THE UNIVERSAL FORCE OF TIME

Macular Degeneration

The Densest Register in the Body — Three Ways It Erodes, and the Three Corrections That Set It Right

Stephen Daubney · The Daubney Foundation · 2026 · Rev 5

***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

At the very centre of your sight is a patch smaller than the letter o, and into it the body packs the densest array of light-detectors it owns. This is the fovea, the heart of the macula, and in the Universal Force of Time it is the body's densest **T_λ register** — its finest grid of wavelength-reading nodes, packed at about **160,000 cones/mm²** (= $2^8 \times 5^4$) with roughly **125,000** (= $2^3 \times 5^6$) in the fovea itself, a pentagonal {5}-branch lattice whose peak sensitivity sits at **555.165248 nm** (= $225\pi^2/4$), a clean {2,3,5,π} address. Age-related macular degeneration is the decay of that register. This paper does what a Force of Time medical paper is built to do: it acknowledges the illness, reads the problem as the distinct routes by which it arises, and pairs each route, one to one, with the correction that would set it right — and the honest count here is **three**, not four. Route one — the register **erodes and its residue calcifies**: the foveal nodes lose address coherence, the supporting layer can no longer clear its daily waste, and off-lattice **drusen** build up, lit by a self-amplifying blue-light burn; so the correction is to **clear the calcification** before it locks a register cube and to keep the body's own {2,5} carotenoid screen intact. Route two — **wet AMD**: sensing a starved, locked register the field floods VEGF and grows fragile, leaky vessels; so the correction is to **re-feed the register at its own address** so the distress signal stops — restoration with anti-VEGF outperforming anti-VEGF alone. Route three — **dry AMD**: the nodes are **destroyed outright**, leaving register voids the lattice cannot rebuild, the same dΣT=0 irreversibility found in sensorineural deafness; so the correction is **prevention** — to act before the voids open, while the nodes are merely detuned and still callable. The three corrections carry an order law — the calcification ladder, the off-lattice deposit growing until it locks the first {5}-cube at **125 µm** (= 5^3) and the voids open. Ten propositions, P-AMD-1 to P-AMD-10, are given; every value is at full precision, the two register-restoration wavelengths are held in the Foundation's clinical reference, and the structure resolves into the **clinical trial**.

Universal Force of Time = the creation of life = the healing of life = the destruction of life

1 The Centre of Sight

Look at a single word on this page and hold your gaze still. Everything you see sharply — the letters, their edges, the white between them — is landing on a patch of retina smaller than a pinhead. That patch is the fovea, the bullseye of the macula, and it does almost all of your detailed seeing; the rest of the retina sketches the world in rough strokes, and the fovea reads it. When macular degeneration takes that patch the world does not go dark — it goes **blank in the middle**, a smudge sitting exactly where you try to look. It is the leading cause of sight loss in people over fifty, and medicine has good descriptions of it and few cures. The Force of Time begins not with the damage but with what the fovea **is** — because once you see what it is, what fails in it becomes clear, and the small number of ways it fails can each be named and answered. There are three of them, and the honest count is three.

2 The Densest Register in the Body

In the Force of Time the eye is a **T_λ receiver** — an organ that reads the wavelength face of the time-field — and every photoreceptor is one T_λ node, tuned to its own slice of light. The fovea is where those nodes are packed tightest of anywhere in the body. Cone density there reaches about **160,000 per mm²** (= $2^8 \times 5^4$), and the fovea holds on the order of **125,000 cones** (= $2^3 \times 5^6$) — a pentagonal {5}-branch lattice, the body's single densest grid of wavelength registers (Figure 1). Its peak sensitivity falls at **555.165248 nm** (= $225\pi^2/4 = 3^2 \times 5^2 \times \pi^2/2^2$), a clean {2,3,5,π} address — the T-field's own optimal register, not an accident of pigment chemistry, and conventionally quoted by science as a round 555 nm. Nowhere else does the field read the world at such resolution. That is the fovea's glory and its vulnerability: the finest register has the least margin for decay, and it works only at temperature — the body holds **36.864 °C** (= $2^9 \times 3^2/5^3$) precisely because the T_λ register only functions there. Seeing, in the Force of Time, is a **four-stage T_λ chain**: a photon is captured, the visual pigment is read, the signal is sent on, and then the supporting layer beneath resets the spent pigment and clears the day's residue. Most retinal disease strikes a single stage and the eye can often weather it. AMD is the cruel exception — it attacks two stages at once: stage two, the photoreceptor that reads, and stage four, the layer that resets and clears. A dual stage-two-and-four failure is why, of all the ways sight can fail, this one takes the very centre and does not give it back. With the register named, the three ways it decays can be named in turn, and each one answered.

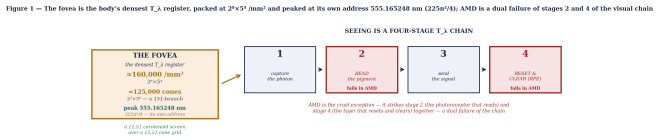

Figure 1 — The fovea is the body's densest T_λ register: ≈160,000 cones/mm² (= $2^8 \times 5^4$), ≈125,000 in the fovea (= $2^3 \times 5^6$), peaked at its own address 555.165248 nm (= $225\pi^2/4$). Seeing is a four-stage chain, and AMD is the dual failure of stage 2 (the photoreceptor that reads) and stage 4 (the layer that resets and clears) — guarded by a {2,5} carotenoid screen set over the {2,5} cone grid.

3 Three Routes, Three Corrections

A Force of Time medical paper has one job. It acknowledges the illness, it identifies the problem — and the problem is rarely single — and it pairs each route, one to one, with the correction that would set it right (Figure 2). With macular degeneration the register decays along three distinct routes. It can **erode and calcify** — the foveal nodes losing address coherence while off-lattice deposits build beneath them, lit by a fire of the eye's own making. The starved register can call for new supply and turn **wet** — vessels grown in emergency that leak and scar. Or the nodes can be **destroyed outright** and the register go **dry** — voids the lattice cannot fill. Cleared, re-fed, prevented. We give three routes, not four, because three is the honest count: erosion, the wet over-response, and the dry node-loss are the genuine ways this register fails, and we do not inflate the architecture to a number it does not have.

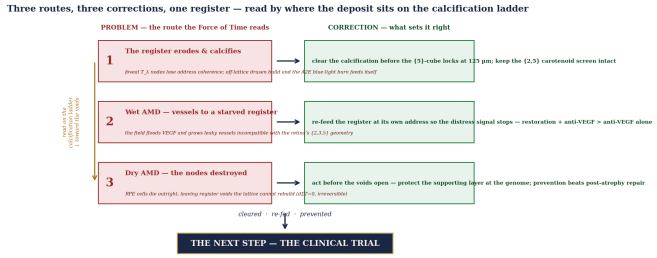

Figure 2 — The architecture of the paper: each of the three routes by which the one register decays is paired with the one correction that sets it right — erosion/calcification (cleared), wet over-response (re-fed), dry node-loss (prevented). The whole structure resolves into the clinical trial.

Route 1 — The Register Erodes and the Residue Calcifies

The first route is the slow common ground of the disease. Age-related macular degeneration, in T-terms, is **erosion of the foveal T_λ register** — the loss of address coherence among the most tightly packed nodes in the body. As coherence falls, the supporting layer beneath the photoreceptors (the retinal pigment epithelium, RPE) can no longer clear its daily residue, and deposits called **drusen** build up. In the Force of Time drusen are not mere litter; they are **register calcification** — off-lattice material settling where the register has weakened, so the T-field can no longer flow through. And here the lattice speaks by its absence: the small drusen that first appear, around **63 µm** across, sit **off** the lattice — that diameter has no clean {2,3,5,π} address at all, which is exactly what off-lattice calcification should look like. Beneath the deposits is the engine that lights them. Each night the RPE swallows the worn-out tips the cones shed; what it cannot fully digest piles up as lipofuscin, and the most dangerous thing in it is **A2E**, a molecule built from two spent visual pigments fused together. A2E is itself a T_λ-absorber: strike it with high-energy blue light and it releases reactive oxygen that damages the very cell holding it; the damaged cell clears still less, makes more A2E, and the next flash of blue does more harm — a **self-amplifying register burn**, the fire whose ash is the drusen. This same clearance failure appears, organ for organ, in the failing kidney, where off-lattice uremic toxins accumulate because the clearance register can no longer carry them out: the macula and the kidney fail in the same grammar.

Correction 1 — clear the calcification before the cube locks; keep the {2,5} screen intact

If the fault is a clearance register silting up with off-lattice residue, the correction is to **clear the calcification before it locks a register cube** — and to keep the body's own defence in place. For the body anticipated exactly this burn: across the macula it lays down two carotenoid pigments, lutein and zeaxanthin, and each is a $\mathbf{C_{40}}$ $(= 2^3 \times 5)$ chain, sharing the very same {2,5} register family as the cone grid it shades (Figure 4). They drink the high-energy violet/blue band — the precise band where A2E burns — before it ever reaches the photoreceptors. The macular screen is the body's own register identity, a {2,5} filter set deliberately in front of a {2,5} grid, not a supplement to be prescribed. The principle of correction is therefore double: clear the off-lattice deposit while the register is still callable, and hold the {2,5} screen intact so the blue band never reaches the burn. The decisive window is before the deposit grows to the first {5}-cube; the supportive specifics are held in the Foundation's reference, not prescribed to a reader.

Route 2 — Wet AMD: Vessels Called to a Starved Register

The second route is the aggressive turn. In its wet form AMD grows new, fragile blood vessels up into the retina, and they leak and scar, destroying central vision fast. Medicine names the driver — a signalling molecule, **VEGF** — and treats it by injection into the eye. The Force of Time reads VEGF as a **register-distress signal**. Faced with a foveal register that has calcified and starved, the field calls for new supply and tries to vascularise the locked register. The vessels are not the primary fault; they are the field's desperate answer to a register it can no longer feed. VEGF itself is a nitrogen-junction signal — the same T-overactivation node that drives vascular distress in the cardiovascular and respiratory registers — here switched on by a starved, calcified fovea. And because they are grown in emergency, the vessels are structurally incompatible with the **{2,3,5} geometry** of the retina, which is why they leak. This is an **over-response**: not too little signal but too much, a register screaming for supply it cannot use.

Correction 2 — re-feed the register at its own address so the distress call stops

If the fault is a starved register flooding the field with a distress call, the correction is to **re-feed and re-power the register at its own address**, so the signal that drives the vessels is removed at source. Anti-VEGF injection silences the distress call without restoring the register beneath it — it quiets the symptom and leaves the starvation. So the Force of Time makes a clean prediction: **register-restoration combined with anti-VEGF will outperform anti-VEGF alone**, because restoration addresses the underlying register failure the injection leaves untouched. The register has its own address — the fovea's peak at 555.165248 nm $(= 225\pi^2/4)$ — and a detuned register can be called back by playing it its own frequency; the target is the register, not only the vessel. The precise restoration wavelength is held confidentially in the Foundation's clinical reference pending trials, not printed here.

Route 3 — Dry AMD: The Nodes Destroyed Outright

The third route is the quiet, irreversible one. In the slow, dry form — geographic atrophy — the RPE cells die outright, and where they die the T_λ receiver array is not merely disrupted but **destroyed**, leaving **register voids** that cannot be reconstructed from the surrounding lattice. This is the same irreversibility the Force of Time describes for sensorineural hearing loss: when register nodes are destroyed rather than detuned, the **dΣT=0** conservation law governs the redistribution of T to the surviving nodes, not the restoration of the lost ones. A destroyed T_λ address cannot be re-issued. This is the hard fact that sets the whole strategy: once a register has calcified past the **125 µm = 5³** threshold and the cells beneath have died, it cannot be returned to lattice coherence. Unlike Routes 1 and 2, where the register is detuned and still callable, here the register is gone — and no correction reaches a node that no longer exists.

Correction 3 — act before the voids open; prevention beats post-atrophy repair

If the fault is outright node-destruction, the only correction is to **act before the voids open** — while the nodes are merely detuned and still callable. The decisive intervention is early: protect and re-cohere the supporting layer at the **genome** itself, driving the RPE's repair so the deposits are headed off before they form, and re-enable the **retinoid/RXR writer** — the same node-writer the Force of Time identifies behind Parkinson's and multiple sclerosis — so the RPE keeps writing its own addresses. Because dry AMD is irreversible past the {5}-cube threshold, prevention is not one option among several but the whole of the correction: keep the register callable, and never let it reach the void. The principle is early protection at the genome and the retinoid writer; the specific agent, dose and schedule belong to clinical investigation and are held in the Foundation's reference, not prescribed here.

4 The Calcification Ladder and the Order Law

The three corrections are not interchangeable, and the way they bind is the order law itself (Figure 3). The binding is the **calcification ladder**: **callable detuned nodes**, still on the lattice and still re-callable → small drusen near **63 µm**, off the lattice with no clean address → sight-threatening drusen at **125 µm** (= 5^3), the first {5}-cube, where the calcification locks a register cube and clinical risk climbs sharply → **register voids** (dΣT=0), where the cells die and the addresses cannot be re-issued. The disease announces its stages on the lattice not by landing on it but by the moment the off-lattice deposit **collides** with it. And the ladder sorts the routes. While the register sits below the cube it is callable: Route 1 governs — clear the deposit and hold the screen — and Route 2's starved register can still be re-fed at its address. Once the deposit locks the {5}-cube and the cells die toward atrophy, Route 3 governs — and Route 3's only correction is prevention, because past the cube there is no node left to call. The same rungs that say where the register can be restored say where it can only be protected. This is why the framework is emphatic that the decisive intervention is early: the window to act is before the voids open.

5 The Engine and the Screen — the Burn Beneath the Drusen

One mechanism sits beneath the first route and is worth lifting out, because it is the fire whose ash we see and the body's own answer to it (Figure 4). The drusen are the soot; the engine is the lipofuscin bis-retinoid **A2E**, the chromophore the clinic had described without naming its grammar. A2E is itself a T_λ-absorber, and that is the whole trouble: struck by high-energy blue light it does not merely warm — it releases reactive oxygen that damages the very cell holding it. A damaged cell clears its residue still less well, so it makes more A2E, so the next flash of blue does more harm: a register feeding on its own failure. The deposits we see are the ash of a fire lit by the eye's own light. And the body anticipated it. Across the macula it lays down lutein and zeaxanthin, each a **C_{40}** (= $2^3 \times 5$) chain in the same {2,5} register family as the cone grid, and they drink the violet/blue end of the spectrum — the precise band where A2E burns — before it reaches the photoreceptors. The macular screen is a **{2,5} filter set in front of a {2,5} grid**: a register identity, not a supplement to be prescribed. When the screen thins with age the blue band reaches the A2E, the burn accelerates, and the off-lattice residue of Route 1 begins to pile. The fire has an address; so does the shield that was meant to stop it.

6 The Resolution — the Clinic Has Already Corroborated It

With the three routes named and each paired to its correction, the paper resolves where it must. We have acknowledged the illness — AMD told not as a centre going dark with age but as the finest instrument in the body falling out of tune, the densest register we own (≈125,000 foveal cones = $2^3 \times 5^6$, peaked at 555.165248 nm = $225\pi^2/4$), eroding node by node; we have read the problem as three distinct routes — the register erodes and calcifies, it turns wet, or it goes dry; we have given, for each, the Force-of-Time correction that would set it right — clear the calcification before the {5}-cube locks and hold the {2,5} screen, re-feed the starved register at its own address so the vessels stand down, and act before the voids open; and we have bound them with the calcification ladder, the off-lattice deposit growing until it locks the first {5}-cube at **125 µm** (= 5^3) and the voids open. Cleared, re-fed, prevented. And the striking thing is that medicine has already stumbled, by trial and error, onto the same principle the Force of Time states from first principles: a retinal **photobiomodulation** system has been authorised for dry AMD, and independent controlled trials have measurably improved photoreceptor sensitivity and lifted mitochondrial activity in older patients — which, on the Force of Time view, is exactly a register switched back on at its own address. The only honest conclusion left is the one the whole structure points to: **test it**. The two register-restoration wavelengths are calculated and stated here as principle precisely because the next step is not to prescribe them to a reader but to put them to a clinical trial. We give the mechanism in full and at full precision, and we stand by the figures.

Table 1 — The Three Routes and Their Corrections

Each route by which the one register decays, paired one-to-one with the correction that sets it right — erosion/calcification (cleared), wet over-response (re-fed), dry node-loss (prevented). Order law: the calcification ladder, the off-lattice deposit growing until it locks the first {5}-cube at 125 μm = 5^3 and the voids open. The three corrections resolve into the clinical trial.

#	Problem route	State / {2,3,5,π} reading	Correction (principle)
1	The register erodes and the residue calcifies	foveal T_λ nodes lose address coherence; off-lattice drusen build (small ≈63 μm, no clean address) and the A2E blue-light burn feeds itself	Clear the off-lattice calcification before it locks a register cube at 125 μm = 5^3; keep the body's own {2,5} carotenoid screen (C_{40} = $2^3\times5$) intact
2	Wet AMD — vessels called to a starved register	sensing a locked, starved fovea the field floods VEGF (a nitrogen-junction overactivation node) and grows leaky vessels incompatible with the retina's {2,3,5} geometry	Re-feed and re-power the register at its own address (555.165248 nm = $225\pi^2/4$) so the distress call stops — restoration + anti-VEGF > anti-VEGF alone
3	Dry AMD — the nodes destroyed outright	RPE cells die in geographic atrophy, leaving register voids the lattice cannot rebuild; a destroyed address cannot be re-issued (dΣT=0, irreversible)	Act before the voids open, while nodes are detuned and callable — protect the supporting layer at the genome, re-enable the retinoid/RXR writer; prevention beats post-atrophy repair

Appendix A — The Macula on the Calcification Ladder

Every number this paper turns on, given first as its physical reading and then as its place on the {2,3,5,π} lattice. The fovea's own values are clean lattice addresses; the drusen are read by their ABSENCE from the lattice until they collide with the first {5}-cube at 125 μm = 5^3. Values are register identities, not prescribed therapy.

Quantity	Physical reading	{2,3,5,π} reading	Register / meaning
Foveal cone density	≈ 160,000 /mm²	$2^8\times5^4$	the densest T_λ grid in the body
Foveal cone count	≈ 125,000	$2^3\times5^6$	a pentagonal {5}-branch register
Peak sensitivity	555.165248 nm	$225\pi^2/4 = 3^2\times5^2\times\pi^2/2^2$	the fovea's own T_λ address
Working temperature	36.864 °C	$2^9\times3^2/5^3$	the register functions only at temperature
Macular screen	lutein / zeaxanthin	$C_{40} = 2^3\times5$	the {2,5} screen over the {2,5} cone grid
Engine	A2E (bis-retinoid)	T_λ-absorber	the self-amplifying blue-light burn
Small drusen onset	≈ 63 μm	off-lattice (no clean address)	the signature of off-lattice calcification
Sight-threatening drusen	125 μm	5^3	the first {5}-cube — a register cube locks
Visual chain	four stages	dual stage-2 + stage-4	photoreceptor + RPE fail together
Dry AMD (atrophy)	register voids	dΣT=0	destroyed addresses cannot be re-issued

Appendix B — The Ledger

Table B1 — Propositions P-AMD-1 … P-AMD-10

#	Proposition
P-AMD-1	The fovea is the body's densest T_λ register: cone density ≈160,000/mm² = $2^8\times5^4$, ≈125,000 foveal cones = $2^3\times5^6$ (a pentagonal {5}-branch), peak sensitivity 555.165248 nm = $225\pi^2/4 = 3^2\times5^2\times\pi^2/2^2$. Each cone is one T_λ wavelength node; the register functions only at T_body = 36.864 °C = $2^9\times3^2/5^3$.
P-AMD-2	Seeing is a four-stage T_λ chain (capture → read → send → reset/clear). AMD is the cruel exception: it strikes two stages at once — stage 2 (the photoreceptor that reads) and stage 4 (the RPE that resets and clears) — which is why it takes the very centre and does not give it back.
P-AMD-3	ROUTE 1 — the register erodes and the residue calcifies (off-lattice drift): foveal T_λ nodes lose address coherence, the RPE can no longer clear its daily residue, and off-lattice drusen build up. Small drusen (≈63 μm) sit OFF the lattice, with no clean {2,3,5,π} address — the signature of off-lattice calcification. CORRECTION 1: clear the off-lattice calcification before it locks a register cube at 125 μm = 5^3, and keep the body's own {2,5} carotenoid screen intact.
P-AMD-4	The engine beneath Route 1 is the lipofuscin bis-retinoid A2E — itself a T_λ-absorber that, struck by high-energy blue light, releases reactive oxygen, damages its host RPE cell, impairs clearance and breeds more A2E: a self-amplifying register burn. The body's own defence is a carotenoid screen, lutein and zeaxanthin, each $C_{40} = 2^3\times5$, sharing the cone grid's {2,5} register family and filtering the violet/blue band before it reaches the burn — a register identity, not a prescribed supplement.
P-AMD-5	ROUTE 2 — wet AMD, vessels called to a starved register (over-response): sensing a calcified, starved fovea the field floods VEGF (a nitrogen-junction overactivation node, shared with cardiovascular and respiratory distress) and grows fragile vessels structurally incompatible with the retina's {2,3,5} geometry, which leak and scar. The vessels are the field's answer, not the primary fault. CORRECTION 2: re-feed and re-power the register at its own address (555.165248 nm = $225\pi^2/4$) so the distress signal stops. PREDICTION: register-restoration + anti-VEGF > anti-VEGF alone.

#	Proposition
P-AMD-6	ROUTE 3 — dry AMD, geographic atrophy (node deletion): RPE death produces register voids the surrounding lattice cannot rebuild; under dΣT=0, T redistributes to surviving nodes rather than restoring lost ones — a destroyed T_λ address cannot be re-issued, the same irreversibility as sensorineural deafness. CORRECTION 3: act before the voids open, while nodes are detuned and callable — protect the supporting layer at the genome, re-enable the retinoid/RXR writer; prevention beats any post-atrophy correction.
P-AMD-7	The RPE register is maintained by the retinoid/RXR node-writer shared with Parkinson's and multiple sclerosis; the disease-modifying route, in principle, re-powers the register with light while a retinoid re-enables its writer. The specific agent, dose and schedule are held in the Foundation's clinical reference.
P-AMD-8	ORDER LAW: the calcification ladder — callable detuned nodes (on lattice) → small drusen ≈63 μm (OFF lattice, no clean address) → sight-threatening drusen 125 μm = 5^3 (the first {5}-cube; a register cube locks) → register voids (dΣT=0, irreversible). The disease announces its stages not by landing on the lattice but by where the off-lattice deposit collides with it. Routes 1 & 2 act while the register is callable, below the cube; Route 3 governs past it, where prevention is the only correction.
P-AMD-9	The clearance failure of Route 1 is the same T-signature as uremic-toxin accumulation in chronic kidney disease (P-RENAL-7): a clearance node that can no longer carry off-lattice residue out of the register. Different tissue, identical grammar — the macula and the kidney fail in the same way.
P-AMD-10	The clinic has already corroborated the repair principle: red-light photobiomodulation, authorised for dry AMD, measurably preserves photoreceptor function and lifts mitochondrial activity — on the Force of Time view, a register switched back on at its own address. The two register-restoration wavelengths are calculated and held confidentially pending trials; the three corrections resolve into the clinical trial. No prime-7 sits on the lattice; the lattice is {2,3,5,π} only.

A Note on the Numbers

A note on the numbers. Throughout this paper a quantity is given first as the plain physical value a clinician would measure — a count of cells, a wavelength, a length in microns — and only then, in brackets, as its place on the {2,3,5,π} lattice. The lattice form is not a unit and carries no powers of ten of its own: a T-value is one number that wears different clothes in different registers, appearing as a density of detectors here, a colour of light there, a diameter somewhere else. It is why the fovea's peak sits at the clean address 555.165248 nm = $225\pi^2/4$ and its grid is built from {2,5}: the densest register in the body is also the most exactly placed. The drusen tell the opposite story. An off-lattice deposit has no clean {2,3,5,π} address at all, and that very absence is the signature of the pathology — the small drusen near 63 μm sit nowhere on the lattice, and the disease turns dangerous not when the deposit lands on a node but when it grows to collide with the first {5}-cube at 125 μm = 5^3 and locks a register cube. No prime-7 sits on the lattice; the lattice is {2,3,5,π} only, and a register the body can no longer file is a register drifting off it.

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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