THE UNIVERSAL FORCE OF TIME · MEDICINE

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 6 (supersedes Rev 5: the disease given in full as medicine knows it; the Epstein–Barr and mimicry findings, the remyelination trials and the retinoid-receptor research credited by name, with the results that did not hold; foreign-number wording removed; the statement on values corrected)

Tau (T) is the living fabric of time itself — the sole substance of which all physical reality is composed. It is generated by the Sun, as by every star for its own system, and flows outward in the form of the DNA double helix, creating matter, which it deposits at nodal points — the planets — and creating life. Every particle, force, wavelength and conscious experience is a structured configuration of T-flow. The conservation law $d\Sigma T = 0$ governs all change: T is never created or destroyed at a node, only redistributed.

THE MYELIN REGISTER, NUMBER FIRST

healthy myelin **1.5 µm** (3/2) · g-ratio **0.6** (3/5) · the edge **½ µm** (1/2) · a severe lesion **0.2 µm** (1/5)

the leap near 70 m/s; the crawl near 5 m/s · the brain's binding beat **40 Hz** ($2^3 \cdot 5$)

quiet the attack before re-enabling the writer

ABSTRACT

About 2.8 million people worldwide live with multiple sclerosis. A nerve does not pass its signal along like a burning fuse — it throws it: the impulse leaps from one bare gap in its insulation to the next, skipping the insulated stretch between. Physiology calls this saltatory conduction, and the gaps the nodes of Ranvier. Multiple sclerosis strips the insulation, myelin, and the leap fails. Medicine has built a generation of medicines that quiet the immune attack; none yet gives the nerve back its lost myelin.

This paper reads multiple sclerosis in the Universal Force of Time, in full. **Part I sets out the disease as medicine knows it** — the leap, the attack, the plaques, the treatments — and what the research has found: infection with the **Epstein–Barr virus** comes before MS and raises its risk many times over (Kjetil Bjornevik, Alberto Ascherio and colleagues, 2022); antibodies against the virus also bind a protein of the brain — **an address misread** (Tobias Lanz, William Robinson and colleagues, 2022); signalling through the **retinoid X receptor** speeds the rebuilding of myelin (Jeffrey Huang, Robin Franklin and colleagues, 2011); and the first trials of remyelinating medicines have shown the nerve signal speeding up again (Ari Green and colleagues, 2017; Jonathan Brown, Alasdair Coles and colleagues, 2021). **Part II reads it in the theory.** T acts at nodes, and the nerve is a node-to-node relay made flesh. **Myelin holds the register coherent across the gap the signal must cross**, and its settings sit on the lattice: healthy myelin about **1.5 µm** [3/2] at a fibre ratio of **0.6** [3/5]; the leap fails below an edge the theory reads at **½ µm** [1/2]; a severe lesion thins to about **0.2 µm** [1/5].

Part III reads the disease as four routes — the signal fails below the edge, the sheath is stripped by a misread address, nothing regrows because the writer has stalled, the window closes as the bare axon dies — each paired with its correction: re-cohere the register at its own **40 Hz** beat [$2^3 \cdot 5$], restore the address the immune system should read, re-enable the writer, act while the axon lives. One law binds them: **quiet the attack before re-enabling the writer, because the attack holds the writer shut.** Twelve propositions, P-MS-1 to P-MS-12, are stated. The research groups are credited by name; the corrective detail is held in the Foundation's clinical reference.

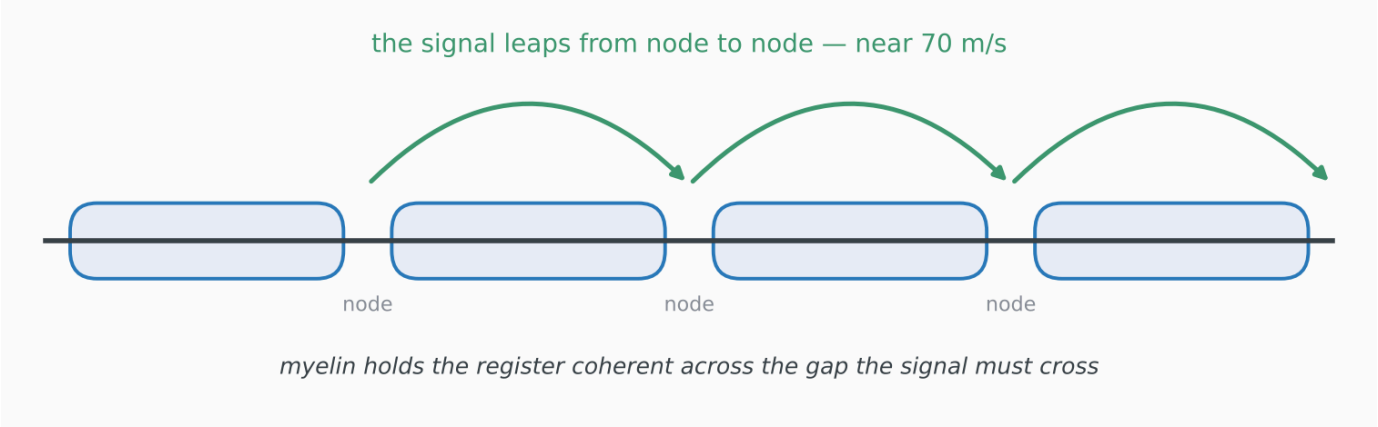


Figure 1 — Healthy myelin lets the impulse leap from node to node, near 70 m/s. T acts at the nodes of Ranvier; myelin holds the register coherent across the gap the signal must cross.

THE TERMS THIS PAPER USES

T, the substance. Tau (T) is the living fabric of time itself, the one substance of which everything is made. It is generated by the Sun and flows outward as the DNA double helix, creating matter and creating life. Everything a body does — a heartbeat, a breath, the holding of its warmth — is T moving in a structured way.

An address. Every value in this framework is a product of powers of 2, 3, 5 and π, and nothing else. That product is the value's address, and the set of all such products is the lattice. A number that needs any other prime is not on the lattice.

Number first. Every value is written with its number first, as a reader meets it, and its address in brackets behind it. The number is what is there; the address is how it is built.

The free decade. Moving a value by a power of ten costs nothing on the lattice, because ten is two times five: the move shifts twos and fives together and changes nothing else. That is why 24 millimetres and 0.024 metres are one address.

The π-degree. The degree of an address is the power of π it carries, and the degree says what kind of thing the value is. An even degree is a **room** — a state, a thing that sits; it pairs, like a pair of circles. An odd degree is a **road** — a transport, a thing that carries; it is the unpaired circle. A half degree is a **door** — a source, which squares to a whole address.

The day, 864. The day is 86,400 seconds [$2^7 \cdot 3^3 \cdot 5^2$], written 864 at its decade. It is the ground state of the DNA helix and the gear through which a per-second value becomes a per-day value.

The time ladder. A rate given per second is carried up the clocks by fixed steps: × 60 to the minute, × 60 to the hour, × 24 to the day. The amount is the same; only the choice of clock changes — the same quantity counted in seconds or counted in days. From the day the orbit is completed one of two ways — × 360 gives the year as a degree value, × 2π gives the year as a radian value. The two answers are two faces of one year, and both are read.

The two gears. Two numbers carry energy between the theory's registers and appear throughout the record. **10,368** [$2^7 \cdot 3^4$] carries an energy in electron-volts to kilojoules per mole. **31,104** [$2^7 \cdot 3^5$] carries an energy in electron-volts to a circumference. When a quantity of the body lands on one of them, it is speaking in the theory's own units.

The veil. The veil is 180/π = 57.29577951308232, the number that turns radians into degrees. In the Universal Force of Time a wavelength in nanometres is an angle in degrees directly; multiply it by π/180 — cross the veil — and the bare value it carries appears.

The register. A register is a level at which T is read. Each organ of the body is a register — a receiver tuned to one face of the time-field — and each cell, molecule and rhythm within it holds an address on that register.

The six faults. A register can fail six ways. It can be **missed** — its target still exists but it has settled at the wrong place; **demoted** — dropped to a lower order while its parts survive; **frozen** — locked at one address and unable to move; **contaminated** — carrying a foreign address; **misdirected** — its own proof-reader turned against it; or

eroded — its nodes deleted. Under dΣT = 0 the field redistributes to the nodes that survive; it does not rebuild an address that has gone.

The conversion grammar. One value takes several forms, and fixed steps move it between them: a frequency divided by 2π is a free fall; a free fall times 49.5035535 [7776/(50π)] is a wavelength; a wavelength times π²/8 is a mass. Run in one line, frequency to mass is a single step of 9.72, and every π cancels.

Free fall. In the Universal Force of Time there is no gravity, and a free fall is not a pull. It is the flow of time at a place, in metres per second, every second — time showing itself in one more form, which current science takes for a gravitational fall. A planet makes one turn; where the turn comes in at less than 24 hours, time makes up the difference with a flow at that place, height or depth, so that the turn and the flow together make 24 hours. For the Earth, the free fall on the celestial register, **9.818362093946918** [$5^{12}/(2^7 \cdot 3^9 \cdot \pi^2)$], times 2400 — the 24-hour day, carried two decades — is the Earth's sidereal rotation, **23,564.06902547260** [$5^{14}/(2^2 \cdot 3^8 \cdot \pi^2)$]: 23 hours, 56 minutes and 4 seconds on the clock.

The three registers, G0, G1 and G2. The record reads the same value at more than one level: G1, the surface register of the Earth; G2, the celestial register; G0, the register below. A value and its neighbour one register up — one level up — stand one **G-bond step** apart, 1.000090150603356 [$5^{10}/(2^4 \cdot 3^9 \cdot \pi^3)$].

The 612 step. A second fixed step, 1.000612842467911 [$2^9 \cdot 3^5 \cdot \pi/5^8$] — helium's weight divided by four (helium divided by four, per nucleon), and 36,864 × 864 × π read at its decade. Where a family of lines stands G1, G2 and then one step further, the further step is this one.

The helical turn. The helical turn, 1.004693930041152 [$5^6/(2^6 \cdot 3^5)$], is the step by which the DNA helix climbs one turn — one revolution of the helix. A value carried "one helical turn up" is multiplied by it.

What a landing is worth. Multiply or divide two lattice numbers and the answer is always on the lattice; that much is guaranteed and is not evidence. What counts is a result that lands on a **named object** the theory had already fixed — the day, body temperature, the heartbeat, the day — at a bare power of ten. Each such landing in this paper is marked as arithmetic or as a landing, in its own sentence.

PART I — MULTIPLE SCLEROSIS AS MEDICINE KNOWS IT

1. The leap

Along every fast nerve fibre the insulation is laid in segments, and between the segments are tiny bare gaps, the **nodes of Ranvier**. The impulse leaps from node to node — **saltatory conduction**, from *saltare*, to leap — which is how a signal crosses a tall body in a fraction of a second. The insulation is **myelin**, wound around the fibre in tight layers by support cells, the oligodendrocytes in the brain and spinal cord. Its thickness is tuned: the ratio of the bare fibre to the whole insulated fibre, the **g-ratio**, sits near **0.6**, the value William Rushton showed gives the fastest conduction. A large insulated fibre carries its signal at tens of metres a second — near 70 m/s; a bare one crawls at a few.

2. The attack

In multiple sclerosis the immune system attacks the myelin of the brain and spinal cord. Where it strips the sheath a **plaque** forms, visible on MRI, and the nerve signal slows or fails: vision blurs, a limb weakens or numbs, balance goes. Most people begin with **relapsing–remitting** disease — attacks that come and then recede — and many later enter a **progressive** phase in which disability accumulates without recovery.

3. The precursors that do not finish

The brain is full of **oligodendrocyte precursor cells**, the most abundant precursor in the central nervous system, and they carry the full programme for making myelin. In MS plaques they gather but often fail to mature into myelinating cells. The repair is blocked, not impossible.

4. The bare axon

A nerve fibre left bare for long loses the support the sheath gives it and eventually degenerates. Once the axon itself is gone, no re-insulation can restore the signal. The loss of axons is the substance of progressive disability.

5. Treatments

A generation of **disease-modifying therapies** suppresses the attack: natalizumab, which blocks immune cells from crossing into the brain and cut relapses by about **68%** in its pivotal trial (Chris Polman and colleagues, 2006); ocrelizumab and ofatumumab, which deplete the B cells that drive the attack; alemtuzumab and cladribine, which reset the immune repertoire. They slow the formation of new lesions. None repairs a lesion that has already formed.

6. What the research has found

These are the results as the research groups reported them; the credit for each is theirs.

A virus comes first. Kjetil Bjornevik, Alberto Ascherio and colleagues followed more than ten million young adults in the US military: infection with the Epstein–Barr virus came before MS, and raised its risk about thirty-two-fold (*Science*, 2022).

An address misread. Tobias Lanz, William Robinson and colleagues found that antibodies made against an Epstein–Barr protein, EBNA1, also bind a protein of the brain's support cells, GlialCAM — the immune system mistaking a piece of the body for the virus it resembles (*Nature*, 2022).

The writer of myelin. Jeffrey Huang, Robin Franklin and colleagues found that signalling through the **retinoid X receptor γ** drives precursor cells to mature and speeds the rebuilding of myelin, and that an RXR-activating medicine enhanced remyelination in animals (*Nature Neuroscience*, 2011).

First repair in people. In the ReBUILD trial, Ari Green and colleagues found that the antihistamine **clemastine** shortened the delay of the visual nerve signal in people with long-standing optic nerve damage — the first evidence of remyelination by a medicine in humans (*The Lancet*, 2017). In the CCMR One trial, Jonathan Brown, Alasdair Coles and colleagues gave **bexarotene**, an RXR activator; its main imaging measure was not met, but the visual nerve signal sped up, and the medicine was poorly tolerated (*The Lancet Neurology*, 2021).

That is multiple sclerosis as medicine knows it, given in full because what follows reads every part.

PART II — MULTIPLE SCLEROSIS IN THE UNIVERSAL FORCE OF TIME

7. The signal that leaps

We picture a nerve impulse running down a fibre like a flame along a fuse. It is nothing of the kind: the signal jumps. The Universal Force of Time recognises this at once, 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, never by what that thing was for.

8. Myelin holds the register in the gap

Medicine treats myelin as a passive wrapper that stops the current leaking. The theory 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 settings carry the lattice. Healthy myelin runs about **1.5 µm** thick [3/2], wound to the fibre ratio of **0.6** [3/5] at which conduction is fastest. These are not free parameters; in the theory they are the settings at which the register between the nodes holds.

9. The edge is a switch, not a slope

While the myelin stays above about **½ µm** [1/2], coherence holds and the signal still jumps. Below it the register fails between nodes, and the impulse falls back to crawling down the bare fibre — from near 70 m/s toward 5 m/s in a severe lesion thinned to about **0.2 µm** [1/5]. The lesion boundary is a **phase transition** of the register: on one side the leap works, on the other it does not, with 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 edge is tipped below by inflammation (the symptom appears) and back above by partial repair (it remits). **Relapsing–remitting MS is a register flickering across its phase transition; the progressive phase is the register settling below the edge for good.**

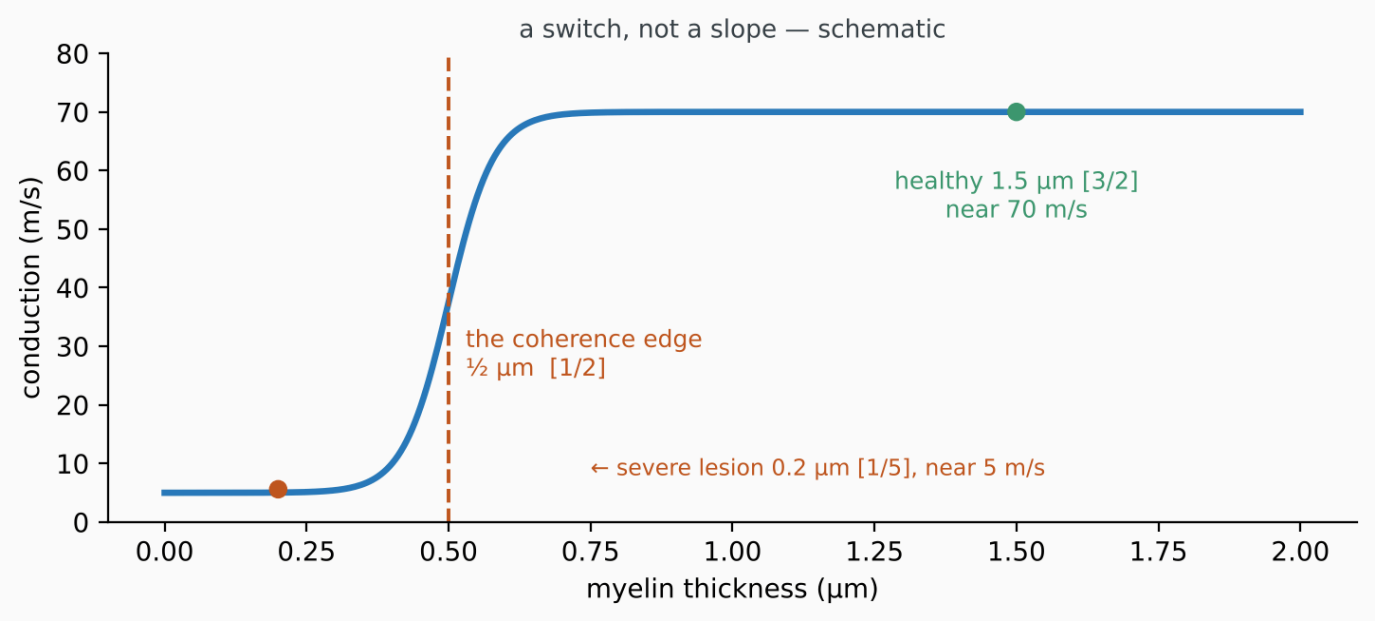


Figure 2 — A schematic of the coherence edge: conduction holds near 70 m/s at healthy thickness, 1.5 µm [3/2], and collapses below the edge the theory reads at ½ µm [1/2]; a severe lesion thins to 0.2 µm [1/5]. The transition is a switch, not a slope.

10. The attack is a misread address

The attack that strips the sheath is, in the theory, an **autoimmune address misdirection** — the same family of failure as Type 1 diabetes, rheumatoid arthritis and, from the other side, HIV: the immune system, the body's proof-reader of addresses, misreads an address and attacks the wrong target. The research has now caught the misreading in the act. An antibody made against the Epstein–Barr virus also binds a protein of the brain's own support cells: the proof-reader has filed a piece of the self under the virus it resembles. **The disease is not, at root, a destruction of tissue; it is a misfiling of identity that causes the destruction.** That places the route in a definite class, with a correction shared across the misdirection family rather than invented for MS alone.

11. The writer has stalled

The third face is why the damage does not simply heal. The precursor cells hold the whole programme in their genome — under $d\Sigma T = 0$ the inscription is preserved — but the **writer** that reads it out has stalled. The writer is the retinoid cascade: retinoic acid made by RALDH2, signalling through the retinoid X receptor — and the theory identifies it as the very same writer stalled in Parkinson's. **Two neurodegenerations, one writer.** It is why the medicines that quiet the attack slow new lesions but cannot repair old ones: they address the attack and leave the blocked transition untouched. The research confirms the writer: RXR signalling drives remyelination in animals, and an RXR activator sped the visual signal in people, though its main measure was not met and its side effects were heavy.

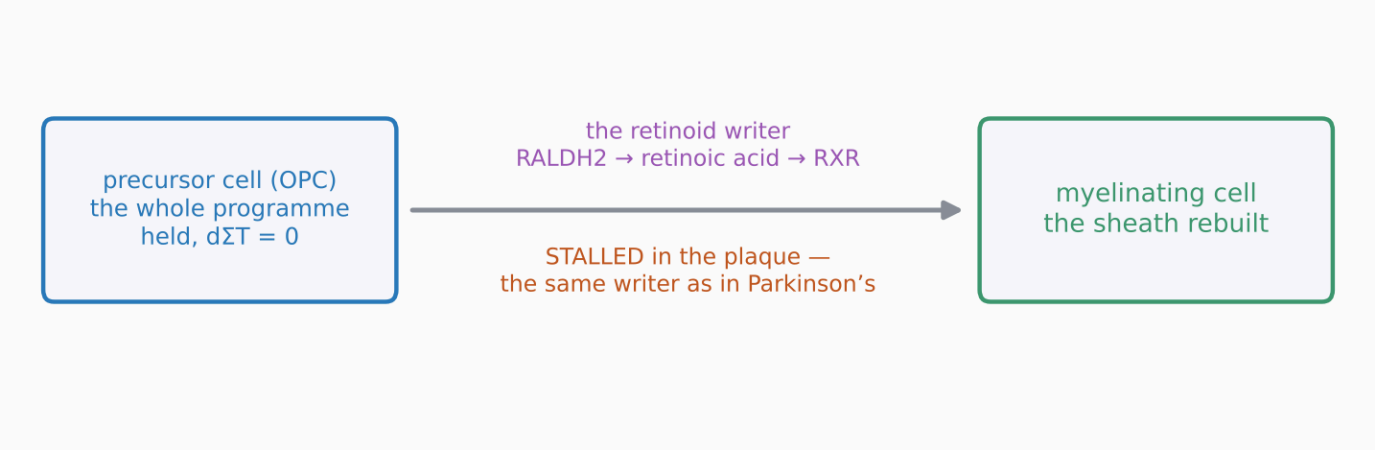


Figure 3 — Remyelination is blocked, not impossible: the precursor cells hold the whole programme (dΣT = 0), and the retinoid writer — RALDH2 to retinoic acid to RXR, the same writer stalled in Parkinson's — is the route that re-enables it.

12. The 40 Hz beat

The brain has a frequency at which it holds wide-area coherence: the gamma beat at **40 Hz** [$2^3 \cdot 5$]. In the theory, a disease of lost coherence between nodes is a disease of that register, and the theory expects the beat to be weakened across the brain rather than in one spot, as a lost timing register would be. The rhythm can be supplied from outside — the research on 40 Hz light and sound in the companion paper on neurodegeneration shows it entrains the brain — but its use in MS has not yet been tested.

13. Where these results come from

The research is theirs. The Epstein–Barr finding is Kjetil Bjornevik's and Alberto Ascherio's and their colleagues'; the mimicry Tobias Lanz's and William Robinson's; the retinoid receptor result Jeffrey Huang's and Robin Franklin's; the clemastine trial Ari Green's group's; the bexarotene trial Jonathan Brown's and Alasdair Coles's; the natalizumab trial Chris Polman's and his colleagues'; the fibre ratio William Rushton's. It was their results that pointed this paper to these parts of the problem, and their work is the supporting evidence on which these sections are built. What this paper adds is the theory's reading — myelin as the register of the leap, the edge as a phase transition, the attack as a misread address, the shared writer, and the 40 Hz beat — offered to those groups, and to any team so minded, to test.

PART III — FOUR ROUTES, FOUR CORRECTIONS

14. Four ways the leap is lost

A medical paper of the Universal Force of Time acknowledges the illness, reads the problem as the distinct routes by which it arises, and pairs each route with the correction that would set it right. The four routes here are four real ways the same disease takes a fibre down, and a given patient may be losing the leap by one, by several or by all four at once.

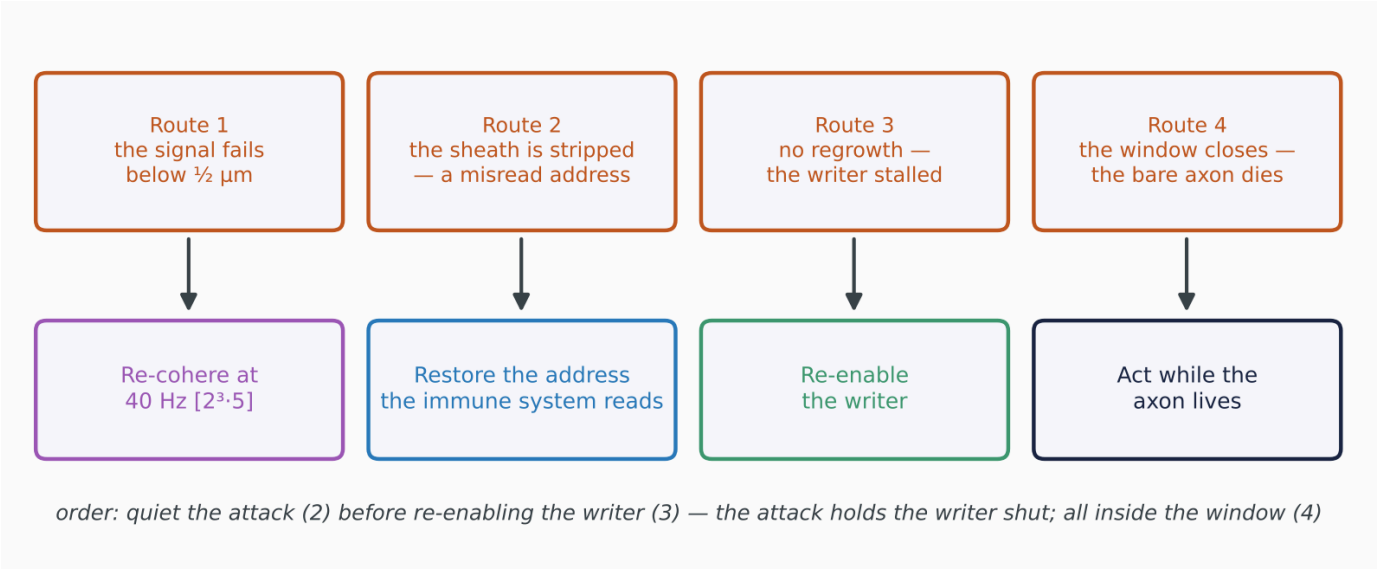


Figure 4 — The four routes by which myelin's register is lost, each paired with its correction. Correction 2 comes before correction 3 by the order law, and all of them inside the window that Route 4 keeps.

15. Route 1 — the signal fails below the edge

The register loses coherence between nodes below ½ µm, and the leap fails.

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 it — at the frequency the brain itself uses to hold wide-area coherence, **40 Hz** [2³·5]. The value is printed; how any rhythm is given is held in the Foundation's clinical reference.

16. Route 2 — the sheath is stripped by a misread address

The proof-reader misfiles myelin under a foreign address and strips it.

Correction 2 — restore the address the immune system should read. The correction for a misdirection is not only to suppress the messenger but to restore the address the proof-reader should be reading, so it stops attacking myelin as foreign. Today's medicines act on this route from the outside, blunting the attack; the theory aims at the misfiling itself — and the Epstein–Barr finding shows where the misfiling begins. It is the same correction principle the series carries for Type 1 diabetes and rheumatoid arthritis: one family, one route home. The detail is held in the Foundation's clinical reference.

17. Route 3 — no regrowth, because the writer has stalled

The precursors hold the programme, but the retinoid writer that reads it out has stopped.

Correction 3 — re-enable the writer, so the precursors re-lay myelin above the edge. The correction is to lift the stall on the retinoid cascade so the precursor cells already present can mature and rebuild myelin back above ½ µm. The programme they need is intact; only the writer is shut. This is the frontier no approved medicine has yet reached: every current therapy works on Route 2; Route 3 is still open, and the first trials have begun to open it. The detail is held in the Foundation's clinical reference.

18. Route 4 — the window closes

A fibre left bare for long degenerates, and a lost axon cannot be re-insulated.

Correction 4 — act while the axon still lives. Here timing is itself the correction. Relapsing–remitting MS is reversible because the axon beneath the stripped sheath is still there, waiting to be re-insulated; once it is gone, nothing can restore its signal. The same irreversibility law governs every node the body cannot remake.

19. The order law

Correction 2 must come before correction 3. Active inflammation suppresses the maturing of the precursor cells — **the attack holds the writer shut** — so quieting the misdirection is the permitting condition for remyelination, not merely the humane first step. And all of it must land inside the window that Route 4 keeps. It is

the same order the theory finds across neurodegeneration: clear the noise from the register before asking the writer to write.

20. The resolution — the clinical trial is the next step

We have acknowledged the illness — a disease long described by what it destroys and never by what that thing was for. We have read myelin as the register of the leap, its settings on the lattice; the lesion edge as a phase transition, which is why the disease relapses and remits; the attack as an address misread, now caught in the act; the failure to regrow as a stalled writer shared with Parkinson's; and the progressive phase as the window closing. We have named four routes and given for each its correction, bound by the order law.

The corrections are stated as principles precisely because the next step is not to prescribe them to a reader but to put them to a clinical trial.

PROPOSITIONS

- **P-MS-1.** Myelin is the register of saltatory conduction: it holds coherence between nodes so the impulse can leap; T acts at the nodes of Ranvier.
- **P-MS-2.** The healthy settings sit on the lattice: thickness about 1.5 µm [3/2], fibre ratio 0.6 [3/5].
- **P-MS-3.** The theory reads the coherence edge at ½ µm [1/2]; below it the leap fails and conduction falls toward a crawl; a severe lesion thins to 0.2 µm [1/5].
- **P-MS-4.** The edge is a phase transition: relapsing–remitting MS is the register flickering across it; the progressive phase is the register settled below it.
- **P-MS-5.** The attack is an autoimmune address misdirection — caught in the act in the Epstein–Barr mimicry finding — in the family of Type 1 diabetes and rheumatoid arthritis.
- **P-MS-6.** The precursors hold the programme ($d\Sigma T = 0$); the retinoid writer through the retinoid X receptor has stalled — the same writer as in Parkinson's.
- **P-MS-7.** The brain's binding beat, 40 Hz [$2^3 \cdot 5$], is the register's own; the theory predicts it weakened brain-wide in MS.
- **P-MS-8.** Routes 1 and 2: the signal fails below the edge; the sheath is stripped. Corrections: re-cohere at 40 Hz; restore the address the immune system should read.
- **P-MS-9.** Routes 3 and 4: no regrowth; the window closes. Corrections: re-enable the writer; act while the axon lives.
- **P-MS-10.** Order law: correction 2 before correction 3 — the attack holds the writer shut.
- **P-MS-11.** Current medicines act on Route 2 only; Routes 1, 3 and 4 are the open frontier, and the first remyelination trials have begun on Route 3.
- **P-MS-12.** A lost axon cannot be re-insulated: timing is itself the fourth correction.

REFERENCES

1. Daubney, S. *Health as Optimal T-Flow — The Universal Force of Time Medical Framework*, Revision 5. The Daubney Foundation (2026).
2. Daubney, S. *Parkinson's Disease*, Revision 6 (the shared retinoid writer), and *Neurodegenerative Diseases*, Revision 6 (the 40 Hz research). The Daubney Foundation (2026).
3. Walton, C. *et al*. Rising prevalence of multiple sclerosis worldwide: insights from the Atlas of MS, third edition. *Multiple Sclerosis Journal* 26, 1816–1821 (2020).
4. Rushton, W. A. H. A theory of the effects of fibre size in medullated nerve. *Journal of Physiology* 115, 101–122 (1951).
5. Polman, C. H. *et al*. A randomized, placebo-controlled trial of natalizumab for relapsing multiple sclerosis. *New England Journal of Medicine* 354, 899–910 (2006).
6. Bjornevik, K. *et al*. Longitudinal analysis reveals high prevalence of Epstein-Barr virus associated with multiple sclerosis. *Science* 375, 296–301 (2022).
7. Lanz, T. V. *et al*. Clonally expanded B cells in multiple sclerosis bind EBV EBNA1 and GlialCAM. *Nature* 603, 321–327 (2022).

8. Huang, J. K. *et al*. Retinoid X receptor gamma signaling accelerates CNS remyelination. *Nature Neuroscience* 14, 45–53 (2011).
9. Green, A. J. *et al*. Clemastine fumarate as a remyelinating therapy for multiple sclerosis (ReBUILD): a randomised, controlled, double-blind, crossover trial. *The Lancet* 390, 2481–2489 (2017).
10. Brown, J. W. L. *et al*. Safety and efficacy of bexarotene in patients with relapsing-remitting multiple sclerosis (CCMR One): a randomised, double-blind, placebo-controlled, parallel-group, phase 2a study. *The Lancet Neurology* 20, 709–720 (2021).
11. Franklin, R. J. M. and ffrench-Constant, C. Regenerating CNS myelin — from mechanisms to experimental medicine. *Nature Reviews Neuroscience* 18, 753–769 (2017).

APPENDIX — EVERY NUMBER, REBUILT

Every value can be rebuilt with any calculator, from twos, threes, fives and π. The script M_calc.py asserts every station; it passed in full.

```
THE MYELIN REGISTER, NUMBER FIRST
   healthy myelin                about 1.5 um              3/2
   fibre ratio (g)               0.6                       3/5
   the coherence edge (theory)   0.5 um                    1/2
   severe lesion                 about 0.2 um              1/5
   binding beat                  40 Hz                     2^3.5
```

A NOTE ON THE NUMBERS

Throughout this paper a value is given first as the plain physical reading — a thickness, a ratio, a frequency — and only then, in brackets, as its place on the {2,3,5,π} lattice. Where a value is the theory's reading rather than a measurement, the paper says so. The corrections are stated as principles; how any rhythm or medicine is given is held in the Foundation's clinical reference and is not prescribed here.

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

© 2026 Stephen Daubney | The Daubney Foundation. This paper may be freely quoted and referenced provided full attribution is given to Stephen Daubney and The Daubney Foundation as the source.