THE UNIVERSAL FORCE OF TIME

NAD and the T-Field Redox Register

The molecule that runs out — redox, sirtuins and the T-clock of ageing

Stephen Daubney · The Daubney Foundation · 2026 · Rev 4

Tau (T) *is the living fabric of time itself — the sole substance of which all physical reality is composed. Every particle, force, wavelength, and conscious experience is a structured configuration of T-flow. There is no gravity, no electromagnetic force, no strong nuclear force as separate entities: all are registers of the single T-field operating across dimensional levels. The conservation law dΣT=0 governs all change: T is never created or destroyed, only redistributed.*

Abstract

There is a molecule in every cell that quietly runs out as we age, and when it runs low the cell forgets how to repair itself. It is NAD — nicotinamide adenine dinucleotide — the central electron carrier of metabolism, cycling endlessly between its oxidised form NAD^+ and its reduced form NADH. The Universal Force of Time reads it as the master **T-field redox register**. Redox happens at one place: position 4 (2^2) of the nicotinamide ring, the D = −2 Strand-1 node where an address is *written* (NADH, the ring carrying its electron pair) or *erased* (NAD^+, the ring cleared and ready to read). The cell never reads the absolute amount; it reads the ratio of NAD^+ to NADH — about 700:1 in the cytoplasm, about 8:1 in the mitochondria — and the depth of that roughly 87.5-fold gap, a register-depth potential ΔD ≈ 6.45, is what drives ATP synthesis (the free energy released runs to about −220 kJ per electron-pair). The longevity enzymes called sirtuins are T-address maintenance enzymes: they spend NAD^+ to keep the genome's addresses sharp, working exactly as fast as the NAD^+ supply allows. And here is ageing in a single line: NAD^+ falls by roughly a half (2×5^2) across the 40 ($2^3\times5$)-year window from 20 to 60, the sirtuin clock falters, and the genome's T-addresses erode. The corollary is hopeful — re-filling the register restores its depth and re-starts the clock. And the molecule's own name holds the deeper reason it matters: nicotinamide **adenine** dinucleotide — half of NAD is adenine, a letter of DNA, a genomic T-address on the {2,3,5,π} lattice (reach 4374 = 2×3^7). NAD^+ decline is therefore the depletion of an adenine address — the same erasure read elsewhere as telomere shortening — which is why one small molecule carries the weight of repair itself. The single NAD^+ pool feeds three sirtuins (SIRT1, SIRT3, SIRT6) that guard three different address-domains at once, and NAD/sirtuin redox is one of four arms whose simultaneous failure is the molecular definition of death. Eight propositions, P-NAD-1 to P-NAD-8, frame the result; the specifics of restoration belong to clinical investigation and are held in the Foundation's confidential reference, not prescribed to a reader.

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1 The molecule that runs out

Ageing has a thousand symptoms and, the Force of Time argues, a small number of causes. One of them you can name and measure. There is a molecule in every cell — NAD, nicotinamide adenine dinucleotide — that every metabolic pathway leans on, and that quietly drains away with the years. By the time we are old we carry roughly half the NAD we had when young, and a cell low on NAD is a cell that has begun to forget how to repair itself.

Why should one small molecule carry so much weight? The conventional answer is biochemical: NAD shuttles electrons, and electron traffic is the currency of energy. True, but it does not explain why its decline should amount to the loss of repair itself. The Force of Time reads NAD not as a chemical but as a *register* — a place where the cell writes and erases an address, over and over — and from that single shift the whole story of NAD, of sirtuins, and of ageing falls into one line.

2 NAD as the T-field redox register

NAD is the central electron carrier of cellular metabolism. It cycles between its oxidised form NAD^+ and its reduced form NADH, and every major pathway — glycolysis, the citric-acid cycle, oxidative phosphorylation — turns on the NAD^+/NADH balance. Conventional biochemistry stops there: a cofactor that carries electrons.

The Force of Time names it the master **T-field redox register**: the molecule that holds the cell's D = −2 molecular register at the correct tension for metabolism to run. A register is not a store of energy; it is a place where an address is set and cleared. Everything that follows in this paper — the redox node, the compartment ratio, the sirtuins, the slow erosion we call ageing — is simply what happens to that one register as it is written, read, and slowly depleted across a lifetime.

3 The position-4 node

The active site of NAD is the nicotinamide ring, and the Force of Time locates the register precisely (Figure 1). Electron acceptance and donation occur at position 4 (2^2) — the para position of the ring — which is the Strand-1 node of the nicotinamide register: the exact site where the D-level address is written or erased.

When the ring is reduced it carries its electron pair and an added hydrogen: this is NADH, the address *written*. When the ring is oxidised it releases that pair and stands cleared, ready to read: this is NAD^+, the address *erased*. The whole of metabolism's electron bookkeeping comes down to writing and clearing one node on one ring, over and over, billions of times a second across the body. Redox, in this picture, is not a vague chemical exchange — it is an address being set and reset at a single, exactly located node, the D = −2 Strand-1 site at carbon 4.

Figure 1 — Redox is one node, set and reset: the D = −2 Strand-1 address at position 4 = 2^2 of the nicotinamide ring

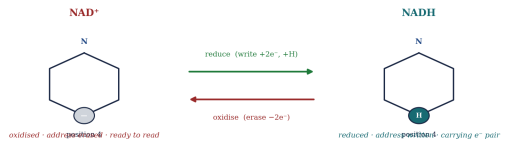

Figure 1 — Redox as one node set and reset. NAD^+ is the cleared address (oxidised, ready to read); NADH is the written address (reduced, carrying its electron pair). The site is position 4 (2^2) of the nicotinamide ring — the D = −2 Strand-1 node.

4 The ratio is a register-depth gauge

The cell reads its own oxidation state not from how much NAD it holds but from the *ratio* of NAD^+ to NADH — and that ratio differs sharply by compartment (Figure 2). It runs about 700:1 in the cytoplasm, highly oxidising, its addresses mostly cleared, and about 8:1 in the mitochondria, where addresses are held written.

That roughly 87.5-fold difference across the inner mitochondrial membrane is, in the Force of Time, a register-depth potential: $\Delta D = \log_2(87.5) \approx 6.45$. The depth of that gap is what drives ATP synthesis — the energy released down the respiratory chain runs to about −220 kJ per electron pair handed from NADH into the machinery. The cell, in other words, powers itself from the *depth* of a register gap, not from a quantity of fuel. A word of honesty on these figures: the compartment ratios are order-of-magnitude biology, not pinned values, so $\Delta D \approx 6.45$ and the −220 kJ are physical readings whose exact place on the {2,3,5,π} lattice is not yet fixed. We report them as they are — measured magnitudes, their lattice address not read yet — rather than dress an approximate ratio in a tidy fraction it has not earned.

Figure 2 — The cell reads its state from the ratio, not the amount: the depth of the gap is the energy it spends

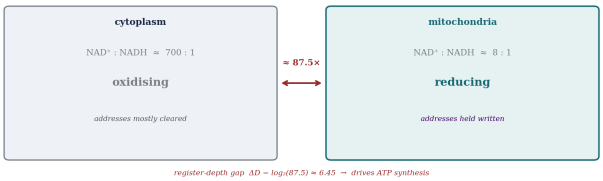

Figure 2 — The ratio, not the amount, is what the cell reads. The ≈87.5-fold gap between the oxidising cytoplasm (≈700:1) and the reducing mitochondria (≈8:1) is a register-depth potential, ΔD ≈ 6.45, and its depth is the energy the cell spends to make ATP.

5 Sirtuins — the T-clock enzymes

Why does running low on NAD make a cell forget how to repair itself? Because of a family of enzymes called sirtuins. Sirtuins consume NAD^+ to deacetylate histones and proteins, regulating gene expression, DNA repair and longevity — and in the Force of Time they are **T-address maintenance enzymes**.

A sirtuin spends NAD^+ — lowering the register's depth by one transaction — to restore a histone's T-address to its optimal register depth, the way a librarian spends effort to re-shelve a book in its correct place. Their activity is directly proportional to NAD^+ level — first-order in $[NAD^+]$ — so sirtuins are quite literally the cell's T-clock: as long as NAD^+ is plentiful they keep the genome's addresses sharp, and they run exactly as fast as the supply of NAD^+ allows. Starve them of the cofactor and the clock slows; the addresses they maintain begin to drift. The link between the molecule that runs out and the repair that fails is this single proportionality.

5.1 The adenine half — why NAD is an address, not just a carrier

There is a clue hidden in the molecule's own name that a century of biochemistry has read past: nicotinamide **adenine** dinucleotide. Half of NAD is adenine — one of the four letters of DNA. In the Force of Time the DNA bases are not arbitrary organic shapes; each is a T-address letter sitting on the {2,3,5,π} lattice, and adenine is the genome's reaching base, the purine that carries its address outward through the nitrogen-9 position where the sugar attaches.

That position lands on a clean lattice node. Read as a wavelength, adenine's reach is **4374** ($N9\text{-adenine} \times H\beta = 2\times3^7$) — the Earth's own infrared window — built on the lattice Balmer line **486** ($2\times3^7 \div 9$), with the ninefold step ($9 = 3^2$) between them. The number to hold is the reach, 4374; the powers of three behind it are only the quiet stamp that says it is on the lattice.

This changes what NAD *is*. It is not merely an electron shuttle that happens to be built around a vitamin. It is an **address-bearing molecule**: one half the nicotinamide redox node where the D-level is written and erased, the other half a genuine genomic T-address letter. So when NAD^+ falls, it is not only a cofactor running short — an adenine T-node is depleting, the very same kind of erasure that, read on the chromosome ends, we call telomere shortening, and read across the whole genome, we call the epigenetic clock. Here at last is the answer to the question the paper opened with — why one small molecule should carry the whole weight of repair. NAD is repair's currency because NAD is written in the genome's own alphabet.

5.2 Three sirtuins, three address-domains — and the four-arm clock

The sirtuins do not all tend the same shelf. Three of them divide the genome's upkeep between them, each spending NAD^+ on a different family of addresses. **SIRT1** keeps the gene-expression and inflammatory addresses sharp; **SIRT3** keeps the mitochondrial electron-transport-chain addresses, where the cell makes its 36 ($(2\times3)^2$) units of ATP; and **SIRT6** keeps the telomere and DNA-repair addresses — the chromosome-end markers themselves.

All three draw from one shared pool of NAD^+. So when that pool halves with age, the three address-domains lose their keeper in the same stroke: gene regulation, energy production, and chromosome-end integrity falter together. This is why NAD decline does not produce one tidy symptom but a coordinated fade across the whole register — the single drained pool is felt at three addresses at once.

And NAD/sirtuin redox is itself only one of four arms by which the living body holds its T-addresses against drift; the other three are the methylation cycle that re-inscribes the addresses, the autophagy balance that clears the misfiled ones, and telomere integrity that guards the chromosome ends. Ageing is the slow loosening of all four. When all four fall below their minimum together — the NAD^+ pool dropping past the floor that sustains the sirtuins among them — the cell can no longer restore itself from within. That simultaneous failure is, in the Force of Time, the molecular definition of death (companion paper, *Death as Time-Equalization Failure*). NAD is one of the four threads; this paper has followed it to its end.

6 Ageing is register erosion

Now the cause of ageing states itself (Figure 3). NAD^+ falls by roughly a half (2×5^2) between ages 20 and 60 — a 40 ($2^3\times5$)-year window, both the halving and the window sitting on clean lattice nodes. As NAD^+ halves, sirtuin activity falls with it, being first-order in the cofactor.

Once sirtuin activity drops below the minimum needed for maintenance, the T-addresses of the genome begin to erode: histones drift from their proper marks, repair lags behind damage, the cell's coordinates blur. That blurring — the steady accumulation of register entropy as the maintenance clock falters — *is* ageing, read at the molecular register. Ageing is not a thousand independent failures piling up by chance; it is one register losing depth, and the maintenance enzymes starving for the cofactor they spend. The thousand symptoms are downstream of one drained register and one slowing clock.

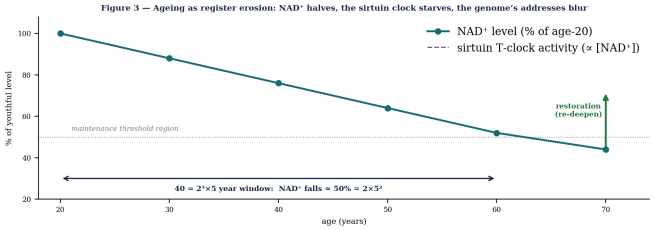

Figure 3 — Ageing as register erosion. NAD⁺ falls about 50% (2×5²) across the 40 (2³×5)-year window from 20 to 60; sirtuin activity, proportional to it, falls in step; below the maintenance threshold the genome's addresses blur. Restoring NAD⁺ re-deepens the register.

7 Restoration — re-starting the clock

The corollary is hopeful, and it is the whole therapeutic point. If ageing is the NAD⁺ register losing depth and the sirtuin clock starving, then in principle restoring NAD⁺ restores register depth and re-starts the clock.

The cell builds NAD⁺ from precursor molecules along the nicotinamide pathway, and supplying those precursors re-deepens the register, lifting sirtuin activity back above the maintenance threshold so that T-address maintenance can resume. This is not a promise of immortality. It is the framework's specific, testable prediction: re-fill one register and you re-enable the enzymes that keep the genome's addresses sharp. The principle is stated here in full; the particulars — which precursor, what amount, on what schedule — are matters for clinical investigation and are held in the Foundation's confidential reference, not prescribed to a reader. What the Force of Time contributes is the *reason* the strategy should work, stated as a register law rather than a hopeful correlation.

8 What this means

NAD has been studied for a century as a metabolic cofactor and, lately, chased as an anti-ageing supplement with an unclear rationale. The Force of Time gives the rationale exactly. NAD is the master redox register, written and erased at position 4 (2²) of its ring.

Its compartment ratios set a register-depth potential, ΔD ≈ 6.45, whose depth powers the cell; sirtuins spend it to keep the genome's addresses sharp; and its decline by about a half (2×5²) across the 40 (2³×5)-year window is the faltering of that maintenance clock — ageing as register erosion. Re-fill the register and the clock restarts. Where a number is a clean lattice node we have said so; where it is an approximate clinical figure we have said that too, and left its address honestly unread rather than force a fit. We give the mechanism in full and at full precision, and we stand by the figures.

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Appendix A — NAD on the Register, at a Glance

Each quantity given first as its plain physical reading, then by its lattice status. Clean {2,3,5,π} nodes are marked; approximate clinical figures are marked honestly as addresses not yet read.

Quantity	Physical reading	Lattice / status	What it is in UFOT
Redox node	position 4 (para)	2^2 — clean node	D = −2 Strand-1 site; address written/erased
Written state	NADH	reduced ring	address set, carrying the e^- pair
Erased state	NAD^+	oxidised ring	address cleared, ready to read
Cytoplasm ratio	≈ 700 : 1	approximate biology	oxidising — addresses mostly cleared
Mitochondrial ratio	≈ 8 : 1	approximate biology	reducing — addresses held written
Register-depth gap	$\Delta D = \log_2(87.5) \approx 6.45$	address not read yet	potential that drives ATP synthesis
Energy per e^- pair	$\Delta G \approx -220$ kJ/mol	address not read yet	register depth spent as ATP
NAD^+ decline	≈ 50% (ages 20–60)	2×5^2 — clean	register losing depth = ageing
Decline window	40 years	$2^3\times5$ — clean	span of the maintenance-clock falter
Adenine half	reach 4374	2×3^7 — clean	genomic T-address letter (NAD's adenine)
Lattice Balmer	486	$2\times3^7 \div 9$ — clean	the line the adenine reach is built on
Three sirtuins	SIRT1 / SIRT3 / SIRT6	genome / ETC / telomere	one NAD^+ pool, three address-domains
ATP node	36	$(2\times3)^2$ — clean	SIRT3's electron-transport-chain address

Appendix B — The Ledger

Table A1 — Propositions P-NAD-1 … P-NAD-8

#	Proposition
P-NAD-1	NAD is the master T-field redox register, holding the cell's D = −2 molecular register at the correct tension; every major metabolic pathway depends on the NAD^+/NADH balance.
P-NAD-2	Redox occurs at position 4 (para) of the nicotinamide ring — the Strand-1 node where the D-level address is written (NADH, reduced) or erased (NAD^+, oxidised). The node sits at 2^2, a clean lattice address.
P-NAD-3	The NAD^+/NADH ratio is the cellular register-depth gauge: ≈700:1 cytoplasm, ≈8:1 mitochondria. The ≈87.5-fold gap gives a register-depth potential $\Delta D = \log_2(87.5) \approx 6.45$ that drives ATP synthesis ($\Delta G \approx -220$ kJ per electron pair). These are approximate clinical figures; their exact {2,3,5,π} address is not read yet and is not forced to a fit.
P-NAD-4	Sirtuins are T-address maintenance enzymes: they consume NAD^+ to deacetylate histones and restore genomic T-addresses; their activity is first-order in $[NAD^+]$ — the cell's T-clock runs as fast as the NAD^+ supply allows.
P-NAD-5	Ageing is register erosion: NAD^+ falls ≈50% = 2×5^2 across the $40 = 2^3\times5$ year window (ages 20–60); sirtuin activity falls with it, the maintenance clock drops below threshold, and the genome's T-addresses erode (register entropy). Both the halving and the window are clean lattice nodes.
P-NAD-6	Restoration re-starts the clock: re-filling the NAD^+ register re-deepens it, lifts sirtuin activity above the maintenance threshold, and resumes T-address maintenance. The principle is stated; the particulars (precursor, amount, schedule) belong to clinical investigation and are held confidentially, not prescribed.
P-NAD-7	NAD is an address-bearing molecule, not merely an electron carrier: its adenine half is a genomic T-address letter on the {2,3,5,π} lattice (N9-adenine reach = $4374 = 2\times3^7$, the Earth IR window, on the lattice Balmer line $486 = 2\times3^7\div9$, ninefold step $9 = 3^2$). NAD^+ decline is therefore adenine T-node depletion — the same erasure read elsewhere as telomere shortening and the epigenetic clock — which is why one small molecule carries the weight of repair.
P-NAD-8	The single NAD^+ pool feeds three sirtuins that divide the genome's upkeep — SIRT1 (gene-expression/inflammatory addresses), SIRT3 (mitochondrial electron-transport-chain addresses, the $36 = (2\times3)^2$ ATP node), SIRT6 (telomere and repair addresses); when the pool halves, all three address-domains lose their keeper at once. NAD/sirtuin redox is one of four T-maintenance arms (with methylation, autophagy, telomere integrity); their simultaneous fall below threshold is the molecular definition of death.

A Note on the Numbers

A note on the numbers. Throughout this paper a value is given first as the plain physical reading and only then, in brackets and in grey, as its place on the {2,3,5,π} lattice. Some of the numbers here are clean lattice addresses and are marked as such: the redox node sits at position $4 = 2^2$ of the nicotinamide ring; NAD^+ falls by about a half = 2×5^2 across a window of $40 = 2^3\times5$ years; these are exact nodes, not approximations. Others are approximate clinical figures, and the Force of Time treats them honestly. The compartment ratios — roughly 700:1 in the cytoplasm and 8:1 in the mitochondria — are order-of-magnitude biology, not pinned values, so the register-depth potential they imply, $\Delta D = \log_2(87.5) \approx 6.45$, does not yet land on a clean {2,3,5,π} node; its address is not read yet, and we do not dress an approximate ratio in a false lattice fraction. The same caution applies to the free energy released per electron pair, $\Delta G \approx -220$ kJ/mol: a measured magnitude whose exact lattice address awaits a cleaner determination of the ratios that set it. A miss is never excused as a residue and never paraded as a fit — it is simply a form not yet read. A T-value is one number that can wear the clothes of a concentration, an energy, or a register depth; here a single redox register, written and erased at one node, governs metabolism, genome maintenance, and the rate at which we age.

References

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[3] S. Imai & L. Guarente, *NAD⁺ and sirtuins in aging and disease*, Trends Cell Biol. 24(8), 464 (2014).
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[5] Daubney, S. *Death as Time-Equalization Failure* (the cellular T-clock; Hayflick 50 = 2×5²). The Daubney Foundation, 2026.
[6] Daubney, S. *The Force of Time Medical Framework* (organs as T-receivers; disease as a register off the lattice). The Daubney Foundation, 2026.

The Daubney Foundation is in ongoing discussions with medical establishments regarding clinical trials of Universal Force of Time solutions to the conditions described in this paper. Any institution or researcher wishing to put themselves forward for participation in these trials is invited to make themselves known through: thedaubneyfoundation@gmail.com