THE UNIVERSAL FORCE OF TIME

Liver Fibrosis

The Organ That Rebuilds Itself — Four Routes by Which a Collagen Scaffold Is Driven Off the Lattice, and the Four Corrections That Set the Rebuilder Free

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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

The liver is the body's great rebuilder — the one major organ that can regrow a lost portion of itself, surviving the loss of up to seventy parts in a hundred — and in the Force of Time it is the **T_E converter**, turning the electrical face of the time-field into the body's chemistry. To run hundreds of conversions at once it holds its tissue in exact register on a scaffold of collagen whose spacing sits squarely on the lattice: healthy type-I collagen repeats at **640 Å** ($2^7 \times 5$), a clean {2,5} node, held beneath a stability ceiling at **864 Å** ($2^5 \times 3^3$) — the very number that builds the day, since 864×100 s = 86,400 s = one solar day. 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**, pairing **each route with the one correction that would realign it**. Route one — the **keeper wakes**: the hepatic stellate cell, the liver's retinol store, dumps its retinol and turns into a collagen-laying myofibroblast — corrected by restoring the retinoid programme-restoration signal it abandoned, a signal native to the liver's own biology. Route two — the **scaffold over-extends**: the activated cell lays collagen past the 864 Å ceiling, into the empty gap beyond, where the over-extended period freezes — corrected by releasing the lock and letting the period settle back onto its 640 Å node. Route three — **one cell, a self-feeding loop**: many injuries (alcohol, hepatitis B/C, fatty-liver disease, autoimmune attack, biliary disease, iron overload) all converge on that single cell, and the stiffening scar itself recruits more cells — corrected by healing the upstream road and breaking the feed-forward loop. Route four — the **ladder runs to the wall**: the damage climbs F0→F4 as coherence falls and collagen rises from about 2% to 65% of liver mass; short of cirrhosis the drift is displaced, not deleted, but at F4 the architecture is destroyed — corrected by reaching it before the wall and letting the liver's standing regeneration finish. The corrections carry an **order law**: quiet the source (routes one and three) before releasing the scaffold (route two) — settling collagen back to 640 while the cell still pours new collagen is futile — and all of it must land before the F4 wall. The off-lattice signatures the tissue picks up on the way out (the prime 67 in the 670 Å soft edge; primes past 864) are the fingerprint of having left the lattice, never destinations it was climbing toward. Ten propositions, P-LIVER-1 to P-LIVER-10, are given. The mechanism is given in full and at full precision; corrective detail is 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 Organ That Rebuilds Itself

Of all the body's organs, the liver alone can regrow. Remove most of it — surgeons routinely take away most of a liver, and the body has survived the loss of up to **seventy parts in a hundred** — and, given the chance, it rebuilds the missing tissue and resumes its work within weeks. This is the organ the ancients made a symbol of endless renewal, the one Prometheus was punished with, eaten each day and whole again by morning. They were not wrong. Which makes liver fibrosis — the slow stiffening of that self-renewing tissue into scar — one of the most *hopeful* diseases to understand, because the engine of repair is already built in. The Force of Time's task is to say exactly what stiffens, and how to set the rebuilder free. In the Force of Time the liver is the body's **T_E converter** — the organ that turns the electrical face of the time-field into the body's chemistry, building and breaking the blood proteins, banking and releasing the body's sugar, dismantling what is spent, and storing the body's reserve of retinol. To do all this in parallel it must hold its tissue in exact register, and it does so on a scaffold of collagen. So the story of liver fibrosis is, at root, the story of that scaffold — what spacing it holds when healthy, what happens when the spacing drifts, and why, uniquely, the liver can rebuild it once the drift is released.

2 Where Medicine Stands

Fibrosis is not a single disease so much as a single ending. Chronic injury reaches the liver by many roads — too much alcohol; the hepatitis B and C viruses; the fatty-liver disease that follows the modern diet (NAFLD, and its inflamed form MASH); the body's own immune system turning on the organ; the slow strangling of the bile ducts; and the iron overload of haemochromatosis. Medicine files these under different names and treats them by different means — and medicine is right that removing the cause can halt the journey. But it has no agent that reliably *reverses* established scar, and it reads the scar itself as a more-or-less one-way road toward cirrhosis. The clinic grades the damage on the **METAVIR** scale, F0 through F4, and tracks it from the outside through the rising liver enzymes ALT, AST and GGT — but once the tissue has stiffened, conventional medicine largely waits and watches. The Force of Time looks past the many names to the common ending, and past the pessimism to the one fact that changes everything: this is the organ that rebuilds. The whole question is what holds the scar in place, and whether that hold can be released before the final stage closes the door.

3 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 collagen scaffold is driven off the lattice — 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 faces of one process: a keeper cell that wakes and starts laying scar, a scaffold that over-extends and freezes off its node, a single cell that every injury converges on and that then feeds its own activation, and a coherence ladder that climbs toward a wall past which displacement becomes deletion. A given patient is being scarred by all four at once, feeding one another. What follows names each route, then its correction, in order. Hold the whole shape in view (Figure 1): four problems on the left, four corrections on the right, bound by one order law, resolving into a single next step.

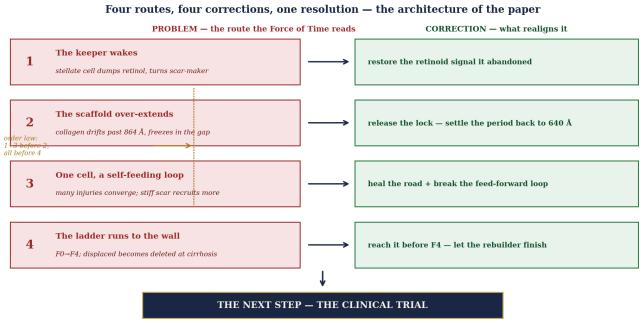

Figure 1 — The architecture of the paper: each of the four routes by which the collagen scaffold is driven off the lattice is paired with the one correction that realigns it; the source must be quieted (corrections 1 and 3) before the scaffold is released (correction 2), and all of it must land before the F4 wall (correction 4); the whole structure resolves into the clinical trial.

Route 1 — The Keeper Wakes: the retinol store turns scar-maker

The cell at the centre of the whole story lives in a thin space between the liver's working cells and its blood channels — the **space of Disse**. In health it is quiet, and it has a quiet job: it is the body's **hepatic stellate cell**, the retinol store, holding the reserve of vitamin A in bright lipid droplets and releasing it to the rest of the body on demand. The liver is the organ most directly in contact with the **retinoid** class — the family of molecules that, in the Force of Time, carries the body's programme-restoration signal. When injury signals arrive — **TGF-β** above all, a molecule whose own mass sits firmly on the lattice at **25,000 Da** ($2^3\times5^5$), a clean {2,5} address — the quiet cell transforms (Figure 2). It empties its retinol droplets, changes shape into a contractile **myofibroblast** marked by α-smooth-muscle actin, and begins to pour out collagen I and III. The loss of the retinol is not incidental: it is part of the drift itself — the cell abandons the very signal that holds it quiet. TGF-β is *on* its node; it is not itself off-lattice. It is the signal that drives the cell, and through the cell the collagen, off-node.

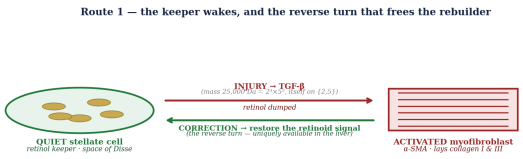

Figure 2 — On injury the hepatic stellate cell empties its retinol store and turns into a collagen-laying myofibroblast; the reverse turn — restoring the retinoid signal it abandoned — is uniquely available in the liver, because the organ is already in continuous contact with that signal.

Correction 1 — restore the retinoid signal the cell abandoned

If the keeper woke because it dropped the signal that held it quiet, the correction is to give that signal back. The corrective family has a natural home: because the stellate cell is the body's retinol keeper, the molecule family that returns it to quiet is **native to the liver's own biology** — the organ is already in continuous contact with the programme-restoration signal it lost, not a stranger forced upon it. Restore the signal and the cell can make the reverse turn, from scar-maker back to quiet keeper. This is why the liver is the natural first place to look for a correction in the whole of Force-of-Time medicine. The principle is restoration of the abandoned signal; the specific corrective and its delivery are held in the Foundation's reference, not prescribed here.

Route 2 — The Scaffold Over-Extends: collagen drifts past its node and freezes

The second route is what the woken cell actually *does* to the tissue. Under the microscope, type-I collagen shows a characteristic banding — the **D-period** — and in health it sits at **640 Å** ($2^7\times5$), a clean {2,5} node. Its soft outer edge at **670 Å** ($2\times5\times67$) already carries the first hint of drift — the 67 is a prime, and a prime has no home on the Earth register's {2,3,5} lattice. Both sit beneath a deeper anchor: **864 Å** ($2^5\times3^3$), the collagen stability ceiling — the maximum stable register address before over-extension begins. The healthy D-period is **74.07%** (640/864) of that ceiling, comfortably below the wall. And the anchor is no arbitrary number: 864 ($2^5\times3^3$) is one of the most fundamental nodes in the whole lattice — it builds the day, because 864×100 seconds is exactly 86,400 s = one solar day. The same number that paces the turning of the Earth sets the spacing of the collagen that holds the liver together. Fibrosis is this scaffold pushed off its node: the activated cell lays collagen at periodicities *greater than 864 Å* — past the ceiling, into the empty gap beyond it (Figure 3) — where the over-extended period **freezes**. Where there was flowing conversion there is now dead stiffness. That is the Force-of-Time definition of scar: not new material so much as the scaffold driven off its {2,3,5} node and held in the gap, trading functional T-flow for rigidity.

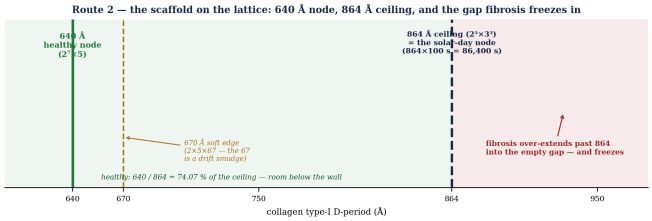

Figure 3 — The collagen scaffold as lattice addresses: the healthy 640 Å node ($2^7\times5$) and its 670 Å soft edge sit beneath the 864 Å ceiling ($2^5\times3^3$ — the solar-day node); fibrosis over-extends the period past 864 into the empty gap and freezes there. The primes that appear in the drifted readings are the smudge of having left the lattice, not a destination.

Correction 2 — release the lock and let the period settle back onto 640 Å

A drifted register is **displaced, not deleted**. The correction is to release the lock that holds the collagen out past 864 and let the over-extended period settle back onto its **640 Å** ($2^7\times5$) node, restoring T-flow — and then the liver, alone among the organs, does the rest. But this correction cannot be applied alone: settling the laid collagen back to 640 while the keeper is still actively pouring out new collagen is futile, which is why the order law puts the source first. The principle is that a register driven off its node can be returned to it; the means are held confidentially pending trials.

Route 3 — One Cell, a Self-Feeding Loop: many roads, one ending, then amplification

The third route is what turns a local injury into a relentless disease. First, the convergence: every one of the many roads to fibrosis — alcohol, hepatitis B and C, NAFLD/MASH, autoimmune attack, biliary disease, haemochromatosis — arrives at the *same place*, the activation of that single cell type, the hepatic stellate cell (Table 1). Medicine treats six diseases; the Force of Time sees one ending reached by six roads. Then, the amplification: once a patch of tissue stiffens, the stiffness itself is a signal. The hardened, over-extended matrix mechanically activates more quiet stellate cells, which lay more collagen, which stiffens more tissue — a **feed-forward loop** in which the scar recruits its own scar-makers. This is why fibrosis, once well underway, can advance even after the original injury has eased: the disease has learned to feed itself. The convergence is why so many different injuries look the same in the end; the loop is why the ending, once reached, accelerates.

Correction 3 — heal the road, and break the feed-forward loop

The correction is twofold and matches the route. **Heal the road**: remove the upstream cause — the alcohol, the virus, the metabolic load, the iron — so no fresh activation signal arrives. Medicine already does this part well, and the Force of Time affirms it: heal the road and the journey can be stopped. But healing the road is not enough once the loop is running, so the second half is to **break the feed-forward loop** — to interrupt the cycle in which stiff matrix wakes more cells — so the scar stops recruiting its own makers. Quieting the source this way (together with Correction 1) is the precondition for Correction 2: only once no new collagen is being laid can the existing scaffold be released back to its node and stay there. The principle is removal of cause and interruption of the loop; specifics are held in the Foundation's reference.

Route 4 — The Ladder Runs to the Wall: displaced becomes deleted at cirrhosis

The fourth route is the clock on all the others. The damage climbs in stages, and medicine already grades them: the METAVIR scale runs F0 (no fibrosis) through F4 (cirrhosis). The Force of Time reads the scale as a **coherence ladder** (Figure 4). At F0, collagen is about **2%** of liver mass and T-register coherence is near total — the hepatic nodes hold their natural 640 Å address. As fibrosis advances the collagen rises and the coherence falls, through about **38%** at F3, until at F4 collagen has reached about **65%** of liver mass and coherence has collapsed toward the floor — the scaffold has overwritten the working tissue. The clinic tracks the same descent from the outside through the liver enzymes **ALT, AST and GGT** — the Force of Time's register-disruption markers — normally below 40 U/L and rising stage by stage, with GGT the most sensitive of the three. When the scaffold drifts, the working cells leak their enzymes into the blood, and the rising numbers are the drift made visible in a blood test. The decisive fact is the wall. Short of cirrhosis the drift is displaced, not deleted, and the rebuilder stands ready; but at F4 the architecture is *destroyed* rather than merely displaced — the channels re-routed, the structure nodular — and the register can no longer be re-coupled. There dΣT=0 redistributes to what survives rather than rebuilding what is lost. Drift is reversible; deletion is not.

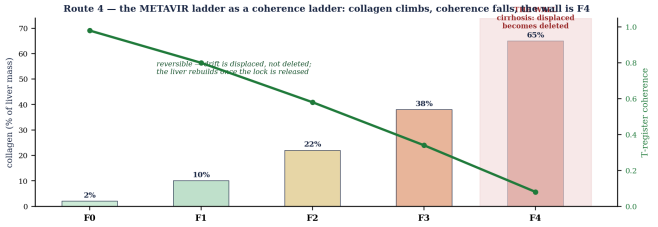

Figure 4 — The METAVIR stages F0→F4 as a coherence ladder: collagen content climbs from about 2% to about 65% of liver mass as register coherence falls toward the floor; short of cirrhosis the drift is displaced and reversible, but F4 is the wall where displacement has become deletion.

Correction 4 — reach it before the wall, and let the rebuilder finish

The correction is timing itself: **reach the drift before F4**, while it is still displaced, and the liver's standing power to regenerate finishes the job — replacing scar with working tissue once the lock is released. This makes early recognition decisive, and it gives the cheapest possible test a sharp meaning: the rising ALT/AST/GGT, GGT most sensitive, are the drift made visible early enough to act inside the window where the tissue is still displaced, not deleted. This is the completing correction — it succeeds only because the other three have been applied before the wall. The principle is to act within the reversible window; the means are held confidentially pending trials.

4 The Order Law, and the 864 Node Across the Body

The four corrections are not freely interchangeable. **Quiet the source before you release the scaffold**: Corrections 1 and 3 — return the keeper to quiet and break the feed-forward loop — must precede Correction 2, because settling the over-extended collagen back onto its 640 Å node while the cell is still actively laying new collagen simply re-drifts it. Stop the laying, then release the laid. And **all of it must land before the wall**: Correction 4 is the clock — every step has to be taken before F4, because past cirrhosis the register is deleted, not displaced, and there is nothing left to re-couple. So the sequence the theory insists on is: heal the road and quiet the keeper, break the loop, release the scaffold back to 640, and let the rebuilder finish — all inside the reversible window. The collagen anchor that sits at the centre of this is not even local to the liver. The same **864** ($2^5 \times 3^3$) node that here holds the hepatic scaffold also builds the day (86,400 s), sets the periodicity of joint cartilage in the arthritis framework, and forms the collagen plaque in Peyronie's disease — one master pivot, several tissues. A correction that learns to release a drifted 864 register in one tissue is, in principle, learning to release it in all of them — which is the Force of Time's deepest claim made concrete: the same few numbers, here just {2,3} as $2^5 \times 3^3$, structure things that look utterly unrelated.

5 A Note on the Primes — Fingerprints, Not Destinations

One point an earlier reading got backwards is worth stating plainly, because it matters for how the corrections are aimed. The disease is **not** the tissue climbing onto some special prime. Seven, sixty-seven, any prime — these never name a place on the Earth register's {2,3,5} lattice; they are the fingerprint of a register that has *left* it. The 67 in the soft edge at 670, the primes that appear once the period drifts past 864 — these are the smudges a register picks up as it wanders off the clean {2,3,5} grid, the mark of having left home, never a destination the tissue was travelling toward. Fibrosis is the period drifting off 864 into the gap, and the primes that show up in the readings are the mark of that drift, never its goal. The correction is therefore not to push the tissue toward any number but to release it *back* to the clean node it came from — 640 Å ($2^7 \times 5$).

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 — and the hopeful fact at its centre, that this is the one organ that rebuilds; we have read the problem as four distinct routes by which the scaffold is driven off the lattice — the keeper wakes, the scaffold over-extends and freezes, one cell that every injury converges on then feeds its own activation, and the coherence ladder that climbs to a wall past which displacement becomes deletion; we have given, for each, the Force-of-Time correction that would realign it — restore the retinoid signal, release the scaffold back to 640, heal the road and break the loop, act inside the reversible window; 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 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. The trial is what decides which routes carry the cure: it may prove that quieting the keeper alone lets the rebuilder reverse the scar, or that the scaffold must be actively released, or that all four, in their proper order, are needed — and that is exactly what a trial is for. Liver fibrosis is usually told as scarring that builds toward cirrhosis, slow and largely one-way. The Force of Time tells it as a scaffold pushed off its node: collagen that should hold 640 Å ($2^7\times5$) over-extended past the 864 Å ($2^5\times3^3$) anchor and frozen in the gap, the liver's T-flow crystallising stage by stage as coherence falls. And it tells it hopefully, because the drift is not a deletion and the liver is the one organ that rebuilds: release the lock, let the collagen settle back onto 640, restore the flow, and the rebuilder finishes the job — provided it is reached before cirrhosis closes the door. We give the mechanism in full and at full precision, and we stand by the figures.

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Table 1 — Many Causes, One Common Ending

Every road to fibrosis converges on the activation of a single cell type — the hepatic stellate cell. Medicine treats six diseases; the Force of Time reads one ending reached by six roads (Route 3).

Road to injury	What it is	Common ending
Alcohol	sustained alcohol-related liver injury	hepatic stellate-cell activation
Hepatitis B / C	chronic viral infection of the liver	hepatic stellate-cell activation
NAFLD / MASH	fatty-liver disease and its inflamed form	hepatic stellate-cell activation
Autoimmune	the immune system attacking the organ	hepatic stellate-cell activation
Biliary disease	slow loss of the bile ducts (e.g. PBC)	hepatic stellate-cell activation
Haemochromatosis	iron overload in the tissue	hepatic stellate-cell activation

Appendix A — The Four Routes and Their Corrections

Each route the Force of Time reads in liver fibrosis, paired one-to-one with the correction that realigns it. Order law: quiet the source (corrections 1 and 3) before releasing the scaffold (correction 2); all of it must land before the F4 wall (correction 4). The four corrections resolve into the clinical trial.

#	Problem route	{2,3,5} reading	Correction (principle)
1	The keeper wakes — stellate cell dumps retinol, turns myofibroblast	TGF-β = $2^3\times5^5$ (on {2,5})	Restore the retinoid programme-restoration signal it abandoned
2	The scaffold over-extends — collagen drifts past 864 Å, freezes in the gap	640 Å = $2^7\times5$; ceiling 864 Å = $2^5\times3^3$	Release the lock; let the period settle back onto its 640 Å node
3	One cell, a self-feeding loop — many injuries converge; stiff scar recruits more	—	Heal the road (remove cause) + break the feed-forward loop
4	The ladder runs to the wall — F0→F4; displaced becomes deleted at cirrhosis	collagen ~2% → ~65% of liver mass	Reach it before F4; let the liver's regeneration finish

Appendix B — The Collagen Scaffold as Lattice Addresses

The healthy scaffold and the stages of its drift. Every healthy value is a clean {2,3,5} form; the primes (the 67) appear only in the drifted readings, as fingerprints of having left the lattice. The physical number is the hero; the lattice form is the address.

Feature	Value	Lattice address	Read
Healthy D-period	640 Å	$2^7\times5$	the clean {2,5} node
Soft outer edge	670 Å	2×5×67	first off-lattice drift (the prime 67)
Stability ceiling	864 Å	$2^5\times3^3$	solar-day node (864×100 s = 86,400 s); over-extension wall
Healthy / ceiling	74.07%	640 / 864	room below the wall
TGF-β mass	25,000 Da	$2^3\times5^5$	the activating signal — itself on {2,5}, not off-node
F0 collagen	~2%	—	near-total coherence; nodes on 640 Å
F3 collagen	~38%	—	advanced drift; still displaced, not deleted
F4 collagen	~65%	—	cirrhosis — architecture destroyed; the wall

Appendix C — The Ledger

Table C1 — Propositions P-LIVER-1 … P-LIVER-10

#	Proposition
P-LIVER-1	The liver is the T_E converter and the body's sole self-regenerating major organ (up to ~70% regrows); its tissue is held on a collagen T-register scaffold.
P-LIVER-2	Many causes, one ending (Route 3, convergence): alcohol, hepatitis B/C, NAFLD/MASH, autoimmune disease, biliary disease and haemochromatosis all converge on hepatic stellate-cell activation.
P-LIVER-3	ROUTE 1 — the keeper wakes. The stellate cell is the body's retinol store (space of Disse); on injury (TGF-β, mass 25,000 Da = $2^3\times5^5$, itself on {2,5}) it dumps its retinol, becomes an α-SMA myofibroblast, and lays down collagen I and III. CORRECTION 1: restore the retinoid programme-restoration signal it abandoned — native to the liver's own biology.
P-LIVER-4	ROUTE 2 — the scaffold over-extends. Healthy type-I collagen D-period 640 Å = $2^7\times5$ (clean {2,5}); soft edge 670 Å = 2×5×67 (the prime 67 is the first off-lattice smudge). Stability ceiling 864 Å = $2^5\times3^3$ (pure {2,3}); healthy = 640/864 = 74.07% of the ceiling. 864 is the solar-day node (864×100 s = 86,400 s). Fibrosis = collagen laid at periodicities > 864 Å, pushed off its node into the empty gap and frozen. CORRECTION 2: release the lock; let the period settle back onto 640 Å.

#	Proposition
P-LIVER-5	ROUTE 3 — one cell, a self-feeding loop. The convergence (P-LIVER-2) plus amplification: stiff over-extended matrix mechanically activates more quiet stellate cells, so the scar recruits its own makers and fibrosis can advance after the original injury eases. CORRECTION 3: heal the road (remove the cause) AND break the feed-forward loop.
P-LIVER-6	ROUTE 4 — the ladder runs to the wall. METAVIR F0→F4 read as a coherence ladder: collagen rises ~2% → ~38% (F3) → ~65% (F4) of liver mass while register coherence falls toward the floor; ALT, AST, GGT (normally < 40 U/L, GGT most sensitive) are the register-disruption markers. Short of cirrhosis the drift is displaced, not deleted; F4 is the irreversible wall where drift has become deletion and dΣT=0 redistributes to what survives. CORRECTION 4: reach it before F4; let the liver's standing regeneration finish.
P-LIVER-7	ORDER LAW: quiet the source (corrections 1 + 3) before releasing the scaffold (correction 2) — settling collagen back to 640 Å while the cell still lays new collagen simply re-drifts it; and all corrections must land before the F4 wall (correction 4 is the clock).
P-LIVER-8	Off-lattice signatures are fingerprints, not destinations: the primes in the drifted readings (67 in 670; any prime past 864) mark a register that has left the {2,3,5} lattice; they are never set-points the tissue climbs toward. This corrects the earlier "prime boundary" reading. The correction releases the tissue back to its clean node, never toward a prime.
P-LIVER-9	Reversible short of cirrhosis: a drifted register is displaced, not deleted, and the liver regenerates; the corrective family is native to the liver's own retinoid biology. F4 (cirrhosis) is the irreversible wall, where drift has become deletion.
P-LIVER-10	The 864 = $2^5 \times 3^3$ node is a cross-body master pivot: the solar day (86,400 s), the hepatic collagen stability ceiling, the joint-cartilage period (arthritis framework) and the Peyronie's plaque — one node, several tissues; a correction that releases a drifted 864 register in one is, in principle, learning to release it in all.

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 length in Ångströms, a span of time, a mass, a percentage — 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 length in a fibre here, a span of time in the heavens there, a mass in a nucleus somewhere else. The same number that spaces the collagen in your liver can read, in another register, as the length of a day. 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.

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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

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