THE UNIVERSAL FORCE OF TIME

Multiple Sclerosis

The Leap That Fails — Four Routes by Which Myelin's T-Register Is Lost, and the Four Corrections That Restore It

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

A nerve does not pass its signal along like a burning fuse — it throws it. The impulse leaps from one bare gap in the insulation to the next, skipping the insulated stretch between; physiology calls this saltatory conduction and the gaps the nodes of Ranvier. The Force of Time recognises it at once, because T acts only at nodes and the nerve is built to make the signal jump from node to node. The insulation that makes the leap possible is myelin, and its job is to hold the T-register coherent across the gap the signal must cross; multiple sclerosis is the loss of that coherence. This paper does what a Force of Time medical paper is for: it acknowledges the illness, then reads the problem as up to **four distinct routes** by which the leap fails, and pairs **each route with the one correction that would realign it**. Route one — the **signal fails** as the register loses coherence below the **½ µm = 1/2** threshold (healthy myelin is **1.5 µm = 3/2** thick at a g-ratio of **0.6 = 3/5**, carrying conduction near **70 m/s**; a severe lesion thins to **0.2 µm = 1/5** and crawls toward **5 m/s**) — corrected by re-cohering the register at its own **40 Hz = 2³×5** gamma beat. Route two — the **sheath is stripped** by an autoimmune T-address misdirection (the family of Type-1 diabetes, rheumatoid arthritis and HIV) — corrected by restoring the address the immune system should read. Route three — **no regrowth**, because the retinoid writer (RALDH2/RXR) that drives precursor cells to remyelinate is stalled, the same writer stalled in Parkinson's — corrected by re-enabling that writer. Route four — the **window closes** as a chronically bare axon degenerates — corrected only by acting while the axon still lives. The corrections carry an order law: route two must be corrected before route three, because the active attack itself holds the writer shut. Laid out this way, the paper resolves where it must — into the **clinical trial** that would test the four corrections. Every diagnostic number is at full precision; corrective detail is held in the Foundation's clinical reference.

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1 The Signal That Leaps

We picture a nerve impulse running down a fibre like a flame along a fuse — smooth, continuous, end to end. It is nothing of the kind. The signal jumps. Along every fast nerve the insulation is laid down in segments, and between the segments are tiny bare gaps; the impulse leaps from one gap to the next, skipping the insulated stretch in a single bound. Physiologists named this saltatory conduction — from *saltare*, to leap — and named the gaps the nodes of Ranvier. It is how a signal crosses a tall human body in a heartbeat. The Force of Time recognises this picture immediately, because it is the picture the theory draws of everything: T acts at nodes; change happens in jumps between coordinates, not as a smooth slide. A nerve is a node-to-node relay made flesh — and that recognition is the key to a disease that has always been described by what it destroys and never by what that thing was for.

2 Myelin Holds the Register in the Gap

The insulation is myelin, wound around the fibre by support cells in tight layers. Medicine treats it as a passive wrapper that stops the current leaking. The Force of Time gives it an active role: myelin holds the T-register coherent across the gap the signal must jump. The leap is not a spark crossing empty space; it is the field staying in phase from one node to the next, so the impulse can land. Strip the myelin and the register loses coherence between nodes — and the jump has nothing to land on. The numbers carry the lattice. Healthy myelin runs about **1.5 µm = 3/2** thick, wound to an optimal axon-to-fibre ratio (the g-ratio) of **0.6 = 3/5** — the value at which conduction is fastest. So insulated, the fibre carries its signal near **70 m/s**. These are not free parameters; they are the settings at which the inter-node T-register holds.

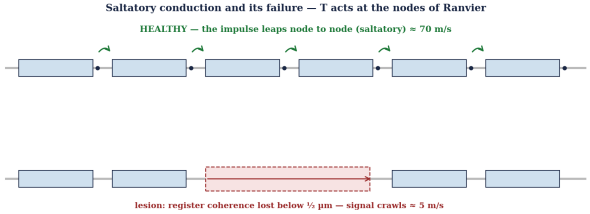

Figure 1 — Healthy myelin lets the impulse leap node to node (saltatory, ≈ 70 m/s); where a lesion strips the sheath below ½ µm the inter-node T-register loses coherence and the signal crawls (≈ 5 m/s). T acts at the nodes of Ranvier.

3 Where Medicine Stands

Multiple sclerosis affects roughly **2.8 million** people worldwide. Medicine has mapped it with great care: it sees the immune attack on myelin, it sees the plaques on MRI, and it has built a generation of disease-modifying therapies that suppress the attack. The most potent of them are precise immune tools — **natalizumab** (anti-VLA-4, which blocks immune cells from crossing into the brain and cuts relapses by about **68%**), **ocrelizumab** and **ofatumumab** (anti-CD20, which deplete the B-cells that drive the attack), and **alemtuzumab** and **cladribine** (which reset the immune repertoire). They work, and they matter: they slow the formation of new lesions. But they all stop at the same wall. Not one of them repairs a lesion that has already formed — none restores the lost myelin or the conduction it carried. Medicine can quiet the attack; it cannot yet give the nerve back its leap. That is exactly the gap the Force of Time reads, and it reads it not as one problem but as four.

4 Four Routes, Four Corrections

A Force of Time medical paper has one job. It acknowledges the illness, it identifies the problem — and the problem is rarely single; here it has up to four distinct routes by which the leap is lost — and it pairs each route, one to one, with the correction that would realign it. The four routes are not rival theories; they are four real ways the same disease takes a fibre down, and a given patient may be losing the leap by one of them, by several, or by all four at once. What follows names each route, then its correction, in order. The reader should hold the whole shape in view (Figure 4): four problems on the left, four corrections on the right, bound by one order law, resolving into a single next step.

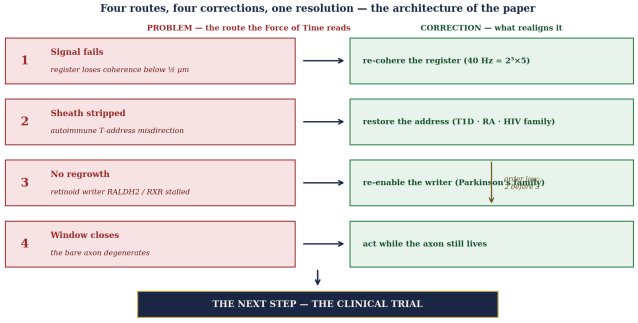

Figure 4 — The architecture of the paper: each of the four routes by which myelin's T-register is lost is paired with the one correction that realigns it; correction 2 precedes correction 3 by the order law; the whole structure resolves into the clinical trial.

Route 1 — The Signal Fails: the register loses coherence below ½ µm

The first route is the loss of the leap itself. While the myelin stays above about **½ µm = 1/2**, coherence holds and the signal still jumps. Below ½ µm the register fails between nodes, and the impulse can no longer leap — it falls back to crawling continuously down the bare fibre, dropping from **70 m/s** toward **5 m/s** in a severe lesion thinned to about **0.2 µm = 1/5** (Figure 2). The lesion boundary is a register phase transition: on one side the leap works, on the other it does not, with very little in between. That sharp edge is why MS deficits appear and vanish so abruptly, and why the disease relapses and remits — a fibre balanced near the ½ µm threshold is tipped below by inflammation (the symptom appears) and tipped back above by partial repair (it remits). Relapsing–remitting MS is a T-register flickering across its phase transition; the secondary-progressive phase is the register settling permanently below the edge.

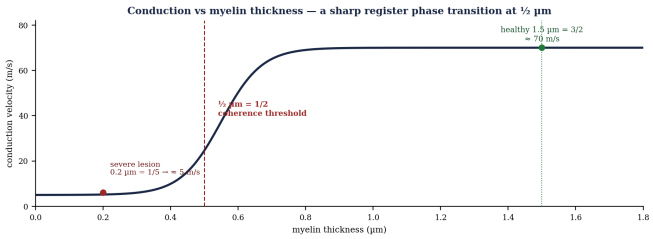

Figure 2 — Conduction holds near 70 m/s at healthy thickness 1.5 µm (= 3/2) and collapses below the coherence threshold ½ µm (= 1/2); a severe lesion thins to 0.2 µm (= 1/5). The transition is sharp — a switch, not a slope.

Correction 1 — re-cohere the register at its own 40 Hz beat

If the problem is a register that has lost coherence, the correction is to restore that coherence. The brain already has a frequency at which it holds wide-area coherence: the **40 Hz = 2³×5** gamma beat (40 = $2^3 \times 5$), a clean lattice frequency and the body's own driver of large-scale neural synchrony. In MS this gamma coherence is measurably reduced — and reduced not in one spot but across all regions of the brain, exactly as a lost timing register would predict. The correction works *with* that identity: re-cohere the T-register at the frequency the brain itself uses to hold it. The principle is what the theory fixes; the specific means of driving it belong to clinical investigation and are held in the Foundation's reference.

Route 2 — The Sheath Is Stripped: an autoimmune T-address misdirection

The second route is the attack that strips the sheath in the first place. In T-terms it is an **autoimmune T-address misdirection** — the same family of failure as Type-1 diabetes, rheumatoid arthritis and HIV, in which the immune system, the body's proof-reader of addresses, misreads an address and attacks the wrong target. Here it misreads the address of myelin and strips it; where it does, a plaque forms — a place where the T-register has collapsed. The disease is not, at root, a destruction of tissue; it is a misfiling of identity that causes the destruction. That places this route in a definite class, with a correction shared across the whole misdirection family rather than one invented for MS alone.

Correction 2 — restore the address the immune system should read

The correction for a misdirection is not to suppress the messenger but to **restore the address** the immune system should be reading, so the proof-reader stops attacking myelin as foreign. This is the same correction principle the framework carries for Type-1 diabetes, rheumatoid arthritis and HIV — one family, one route home. Today's disease-modifying therapies act on this route from the outside, blunting the attack; the Force of Time aims at the misfiling itself. The principle is the fixed point; the corrective means are held confidentially pending trials.

Route 3 — No Regrowth: the retinoid writer is stalled

The third route is why the damage does not simply heal. The brain is full of **oligodendrocyte precursor cells (OPCs)** — the most abundant precursor in the central nervous system — and they carry the complete oligodendrocyte programme in their genome. In the MS plaque they do not differentiate into the mature, myelin-making cells that would repair the sheath. This is not an absence of potential; it is a **blocked transition** (Figure 3). In UFOT terms the oligodendrocyte T-address is intact ($d\Sigma T=0$) — the inscription is preserved — but the **writer** that reads it out has stalled. The writer is the retinoid cascade: retinoic-acid synthesis (RALDH2) signalling through the RXR receptor — and the framework identifies this as the very same retinoid-X-receptor writer stalled in Parkinson's. Two neurodegenerations, one writer. It is why immune-suppressing therapies slow new lesions but cannot repair old ones: they address the attack and leave the blocked transition untouched.

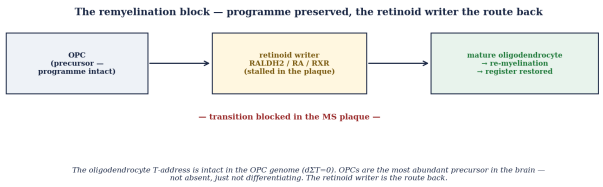

Figure 3 — Remyelination is blocked, not impossible: OPCs hold the intact oligodendrocyte programme (dΣT=0); the retinoid writer (RALDH2/RA/RXR) — the same one stalled in Parkinson's — is the route that re-enables differentiation.

Correction 3 — re-enable the writer so OPCs remyelinate above ½ µm

The correction is to **re-enable the writer** — to lift the stall on the RALDH2/RXR retinoid cascade so the OPCs already present can differentiate and rebuild myelin back above the ½ µm coherence threshold. The programme they need is intact; only the writer is shut. That RXR signalling drives remyelination is not a UFOT conjecture alone — it is the experimental finding of Huang and colleagues, who showed RXR-γ signalling accelerates remyelination in the central nervous system. This is the frontier no approved agent has yet reached: every current therapy works on Route 2, the attack; Route 3, the regrowth, is still open. The principle is the fixed point; the corrective means are held in the Foundation's reference.

Route 4 — The Window Closes: the bare axon degenerates

The fourth route is the one the clock opens. Relapsing–remitting MS is reversible because the axon underneath the stripped sheath is still there, waiting to be re-insulated. But a nerve fibre left bare does not wait forever. An axon that runs chronically without myelin loses the support the sheath provides and eventually degenerates — and once the axon itself is gone, no amount of remyelination can restore the signal, because there is no longer a fibre to insulate. This is the same **irreversibility law** the framework meets wherever a T-node fails to regenerate: a register can be re-cohered only while the structure that carries it survives. It draws a hard line through the disease. While the demyelinated axon is intact, the deficit is a conduction block — reversible, a switch waiting to be flipped back. Once the axon degenerates, the deficit converts into permanent disability — a node lost, not a switch thrown. The long secondary-progressive decline is, in large part, this conversion accumulating.

Correction 4 — act while the axon still lives

This route's correction is not a substance but a **timing**: act while the axon still lives. The window for restoration is real but finite, and it argues for re-cohering the register and re-enabling the writer early, while the fibres that carry the signal are still alive. Timing is not a footnote to the other three corrections — it is itself the fourth correction, the one that decides whether the others can land. It also yields a clean, testable prediction: a corrective that genuinely re-drives the writer should show rising myelin signal on MRI magnetisation-transfer imaging in treated lesions whose axons remain intact, and none where the axon has already been lost.

5 The Order Law — Correction 2 Must Precede Correction 3

The four corrections are not freely interchangeable, and one ordering is forced. **Correction 2 must come before correction 3.** The reason is mechanistic, not merely humane: the inflamed environment of the active plaque is itself the block on the writer. Active neuroinflammation actively suppresses the OPC→oligodendrocyte transition — the very signalling that drives the attack holds RALDH2/RXR shut. So a remyelinating correction applied to a still-inflamed lesion has nothing to act on; the writer it would re-enable is being held closed by the attack. Quieting the misdirection (correction 2) is therefore the **permissive condition** for re-enabling the writer (correction 3), not just the kinder first move. In T-terms: you cannot re-inscribe an address while the proof-reader is still scribbling over it. Correction 1 (re-cohering the register) supports throughout, and correction 4 (timing) governs all of them — but the 2-before-3 sequence is the law the theory insists on.

6 The Resolution — the Clinical Trial Is the Next Step

With the four routes named and each paired to its correction, the paper resolves where it must. We have acknowledged the illness; we have read the problem as four distinct routes by which the leap is lost; we have given, for each, the Force-of-Time correction that would realign it; and we have bound them with the order law. The only honest conclusion left is the one the whole structure points to: **test them**. The four corrections — re-cohere the register, restore the address, re-enable the writer, act while the axon lives — are stated here as principles precisely because the next step is not to prescribe them to a reader but to put them to a clinical trial. That is what a finished Force of Time medical paper does: it lays the routes and the corrections side by side until the trial is the obvious next move. Multiple sclerosis has always been told as a story of insulation worn away. The Force of Time tells it as a leap that fails by four routes — and as four corrections, in their proper order, that could give the nerve back its leap while the fibres that carry it are still alive. We give the mechanism in full and at full precision, and we stand by the figures.

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Appendix A — The Four Routes and Their Corrections

Each route the Force of Time reads in multiple sclerosis, paired one-to-one with the correction that realigns it. Order law: correction 2 precedes correction 3 (active inflammation holds the writer shut). The four corrections resolve into the clinical trial.

#	Problem route	{2,3,5} reading	Correction (principle)	Shared family
1	Signal fails — register loses coherence below ½ µm	½ µm = 1/2 · 40 Hz = $2^3 \times 5$	Re-cohere the register at its 40 Hz gamma beat	—
2	Sheath stripped — autoimmune T-address misdirection	—	Restore the address the immune system should read	Type-1 diabetes · RA · HIV
3	No regrowth — retinoid writer RALDH2/RXR stalled	—	Re-enable the writer so OPCs remyelinate above ½ µm	Parkinson’s
4	Window closes — the bare axon degenerates	—	Act while the axon still lives (timing IS the correction)	—

Appendix B — The Myelin T-Register and Conduction

The healthy and lesioned settings of the myelin sheath as lattice values. Conduction collapses sharply across the ½ µm = 1/2 coherence threshold — a register phase transition, not a gradual slope. Every value is a clean {2,3,5} ratio; no prime-7 anywhere.

Myelin state	Thickness	{2,3,5} reading	Conduction	Register
Healthy	1.5 µm	3/2	≈ 70 m/s	leap holds — saltatory
Optimal g-ratio	axon/fibre 0.6	3/5	fastest	tuned for the leap
Coherence threshold	0.5 µm	1/2	leap fails at/below	phase transition edge
Severe lesion	0.2 µm	1/5	≈ 5 m/s	crawl — continuous
Gamma carrier	—	40 Hz = $2^3 \times 5$	—	brain-wide timing register

Appendix C — The Ledger

Table C1 — Propositions P-MS-1 … P-MS-9

#	Proposition
P-MS-1	Myelin is the T-register of saltatory conduction: it holds inter-node coherence so the impulse can leap (T acts at the nodes of Ranvier). Healthy settings: thickness 1.5 µm = 3/2, g-ratio 0.6 = 3/5, conduction ≈ 70 m/s.
P-MS-2	ROUTE 1 — Signal fails: above ½ µm = 1/2 the leap holds; below it the register fails and conduction falls 70 → 5 m/s (severe lesion 0.2 µm = 1/5). The lesion edge is a sharp phase transition — hence abrupt symptoms; relapsing–remitting MS is the register flickering across it. CORRECTION 1: re-cohere the register at its 40 Hz = $2^3 \times 5$ gamma beat (measurably reduced brain-wide in MS).
P-MS-3	ROUTE 2 — Sheath stripped: MS begins as an autoimmune T-address misdirection (family of Type-1 diabetes, RA, HIV); the immune system misreads myelin’s address and the plaque is a collapsed T-register. CORRECTION 2: restore the address the immune system should read (shared across the misdirection family).
P-MS-4	ROUTE 3 — No regrowth: OPCs hold the intact oligodendrocyte programme ($d\Sigma T=0$) but the OPC→oligodendrocyte transition is blocked; the retinoid writer (RALDH2 → retinoic acid → RXR — the same writer stalled in Parkinson’s) has stalled. CORRECTION 3: re-enable the writer so OPCs remyelinate above ½ µm (RXR-γ drives remyelination — Huang et al. 2011).
P-MS-5	ROUTE 4 — Window closes: a chronically demyelinated axon eventually degenerates, and a lost fibre cannot be re-insulated (the same irreversibility law as a non-regenerating T-node), converting a reversible conduction block into permanent disability — the substance of secondary progression. CORRECTION 4: act while the axon still lives — timing is itself the correction.
P-MS-6	ORDER LAW: correction 2 must precede correction 3. Active neuroinflammation suppresses the OPC→oligodendrocyte transition — the attack holds the writer shut — so quieting the misdirection is the permissive condition for remyelination, not merely the humane first step.
P-MS-7	RESOLUTION: with four routes named and four corrections paired, the paper resolves into the clinical trial as the next step. The corrective modalities are held confidentially pending those trials; only the principles appear here.
P-MS-8	Current disease-modifying therapies (natalizumab anti-VLA-4 ≈ 68% relapse reduction; ocrelizumab/ofatumumab anti-CD20; alemtuzumab; cladribine) act on Route 2 (the attack) only; they slow new lesions but repair none — Routes 1, 3 and 4 are the open frontier. ~2.8 million people are affected worldwide.
P-MS-9	40 Hz = $2^3 \times 5$ is the brain’s gamma timing register — the lattice frequency at which wide-area neural coherence holds. It is a register identity, not a prescribed therapy.

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 thickness, a conduction speed, a frequency — 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 thickness in a nerve here, a span of time in the heavens there, a mass in a nucleus somewhere else. We do not "solve to a power" in a single dimension. The bracket is simply the address; the physical number is the thing you can hold.

References

[1] Daubney, S. *The Universal Force of Time — Master Compendium*, v5. The Daubney Foundation, 2026.
[2] NIST CODATA, *Recommended Values of the Fundamental Physical Constants*, 2022.
[3] Daubney, S. *Parkinson's Disease in the Force of Time* (the shared retinoid/RXR node-writer), The Daubney Foundation, 2026.
[4] J. K. Huang et al., *Retinoid X receptor gamma signaling accelerates CNS remyelination*, Nat. Neurosci. 14, 45 (2011).
[5] Intrinsic retinoic-acid synthesis (RALDH2) is required for OPC expansion during remyelination, *Front. Cell. Neurosci.* (2025).
[6] R. J. M. Franklin & C. ffrench-Constant, *Regenerating CNS myelin — from mechanisms to experimental medicine*, Nat. Rev. Neurosci. 18, 753 (2017).
[7] C. Stadelmann et al., *Myelin in the central nervous system: structure, function, and pathology*, Physiol. Rev. 99, 1381 (2019).

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