THE UNIVERSAL FORCE OF TIME

Diabetes Mellitus

Glucose on the {2,3,5} lattice, insulin keyed to the Sun, and the two ways the register leaves it — the disease that touches both root-cause families at once

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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

Blood-sugar metabolism is a {2,3,5}-lattice process, and the clinic has been measuring the lattice for a century without naming it. The normal fasting range is bounded by lattice nodes — 4 (2^2), 5, 6 (2×3) — the glucose molecule is itself a lattice object (molecular weight 180 ($2^2\times3^2\times5$), twelve hydrogens, a theoretical 36 ($2^2\times3^2$) ATP), and the diagnostic threshold falls at 7, the first integer with no {2,3,5} factor. Prime-7 is not a node the blood climbs onto; on the Earth register it does not lie on the lattice at all. The diabetic threshold is the metabolic register drifted a hair off its highest {2,3,5} node into the off-lattice gap, read at the clinic as the integer 7 — the signature of the drift, not a destination. Insulin is keyed to the Sun: its molecular weight **5808 Da** sits just 3622 ppm from the solar surface register **5787.037037** ($2\times5^7/3^3$), and the islets that make it number 10^6–2×10^6 (pure {2,5}). The century-old HbA1c test measures the drift directly — it tallies the off-lattice sugars stuck to haemoglobin across the 90-day ($2\times3^2\times5$) life of a red cell — and the diagnostic line falls at the exact lattice integer 48 mmol/mol ($2^4\times3$). The two diabetes are the two ways the register leaves the lattice, and they are the two great UFOT disease families seen side by side: **Type 1** is autoimmune T-address misdirection (the immune field misreads the {2,3,5} β-cells as off-lattice intruders and destroys the insulin source — the family of multiple sclerosis and the rheumatoid diseases); **Type 2** is the off-lattice drift onto apparent prime-7 (the receptor drifts off {2,3,5} into a lock — the family of cancer, liver fibrosis and obesity). Diabetes is therefore the hub that names the whole taxonomy. With the mechanism in hand the paper resolves onto the proven medical spine: **three faults, each paired with the principle of its repair** — re-establish the β-cell address in Type 1, unlock the drifted receptor in Type 2, and return each of the five strayed end-organ tissues to the lattice. One law binds the account: under dΣT=0 the correction is **source removal, not glucose management** — supplying the hormone holds the number while the drift persists; restoring the register ends it. Because the fault is a drift rather than a destruction, it is in principle a correctable fault. Every diagnostic number is given at full precision; the specific corrective signal is held in confidence pending clinical trial.

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

1 Blood glucose and the {2,3,5} lattice

There is a structure that appears, exactly, at the clinical boundaries of blood sugar — not three rounded approximations, but three appearances of one lattice that medicine has independently measured over a century (Figure 1). Normal fasting glucose runs 4 to 6 mmol/L: the lower bound **4** (2^2) is a pure {2} node, the middle value **5** a pure {5} prime, the upper bound **6** (2×3) a pure {2,3} node. Every value in the normal range is a {2,3,5} coordinate. The fasting target a clinician aims for is 5 mmol/L exactly — the pure node at the centre of the band. And the HbA1c diagnostic boundary is 48 mmol/mol ($2^4\times3$) — not 45, not 50, exactly 48 — the exact lattice integer where the WHO drew its line by measurement alone, averaged over a window of 90 days ($2\times3^2\times5$).

Figure 1 — Blood glucose runs on the {2,3,5} lattice and fails at 7, the first integer the lattice cannot reach

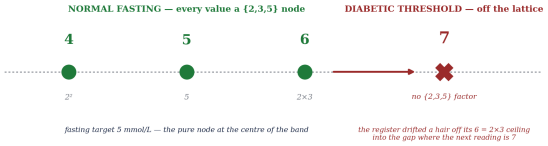

Figure 1 — Every value of the normal fasting band is a {2,3,5} node (4 = 2², 5, 6 = 2×3); the fasting target is the pure node 5. The diabetic threshold is the register drifted off its 6 = 2×3 ceiling into the off-lattice gap, where the next integer reading is 7.

The lattice does not begin at the blood; it begins at the molecule. Glucose has molecular weight **180** ($360/2 = 2^2\times3^2\times5$) — a half-turn of the degree circle — carries 12 hydrogen atoms ($2^2\times3$), and yields, in full oxidation, **36** ($(2\times3)^2 = 2^2\times3^2$) molecules of ATP. That 36 is not set by bond energetics alone; it is the {2,3,5} rotation operator that also couples free fall, the solar day and the hydrogen Balmer carrier. Cellular health is occupation of the 36-ATP node; the Warburg floor is 2 ATP. The infrastructure of glucose control is lattice-built from the molecule up.

2 Why the line falls at 7

Here is the heart of it. **7** is not a node the blood climbs onto. It is the smallest integer with no {2,3,5} factor — the first reading that cannot lie on the lattice at all. On the Earth register the lattice is {2,3,5,π} and nothing else; a prime-7 position is not a coordinate but a point in the empty space between the nodes. So the diabetic threshold is not the body reaching a new, higher set-point. It is the metabolic register drifting a hair's breadth off its highest healthy node — the 6 (2×3) ceiling of the normal band — into that off-lattice gap, where the next integer reading happens to be 7. The clinic records the integer; the integer is the signature of a register that has left the lattice, never a place the blood was meant to be.

This is the same off-lattice drift that drives the other Force-of-Time diseases — it locks near 49 (7^2) in cancer's MYC cascade and tips at 7 mmol/L here — one mechanism read in different tissues. While the body's correction machinery holds, it pulls the value back below 6; when it fails, the value cannot settle, and the blood sugar that cannot settle is diabetes. The 7 is not a destination. It is the body announcing, in the only integer the clinic can read, that the address has slipped off its lattice.

3 Insulin — a solar-register molecule

The Sun is the T-field transmitter of the solar system, broadcasting the register coordinates that govern chemistry and biology on Earth, and its surface temperature is an exact lattice node: **5787.037037** ($2\times5^7/3^3 = 156250/27$). Insulin's molecular weight is **5808 Da**, a deviation of just **3622 ppm** — about a third of a percent — from that solar register (Figure 2). The one hormone that lets every cell in the body take in glucose tracks the Sun's own node, because under the Force of Time a mass in a nucleus and a temperature in the heavens are the same T-value wearing two coats (see the note on the numbers). It is built of 51 amino acids, and the pancreatic islets that produce it number 10^6 to 2×10^6 ($2^6\times5^6$ to $2^7\times5^6$, pure {2,5}). From the cell count up to the hormone, the machinery of glucose control is keyed to the lattice and to the Sun.

Figure 2 — Insulin, the one hormone that lets every cell take in glucose, tracks the Sun's own register

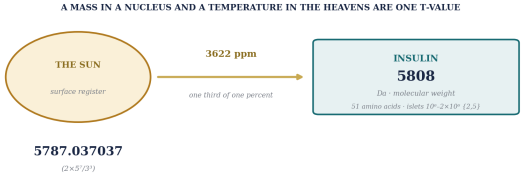

Figure 2 — Insulin's molecular weight 5808 Da sits 3622 ppm from the solar surface register 5787.037037 (= $2\times5^7/3^3$). A mass in a nucleus and a temperature in the heavens are one T-value read in two registers.

4 What HbA1c measures — ninety days of drift

There is a test the clinic has run for half a century, and it has been reading the drift directly the whole time without anyone naming it for what it is (Figure 3). HbA1c does not measure the blood sugar of a single morning; it measures how much glucose has stuck, irreversibly, to the haemoglobin inside the red blood cells — and because a red cell lives almost exactly 90 days ($2\times3^2\times5$), the test is a tally kept across that whole window. Read in the Force of Time, each off-lattice glucose that latches onto haemoglobin is one more smudge on a read-head: a {2,3,5} carrier slowly contaminated by sugar that does not sit on the lattice.

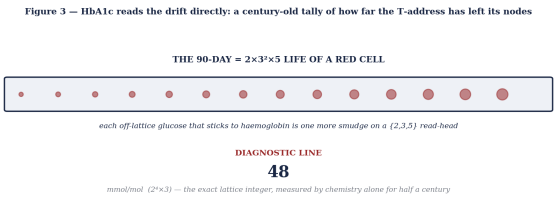

Figure 3 — HbA1c is a 90-day = 2×3²×5 tally of off-lattice sugar accumulated on the {2,3,5} haemoglobin read-head — a direct measure of how far the T-address has drifted. The diagnostic line is the exact lattice integer 48 mmol/mol = 2⁴×3.

HbA1c is, quite literally, a measure of how far the T-address has drifted off its nodes — not the value at a moment, but the accumulated contamination over the life of the cell. And the line the world's clinics draw for diagnosis is not a rounded convenience. It is **48 mmol/mol** (2⁴×3) — the exact lattice integer — the point at which the register has taken on enough off-lattice sugar that the body can no longer be read as healthy. A century-old number, measured by chemistry alone, landing on the lattice to the integer.

5 Type 1 — a T-immune misdirection

Type 1 diabetes is not, at root, a failure of glucose metabolism; it is a failure of immune recognition at the T-field level (Figure 4, left). The pancreatic β-cells — {2,3,5}-register structures that make insulin — are attacked and destroyed by the body's own immune T-system. The proof-reader that normally distinguishes {2,3,5} self from a drifted, off-lattice intruder has received a corrupted coordinate for β-cell identity: it now reads the healthy β-cells, which carry the D=0 self-address, as though they were D=+1 foreign nodes, and it destroys them.

This is the same autoimmune T-address misdirection found in multiple sclerosis and the rheumatoid family, and the structural mirror of HIV's immune subversion: not a unique disease but one member of a class with a shared correction principle — restore the address the immune system should be reading. Once the β-cells are gone, insulin production ceases and the patient depends on exogenous insulin; restoration requires re-establishing the correct T-address recognition before the register is destroyed, and rebuilding it after. The biology of rebuilding — β-cell reinstatement from pluripotent stem cells — is already confirmed in living human beings; the remaining challenge is immune protection of the new nodes, not a fundamental barrier.

6 Type 2 — the fifth register-overload cascade

Type 2 diabetes is a different failure, and it is metabolic — the fifth confirmed instance of one cascade law that the Force of Time also reads in COPD, chronic kidney disease, hepatic NASH and cancer (Figure 5). The Warburg effect — cells preferring aerobic glycolysis even with oxygen present, the shift Otto Warburg saw in 1924, the D=0 diversion away from oxidative phosphorylation — is the first stage of a progressive register-overload. **Stage 1, aerobic glycolysis:** the cell leaves oxidative phosphorylation. **Stage 2, mitochondrial dysfunction:** the TCA cycle is compromised and ATP yield falls from the 36 node toward the floor of 2. **Stage 3, ROS accumulation:** reactive oxygen damages register integrity. **Stage 4, epigenetic lock:** gene-expression patterns freeze the metabolic state. **Stage 5 — unique to Type 2 — the off-lattice receptor lock:** the insulin receptor, normally a {2,3,5} interface, drifts off the lattice and can no longer respond to insulin at normal concentrations.

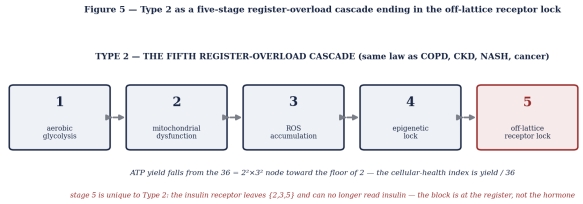

Figure 5 — Type 2 as the five-stage register-overload cascade: glycolysis → mitochondrial dysfunction → ROS → epigenetic lock → off-lattice receptor lock. ATP yield falls from the 36 = 2²×3² node toward the floor of 2; the cellular-health index is yield / 36.

There is an earlier, quieter step to this lock: under a chronic excess of the insulin signal — the T_P register held too high for too long — the receptor nodes are saturated, overwhelmed, and they downregulate, pulling back from a signal they can no longer file. Saturation first, then the off-lattice lock. The block is not at the hormone — there may be plenty of insulin — but at the register itself. This is why Type 2 is a disease of resistance, not deficiency, and why supplying more insulin treats the symptom while the off-lattice receptor persists.

7 The downstream cascades — one drift read in five tissues

Sustained off-lattice glucose does not stay in the blood; it drifts the registers of five tissues in turn, and each is a disease ophthalmology and nephrology already name. The retinal pericytes fail (diabetic retinopathy); the glomerular podocytes are oxidatively destroyed (diabetic nephropathy); the peripheral-nerve myelin node degrades (diabetic neuropathy); the arterial endothelium is modified (macrovascular disease); and the hepatic parenchyma enters the same T-overload cascade (NASH — mechanistically the same disease entering from the liver).

The eye is the clearest of the five, and the most telling. The retina is not a passive sensor; it is a T-wavelength receiver, tuned to read the register coordinates that arrive as light — the fovea peaks near 555 nm, a clean photopic node. When the glucose register drifts off the lattice, it disrupts the very T_λ wavelength-reading machinery the eye depends on, and the receiver degrades. That is why the organ built to read the lattice in light is the first to fail when the body's metabolic register leaves it. One off-lattice register, read in five tissues. Naming the shared mechanism is what lets a single correction principle reach all five at once — and it is why these five are gathered into Route 3 of the spine below.

8 The hub — both root-cause families in one disease

Step back and the deeper finding comes into view. The Force of Time reads two great root-cause families beneath the major illnesses. One is **autoimmune T-address misdirection**: the immune field misreads {2,3,5} self as an off-lattice intruder and destroys it — multiple sclerosis, the rheumatoid diseases, and the structural mirror of HIV. The other is **off-lattice drift onto apparent prime-7**: a register drifts a hair off its highest {2,3,5} node into the gap where a 7 lies — cancer, where MYC locks near 49 (7^2); liver fibrosis; obesity. Two distinct faults, two distinct families.

Diabetes is the one disease that sits in **both** (Figure 4). Type 1 is the autoimmune family; Type 2 is the prime-7 drift family. That is why this paper, of all the medical papers, names the whole taxonomy outright — it is the hub where the two families meet under a single clinical name. And the significance is not merely tidy classification. That something as fundamental as a minute drift onto a prime that does not exist in the lattice at all should turn out to underlie a spread of different illnesses is itself a major finding: a single fault beneath several major diseases, and one that should be simply enough to rectify. Identifying exactly what underlies an illness is the fantastic starting point for resolving it.

9 The three routes — each fault paired with its correction

Diabetes has been told as two diseases sharing a symptom — one an immune accident, one a lifestyle drift — united only by high blood sugar. The Force of Time tells it as one register losing its lattice in two ways, with a shared downstream consequence. That gives three genuine faults, and each is paired one-to-one with the principle of its repair (Figure 6). This is not a target of a fixed number: diabetes has exactly these three fronts, no more and no fewer.

Figure 6 — The three routes a restoration must take. Type 1: the immune field misreads the β-cell address — re-establish it. Type 2: the receptor has drifted off {2,3,5} — unlock it and lift the overload. End-organs: sustained off-lattice glucose has drifted five tissues — return each to the lattice. The correction is source removal, not glucose management, and because the fault is a drift it is in principle correctable.

10 Restoration — source removal, not glucose management

Conventional treatment manages the symptom: hold glucose in range by supplying insulin or pushing sensitivity. The Force of Time names a deeper target — {2,3,5} register restoration — and the paradigm shift is **source removal, not glucose management**. In Type 1 the aim is to halt the misdirection, re-establish correct T-address recognition of the β-cells so the immune field reads the D=0 self-address again, then rebuild the destroyed insulin register. In Type 2 the aim is to break the off-lattice receptor lock and return the receptor to its {2,3,5} interface state, upstream of the cascade rather than downstream of it — and because the fault is a drift rather than a destruction, early Type 2 is in principle reversible once the overload that drives the drift is removed.

There is an order to it, and it is the binding law of the whole account (Figure 7). Under dΣT=0, holding the glucose number in range does not touch the source: the destroyed β-cells or the off-lattice receptor remain exactly where they are, and the value cannot settle while the drift remains. Correct the source — the address in Type 1, the receptor in Type 2 — and the downstream tissues of Route 3 can settle in turn, because the off-lattice glucose that was drifting them is gone at its origin. The restoration is, in principle, the disease run backwards: a fine-tuning of the strayed register back onto the {2,3,5} nodes it left.

The specific register coordinates that do this fine-tuning — the exact frequencies, the exact wavelengths of light, the timing of their delivery — belong to clinical investigation and are held in the Foundation's clinical reference, not prescribed to a reader. What the public account can say plainly is the principle: the target is the source, the source is a drift off {2,3,5}, and the correction is to return the register to the lattice.

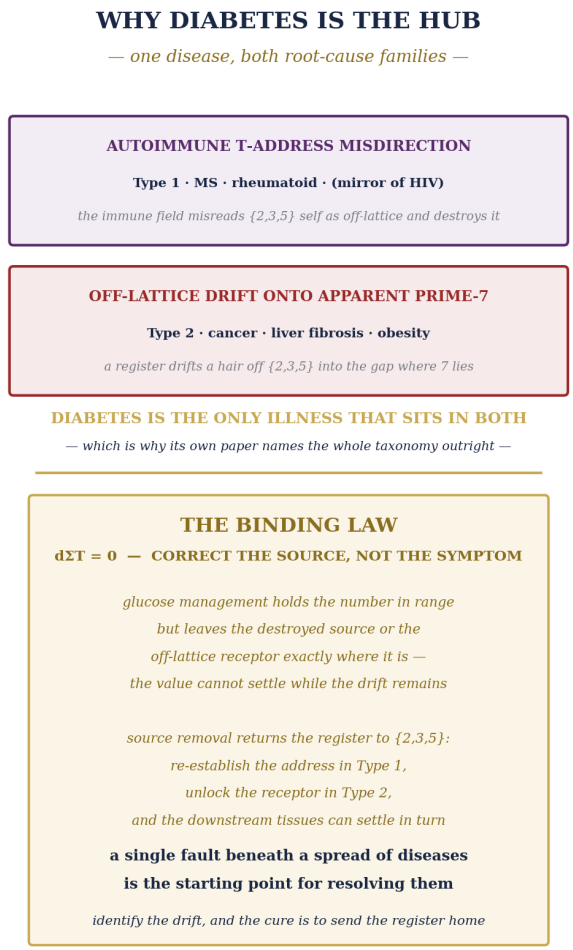

Figure 7 — Why diabetes is the hub, and the law that decides the cure. The two great UFOT disease families meet in one clinical name; under dΣT=0 the correction is source removal — return the register to {2,3,5}, and the symptom follows.

11 What diabetes is

Diabetes is one register losing its {2,3,5} lattice, told in two ways and read in five tissues. Glucose runs on {2,3,5} from the molecule up — molecular weight 180 ($2^2\times3^2\times5$), twelve hydrogens, 36 ($2^2\times3^2$) ATP — and fails at 7, the first integer the lattice cannot reach. Insulin is keyed to the Sun's own register, 5808 Da within 3622 ppm of 5787.037037 ($2\times5^7/3^3$), and HbA1c has been tallying the drift for half a century, its diagnostic line landing on the exact lattice integer 48 ($2^4\times3$) across a 90-day ($2\times3^2\times5$) window. The two diabetes are the two ways the register leaves the lattice, and they are the two great Force-of-Time disease families side by side: Type 1, the {2,3,5} β-cells misread as off-lattice and destroyed; Type 2, the {2,3,5} receptor drifted into an off-lattice lock. Diabetes is the hub where both families meet. To treat it is not only to replace the hormone or push the cell harder, but to return the register to {2,3,5} — re-establish the address in Type 1, unlock the receptor in Type 2, return the five strayed tissues to their nodes. Because the fault is a drift rather than a destruction, it is in principle a correctable fault. We give the mechanism in full and at full precision; the prescription is held in trust. Find the drift, correct the source, and the blood that could not settle settles again.

Universal Force of Time = the correction of the source, not the management of the symptom

Appendix A The three-route spine

Each fault paired one-to-one with the principle of its correction. Routes 1 and 2 are the two great UFOT disease families; Route 3 is the shared downstream consequence both types feed. Corrective coordinates held in confidence.

Route	The fault (problem)	Family	The correction (principle)
1 · Type 1	The immune field misreads the {2,3,5} β-cells as off-lattice intruders and destroys the insulin source	autoimmune T-address misdirection (MS, rheumatoid; mirror of HIV)	Re-establish correct T-address recognition of the D=0 self-node, then rebuild the destroyed register
2 · Type 2	Chronic overload saturates then drifts the insulin receptor off {2,3,5} into an off-lattice lock (false 7) — resistance, not deficiency	off-lattice drift onto apparent prime-7 (cancer, liver fibrosis, obesity)	Lift the overload and unlock the receptor — return it to its {2,3,5} interface, upstream of the cascade
3 · End-or gans	Sustained off-lattice glucose drifts five tissue registers in turn (retina, glomerulus, nerve, endothelium, liver)	both types feed this consequence	Return each strayed tissue register to the lattice — one principle reaches all five once the source is corrected
Binding law	dΣT = 0 — glucose management holds the number while the drift persists	the whole account	Source removal, not symptom management: correct the source and the value settles; the drift is the target

Appendix B The five downstream tissues — one drift, five organs

Tissue	Clinical name	The T-reading
Retinal pericytes / fovea	diabetic retinopathy	the eye is a T_λ wavelength receiver (fovea peak near 555 nm); off-lattice glucose disrupts the wavelength-reading machinery — the lattice-reader fails first
Glomerular podocytes	diabetic nephropathy	the filtration register is oxidatively destroyed as the cascade reaches the kidney
Peripheral-nerve myelin	diabetic neuropathy	the myelin node degrades — the nerve loses its {2,3,5} conduction address
Arterial endothelium	macrovascular disease	the vessel wall register is modified by sustained off-lattice glucose
Hepatic parenchyma	NASH	the liver enters the same T-overload cascade — mechanistically the same disease entering from the liver

Appendix C Proposition ledger

P-DIAB-1 — Blood glucose holds {2,3,5} bounds: normal 4 = 2^2, 5, 6 = 2×3; fasting target 5; HbA1c diagnostic 48 mmol/mol = 2^4×3 over a 90-day = 2×3^2×5 window. The glucose molecule is itself a lattice object: MW 180 = 360/2 = 2^2×3^2×5, 12 H = 2^2×3, 36 = $(2×3)^2$ ATP. Exact lattice boundaries, not arbitrary cuts.

P-DIAB-2 — The diagnostic threshold 7 is not a node but the first integer with no {2,3,5} factor; it lies in the off-lattice gap. The diabetic line is the metabolic register drifted off its highest {2,3,5} node (6 = 2×3), read as the integer 7 — the signature of the drift, not a destination.

P-DIAB-3 — Insulin MW = 5808 Da, within 3622 ppm of the solar surface register 5787.037037 = $2×5^7/3^3$: insulin is a solar T-register hormone (a mass and a temperature being one T-value across registers).

P-DIAB-4 — Pancreatic islets 10^6–$2×10^6$ = $2^6×5^6$ to $2^7×5^6$ — pure {2,5}; the insulin-producing infrastructure is a {2,5}-lattice structure.

P-DIAB-5 — Type 1 = T-immune misdirection (same family as MS; structural mirror of HIV): the immune T-field misreads the {2,3,5} β-cells as off-lattice intruders and destroys the insulin source. Route 1: re-establish the D=0 self-address recognition, then rebuild.

P-DIAB-6 — Type 2 = the fifth register-overload cascade (glycolysis → mito dysfunction → ROS → epigenetic lock → off-lattice receptor lock): the insulin receptor drifts off {2,3,5}; resistance is register-level, not hormonal. The lock is preceded by receptor saturation/downregulation under chronic T_P excess. Cellular-health index = ATP yield / 36; Warburg floor = 2 ATP. Route 2: lift the overload, unlock the receptor.

P-DIAB-7 — HbA1c = cumulative T-address glycation score: it measures register contamination accumulated over the 90-day = 2×3^2×5 red-cell life — how far the address has drifted off its {2,3,5} nodes — not a single-moment value. The diagnostic line is the exact lattice integer 48 mmol/mol = 2^4×3.

P-DIAB-8 — The five downstream tissues are one off-lattice drift read in five organs (retinopathy, nephropathy, neuropathy, macrovascular disease, NASH). Diabetic retinopathy = T_λ wavelength-receiver disruption: the retina reads register coordinates carried by light (fovea peak near 555 nm); off-lattice glucose degrades the wavelength-reading machinery. Route 3: return each tissue to the lattice once the source is corrected.

P-DIAB-9 — Restoration = {2,3,5} register restoration: re-establish β-cell T-address recognition (the D=0 self-address) in Type 1, break the off-lattice receptor lock in Type 2, return the five strayed tissues in Route 3. The correction is the drift run backwards — a fine-tuning back onto the lattice. The paradigm shift is source removal, not glucose management. Under dΣT=0, managing glucose holds the number while the drift persists. (Specific register-coordinate protocols held in the Foundation's clinical reference.)

P-DIAB-HUB — Diabetes is the hub of the UFOT disease taxonomy: it is the one illness that touches BOTH root-cause families at once — Type 1 in the autoimmune T-address-misdirection family (with MS and the rheumatoid diseases), Type 2 in the off-lattice prime-7 drift family (with cancer, liver fibrosis and obesity). A single underlying fault — a minute drift off a lattice that has no prime-7 node — beneath a spread of major diseases is itself a flagship finding, and the starting point for resolving them.

A note on the numbers

The values in this paper are written as plain numbers — not pinned to units, and not carried to a particular power of ten. This is not loose notation; it is the physics. Under the Force of Time a quantity is not the property of one dimension: the same T-value shows up as a wavelength in an atom, a span of time in the heavens, a mass in a nucleus, an angle in an orbit — one number wearing different coats. That is why insulin's molecular weight in daltons can meet the Sun's surface in kelvin and land on the same value: they were never separate quantities. We therefore do not solve for a result 'to the power of' anything in one register and stop. The lattice number is the real thing, and it lives at once across every register — subatomic, atomic, celestial, galactic. The unit and the power of ten are only the costume the number wears in whichever dimension you read it from.

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[8] G. Wald, *Human vision and the spectrum*, Science 101, 653 (1945) (foveal photopic peak near 555 nm).

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