of the human evidence and commercial claims in the four areas reviewed

RCT: randomised controlled trial; NAD+: nicotinamide adenine dinucleotide.

How commercial claims outrun the evidence

The gap between evidence and marketing is not random: five recurring patterns account for most of it, each taking a real scientific finding and stretching it beyond what the data support.

Extrapolation From Animal and Laboratory Models

Most striking longevity findings come from mice, worms, yeast or cell culture. Marketing often presents these as if they applied directly to humans, describing a compound as "proven to reverse ageing" when the evidence is a rodent lifespan study. Doses used in animal work frequently scale to amounts far above those in consumer products, and mouse models often identify treatments that then fail in humans [2]. Much of the lifespan evidence for NAD+ precursors comes from models of rare premature-ageing disorders rather than normal ageing, including a worm study reporting a 117% increase in lifespan [23]. The senolytic literature described above shows the same gap between animal and human evidence [21].

Surrogate Biomarkers Presented as Outcomes

A rise in blood NAD+ or a fall in an epigenetic age estimate is a biomarker change, not a clinical outcome, and neither marker has been validated as a surrogate for healthy ageing. Commercial offers often link the two directly: a biological age test identifies an "older" result, a supplement or protocol is recommended, and a repeat test shows "improvement". Given the technical noise and inter-clock disagreement described earlier, such a test-treat-retest loop can show apparent benefit from measurement variation alone [39,41]. It also gives consumers a number that feels objective, which makes the claim harder to question.

The same drift appears in the research literature itself. When the first randomised trial of NMN's metabolic effects was published in 2021, its authors noted that NMN was already marketed to improve glucose control

and "reverse the metabolic complications of aging", although they knew of no human studies of its metabolic effects [26]. The NADPARK report's highlights stated that "cerebral NAD increase is associated with clinical improvement", although disease severity scores did not change significantly [30], and the abstract of the Igarashi trial described NMN as a booster "for preventing aging-related muscle dysfunctions" despite a null primary outcome [29].

Rebranding and Borrowed Vocabulary

Commentators have noted that much of the industry has replaced "anti-ageing" with "longevity", a term that sounds more scientific and modest while the underlying promises are unchanged [43]. Terms from geroscience, such as "senolytic", "hallmarks of ageing" and "cellular rejuvenation", are used to describe products that have never been tested against those mechanisms in humans. Borrowed vocabulary lends a product the credibility of the research field without its evidence.

Influencer-Led and Conflicted Communication

The internet, including social media, has become a popular source of health information, and on platforms such as YouTube, lower-quality health content often attracts more engagement than accurate content [7,8]. Popularity online is driven mainly by broadcasts from large accounts rather than person-to-person sharing, so high-follower accounts carry particular responsibility [8]. Longevity content is often produced by people who sell, co-own, or are paid to promote the products they discuss, and these interests are not always clear to the audience. Medical credentials can add authority to claims that go well beyond the evidence, and credentialed people who stoke unfounded fears are among the most damaging spreaders of misinformation [8]. A newer development is the use of AI-generated personas, some presented as doctors, to promote supplements; a 2026 investigation reportedly found hundreds of such accounts, and US advertising self-regulators have stressed that the same truth-in-advertising standards apply whether a spokesperson is human or AI-generated [44].

Financial ties also run through the research literature. Several early NAD+ precursor trials were sponsored by, or included employees of, the manufacturers [24,25,29]; other authors hold patents or licensing income related to the products studied [26,30]; and the developers of DunedinPACE and of more reliable versions of other clocks have licensed them commercially [35,38,39]. These ties are declared, and the work is peer reviewed, which is the difference from most commercial promotion, but they are a reason to read headline conclusions with care.

Clinics, Testing and Off-Label Prescribing

Private longevity clinics typically combine extensive testing with ongoing membership fees. Testing people without symptoms finds abnormalities that would never have caused harm, and labelling them can have lifelong effects on wellbeing, insurability and health [45]. Screening also has a self-reinforcing appeal: the more overdiagnosis it causes, the more people feel they owe it their life, and the more popular it becomes [45]. Some services also prescribe drugs off-label for ageing, such as metformin or rapamycin. These drugs have plausible rationale and are being studied properly: the TAME trial of metformin was designed specifically to test whether a drug can delay multiple age-related diseases [46], and a six-week course of the related drug everolimus improved influenza vaccine antibody responses in older adults [47]. Neither is risk-free: in a diabetes prevention trial, metformin was associated with mild anaemia and vitamin B12 deficiency [46], and treatment-related adverse events, mostly mouth ulcers, occurred in 38% to 51% of everolimus recipients versus 20% on placebo [47]. Prescribing these drugs routinely to healthy people before such trials report moves the evidence burden onto the patient.

Named Cases

Regulators, courts and advertising bodies have acted against a number of longevity and wellness claims, and these cases (Table *5*) show the patterns above in practice. We restrict named examples to cases with a formal regulatory, legal or self-regulatory finding, or a documented public retraction, and report only what those findings or reports state.

Case	Year	What was claimed or sold	Finding or outcome
Tru Niagen (Niagen Bioscience), US	2026	NR supplement advertised as raising NAD+ “up to 150%”, with “clinically proven” ageing, vitality and organ-health benefits [48]	US National Advertising Review Board recommended discontinuing “clinically proven”, anti-ageing, vitality and organ-health claims, finding that the cited studies did not show the NAD+ increases applied to the general population, and rejected the defence that these were permissible structure/function claims; the company said it would comply [48]
Get A Drip, UK	2023	Intravenous vitamin drips, including an “Anti-Ageing Drip” said to “help prevent premature ageing”; courses priced up to £3,000 [49]	Advertising Standards Authority upheld complaints that the health-benefit claims, including anti-ageing claims, were misleading and unsubstantiated, and told the company not to make health claims until it held evidence from normally healthy people [49]
US Stem Cell Clinic, US	2019	Unapproved stem cell products made from patients’ fat tissue, given for serious conditions including Parkinson’s disease, ALS, COPD and heart disease [50]	Federal court granted the government summary judgment, holding that the clinic and its chief scientific officer had adulterated and misbranded the product; the FDA had earlier cited failures to prevent microbial contamination [50]
Ambrosia, US	2019	Infusions of plasma from donors aged 16-25, reported at $8,000 per litre, promoted against ageing and age-related disease [51]	FDA warned that young-donor plasma had no proven clinical benefit for ageing and carried transfusion risks; the company announced the same day that it had stopped treatments [51]
Telomerase Activation Sciences (TA-65), US	2018	Supplements and creams claimed to reverse ageing, repair DNA, restore ageing immune systems and reduce cancer risk [52]	FTC settlement order barred the unsupported claims; the FTC also alleged that endorsers given free product worth up to $4,000 were presented as independent users, and that a paid TV segment was presented as independent programming [52]
Belle Gibson (The Whole Pantry), Australia	2017	Wellness app and cookbook built on her claim to have treated brain cancer through diet and alternative therapy [8,53]	Federal Court found misleading and deceptive, and unconscionable, conduct over promised charity donations and imposed penalties totalling A$410,000; she had earlier admitted she never had cancer [53]

TABLE 5: Named longevity and wellness cases with a formal finding or documented public retraction

Each case is reported only as described in the cited finding or report.

NR: nicotinamide riboside; NAD+: nicotinamide adenine dinucleotide; ALS: amyotrophic lateral sclerosis; COPD: chronic obstructive pulmonary disease; FDA: US Food and Drug Administration; FTC: US Federal Trade Commission.

Three features recur across these cases. First, mechanistic or animal data were presented as proof of human benefit, as with young plasma, telomerase activation and NAD+ levels. Second, endorsements, testimonials and clinical anecdote stood in for trials, sometimes without disclosure of payment; in the Get A Drip case, the supplier's own training manual stated that evidence of efficacy was "based mainly on anecdotal clinical experience" [49]. Third, enforcement came late and was narrow: most actions addressed specific wording after sales had occurred, and similar products often remain on sale under revised claims. Formal findings are rare relative to the volume of marketing, which is why the general patterns matter more than any single case. Unregulated invasive procedures can also cause serious harm: intravitreal injections of autologous "stem cells" at a US clinic left three patients with severe vision loss [54].

Summary of patterns

Table *6* summarises the five patterns, the typical claim associated with each, and what the evidence actually supports.

Pattern	Typical claim	What the evidence supports
Animal extrapolation	"Reverses ageing" [52]	Lifespan or healthspan effects in model organisms only[2,17,23]
Surrogate as outcome	"Lowers your biological age by 5 years"	Change in a noisy biomarker of uncertain clinical meaning[39-42]
Rebranding	"Evidence-based longevity protocol"	Same unproven products under a new label[43]
Conflicted promotion	"What I take every day"	Personal testimony from someone with a financial interest[8,52]
Clinic and testing model	"Catch disease before it starts"	Unclear benefit of broad screening in healthy people; risk of overdiagnosis [45]

TABLE 6: Recurring patterns by which commercial claims exceed the evidence

Except where cited, typical claims are illustrative paraphrases of common marketing language, not quotations from specific products.

Regulation, clinical implications and recommendations

Why the Gap Persists: Regulatory Structure

Most longevity products avoid medicines regulation because they are sold as food supplements or wellness services. In the United States, the Dietary Supplement Health and Education Act 1994 allows supplements to be marketed without pre-market approval, places the burden of proving that a supplement is unsafe on the government, and permits claims about effects on body structure or function, rather than disease, without FDA evaluation [55]. In the United Kingdom, supplements are regulated as foods; health claims must appear on the Great Britain nutrition and health claims register, and advertising is governed by the CAP Code and enforced by the Advertising Standards Authority [56,57]. These rules apply online, but enforcement struggles with the volume of influencer content, cross-border sales and claims made in podcasts or videos rather than formal advertisements.

A further structural problem is that ageing itself is not a recognised indication for treatment or target for drug development [1]. There is therefore no licensed pathway for an "anti-ageing" drug, which pushes interest towards supplements, off-label prescribing and unregulated services. Biological age tests may be regulated as laboratory products for their analytical performance, but the clinical interpretation offered to consumers usually falls outside any formal oversight.

Clinical and Public Health Implications

The harms of overstated longevity claims fall on individuals, on health services and on public trust. Table 7 summarises them; the paragraphs that follow expand on the most important.

Harm	Mechanism	Example	Who bears it
Physical harm	Adverse effects, contamination, drug interactions, invasive procedures	About 23,000 US emergency visits a year linked to supplements (2004-2013) [58]; vision loss after unregulated stem-cell injections [54]	Individual patients
Financial harm	Memberships, repeat testing, IV drips, multi-product regimens	IV drip courses up to £3,000 [49]; plasma infusions at $8,000 per litre [51]	Individuals, often older adults fearful of decline
Psychological harm	Anxiety from an “older” biological age; false reassurance from a “younger” one	Individual clock results that are uninformative and potentially harmful [41]	Individuals
Opportunity cost	Money and attention diverted from proven prevention	Supplements in place of exercise, smoking cessation or blood pressure control	Individuals and population health
Overdiagnosis and cascades	Broad screening of healthy people finds incidental abnormalities	Follow-up scans, referrals and procedures [45]	Patients and health services
Displaced workload	Private tests advise follow-up with the GP	NHS appointments to interpret private results [59]	NHS primary care
Inequity	Premium services for those who can pay; proven prevention underused elsewhere	Two-tier “longevity” market	Disadvantaged groups
Erosion of trust	Overclaims by credentialed promoters and in research reports	Headline claims that outrun the data, including in peer-reviewed papers [29,30]	Legitimate geroscience and the medical profession

TABLE 7: Harms of overstated longevity claims

Examples without a citation are illustrative.

IV: intravenous; GP: general practitioner; NHS: National Health Service.

Harm to Individuals

Supplements were linked to an estimated 23,000 US emergency department visits a year in 2004-2013, mostly from weight-loss and energy products and from swallowing problems with large pills in older adults, who were also more likely to be admitted; the figure is likely an underestimate [58]. Products may be adulterated or mislabelled, with more than 500 found to contain pharmaceuticals or their analogues, and doctors often know little about interactions between supplements and prescribed drugs [58,60]. Post-market safety monitoring is weak: when one supplement caused a cluster of acute hepatitis, eventually 97 cases with three liver transplants and one death, the FDA learned of it about four months after the first cases [60]. Prescription drugs sourced outside medical care carry known toxicities: dasatinib can cause pulmonary oedema with prolonged use [17], and rapamycin is an immunosuppressant. NAD+ precursors raise theoretical concerns too: NAD+ metabolism can promote or suppress cancer depending on context, some metabolites may be neurotoxic, and high-dose nicotinic acid can damage the liver [23]. Invasive procedures sold for ageing, such as plasma infusions and unapproved cell therapies, add procedural and infection risk. Psychological harm is less visible but real: a test reporting an "older" biological age can cause anxiety, while a "younger" result may falsely reassure someone who should be addressing a modifiable risk.

Harm to Health Services

Broad screening of healthy people generates incidental findings and further investigations, with effects on wellbeing [45]. In the United Kingdom, much of this follow-up falls on the NHS. Private and direct-to-consumer tests commonly advise customers to discuss results with their general practitioner (GP). The Royal College of General Practitioners has stated that organisations offering screening should not assume GPs will deal with the results and should organise and fund follow-up themselves, citing a 2018 poll in which 91% of 500 doctors had discussed private screening results with a patient at an NHS appointment [59]. Time spent interpreting or countering longevity claims is time taken from patients with clinical need.

Harm to Equity, Trust and Research

Longevity services are concentrated among people who can pay, while cheap, proven prevention remains underused in disadvantaged groups; a two-tier market risks widening existing gaps in healthy life expectancy. Overclaims by credentialed promoters blur the line between research and marketing, and when promises fail they may erode public trust in legitimate geroscience and in doctors. False information tends to spread further and faster than accurate information, although clear corrections from trusted sources do reduce belief in it [8]. Widespread self-supplementation complicates research too: one current trial of NR excludes anyone who has taken NAD+ precursor supplements, including vitamin B3, in the previous six months [32].

Recommendations

For clinicians, the first step is to ask about supplements, longevity protocols and biological age tests routinely and without judgement, because patients often do not volunteer them. Clinicians can explain the difference between a change in a biomarker and a change in health outcomes, using the patient's own results where helpful, and should check for interactions and adverse effects and report suspected reactions through national schemes such as the Medicines and Healthcare products Regulatory Agency (MHRA) Yellow Card scheme. Advice is best anchored in interventions with strong evidence, framed positively as the core of longevity medicine rather than its boring alternative. Clinicians can also correct longevity misinformation, in consultations and in public: health professionals are among the most trusted sources of health information, and clear corrections from trusted sources reduce belief in false claims [8]. Doctors who promote or endorse products or services, including on social media, must be open and honest about any interests that may influence their recommendations, under General Medical Council guidance [61].

Researchers and journals should avoid phrases such as "reverses ageing" in abstracts and press releases unless they are supported by human outcome data. Reports should state clearly whether results come from animals or humans, and whether endpoints are biomarkers or clinical outcomes. Future trials should be controlled and have pre-registered clinical endpoints, and ageing biomarkers should be validated against outcomes before they are used as surrogates.

Regulators and platforms could consider applying health claims rules consistently to influencer and podcast content, not only to formal advertising, requiring clear labelling of paid promotion and of AI-generated personas giving health advice, and requiring direct-to-consumer biological age tests to state their measurement uncertainty and the absence of validated clinical use.

Limitations

Selection bias is the main weakness of this review, and a recognised criticism of narrative reviews in general [9,10]. Sources were chosen purposively, without a database search, predefined search strategy, inclusion criteria or risk-of-bias assessment, so relevant studies, including positive ones, may have been missed, and the choice of evidence may reflect the author's view that commercial claims exceed the data. To allow readers to judge this, the most favourable human result in each area is reported alongside the null ones (the improvement in walking distance with senolytics, improved muscle insulin sensitivity and nominal gains in gait speed and grip strength with NMN, the CALERIE effects on cardiometabolic risk factors and ageing biomarkers, and the population-level predictive value of later-generation clocks), and named commercial cases are restricted to those with formal regulatory, legal or self-regulatory findings.

Evidence on the commercial landscape relies partly on grey literature and journalism, which is less rigorous than peer-reviewed data, and the regulatory evidence is drawn mainly from US sources, some of which predate recent reforms. The field is moving quickly, and ongoing trials may change the evidence summarised here. The patterns described here should be treated as hypotheses to be tested: systematic reviews with prespecified protocols, for example of the functional effects of NAD+ precursors or of the accuracy of consumer longevity claims, would show whether they hold across the wider literature and market.

Conclusions

Longevity medicine is built on real science, but the human evidence for its best-known interventions remains early, and its strength differs between them. Caloric restriction has randomised evidence of improved cardiometabolic risk factors; NAD+ precursors appear safe in the short term but have not shown consistent functional benefit; senolytics remain experimental; and epigenetic clocks are useful research tools that are not yet suitable for individual decisions. None has yet shown benefit on disease, function or survival in humans, which for most reflects an absence of evidence rather than proof that they do not work. Commercial messaging, nonetheless often overstates this position through animal extrapolation, surrogate biomarkers, rebranded vocabulary, conflicted promotion and testing-led business models. Because the evidence was selected purposively, these conclusions describe recurring patterns rather than how common overstatement is across the market, and they do not imply that every longevity product or service is equally unsupported.

Clinicians are well placed to close this gap: by asking about longevity products, explaining what the evidence

does and does not show, and anchoring advice in proven prevention, they can protect patients without dismissing a legitimate field. Researchers, journals and regulators share responsibility for ensuring that the language used to describe geroscience matches the strength of its evidence.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Siddharth Kotikalapudi

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