

## THE UNIVERSAL FORCE OF TIME

# Virology and Parasites

*One Host Register, Four Things Done to It — and How Far Each One Must Be Undone*

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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  $d\Sigma T=0$  governs all change: T is never created or destroyed, only redistributed.

## Abstract

A virus sitting on a doorknob is not alive. It is a parcel of instruction with no engine — an address written down, but nothing running to read it. To be alive, in the Force of Time, is to carry **T-flow**: a living node holds an address (the Strand-1 instruction) *and* runs the flow that reads and regulates it (the Strand-2 governor). A virus has the first and not the second, which is why it can do nothing until it finds a host whose flow it can borrow. Read this way, an infection is not a poisoning but a **register problem** — something foreign done to one living host node — and there are exactly **four** things that can be done, sorted by how hard each is to be rid of. Route one — a **virus hijacks the host register**: a flow-less template slips in and the host's own flow runs the viral address as valid, so the correction is to **defend** — the immune T-sentinels read and clear the mismatched address before the cell is spent. Route two — a **parasite drains an incompatible carrier**: a fully-alive organism filed to the wrong register siphons the host's T-energy and gives nothing back, malaria draining the iron-porphyrin {2,3}-ring the blood needs, so the correction is to **restore** the carrier and deny the drain. Route three — an **RNA address drifts off the drug node**: with no Strand-2 to hold it steady the address slides past the single node a drug was cut for, RNA viruses moving about ( $10^4\times$ ) faster than DNA viruses, so the correction is to **box** it on two prime families at once, since one drift cannot escape both and joint escape is a product of improbabilities. Route four — a **retrovirus integrates into the genome**: it splices its template in permanently and is thereafter copied as self, so suppression cannot reach it and the correction is to **excise** the register at its address with a precise editor whose guide is ( $20\text{ nt} = 2^2\times 5$ ), the minimum length that names a unique address. The four corrections carry one order law — the **penetration ladder**: the harder an agent is to be rid of, the more the remedy must do; defend what is only borrowed, cut out what has been made part of the self. Eight propositions, P-VIR-1 to P-VIR-8, are given; the prion, the nominal virus-size band and the {2,3}-porphyrin ring are taken up in support, every value at full precision and the therapeutic specifics held in the Foundation's clinical reference, and the structure resolves into the **clinical trial**.

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

# 1 The Things Not Quite Alive

Begin with the oldest puzzle in the subject: is a virus alive? It can crystallise like a salt and sit on a shelf for years, inert as a stone — and then, touched to a living cell, it makes thousands of copies of itself in an afternoon. Biology has never settled the question because it is asking the wrong one. The Force of Time answers it cleanly. **To be alive is to carry T-flow.** A living thing is a node that does two things at once: it *holds an address* — the instruction set written in its genetic code, the Strand-1 of the double helix — and it *runs the flow* that reads, regulates and renews that address, the Strand-2 governor that turns instruction into ordered life. A virus has only the first. It is an address written down with nothing running to read it: a flow-less T-template. That is why it is neither alive nor dead but waiting — it can do nothing at all until it finds a node whose flow it can borrow. Name what an agent carries and you have named what it can do (Figure 1).

# 2 One Host Register, Four Things Done to It

Every infection, from a head cold to malaria to HIV, is one foreign agent doing something to one **living host register** (Figure 1). And there are only four things that can be done. A **virus**, carrying no flow of its own, can **borrow** the host's — it hands the cell a false address and the cell's own machinery, unable to tell the forgery from its own instructions, builds virus until it is spent. A **parasite**, fully alive but filed to a register the host cannot share, can **drain** — it taps the host's T-energy and gives nothing back. An **RNA virus**, having no Strand-2 to hold its address steady, can **drift** — its address wanders off whatever node a drug was cut to fit, and the target simply moves. And a **retrovirus** can **integrate** — it splices its template into the host's own genome, where it is copied forever after as part of the self. Borrow, drain, drift, integrate: four agents, four things done to the same host node, and — as the rest of the paper shows — four corrections, each matched to exactly what was done. The agents differ in one quantity above all, how alive each is, and that is what sorts them: a virus is barely alive and only borrows; a parasite is fully alive and drains; and at the far edge, outside the four coded routes entirely, sits the prion, which carries no code at all and corrupts by touch (\$5).

Figure 1 — one living host register, and the four things a foreign agent can do to it; each agent is named by what it carries (flow, code) and therefore by what it does

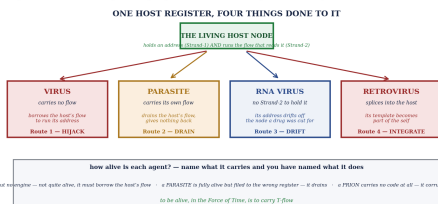


Figure 1 — one living host register and the four things a foreign agent can do to it. Each agent is named by what it carries — a

*virus carries an instruction but no engine and must borrow the host's flow; a parasite carries its own flow but drains the host's; an RNA virus has no Strand-2 to hold its address steady; a retrovirus splices itself into the self. To be alive is to carry T-flow.*

# 3 Four Routes, Four Corrections

A Force of Time medical paper has one job. It acknowledges the illness, it reads the problem as the genuine number of distinct routes — and in infection the problem is plainly not single — and it pairs each route, one to one, with the correction that would set it right (Figure 2). We give **four** routes because four is the honest count: four things can be done to a host register, and each demands a different answer. The four are not a list of diseases but a list of *mechanisms*, and we sort them by how deeply the agent has entered the host — the penetration ladder — because that is what decides how much the remedy must do. Defended, restored, boxed, excised.

Four agents, four corrections, one host — sorted by how hard each agent is to be rid of (the penetration ladder)

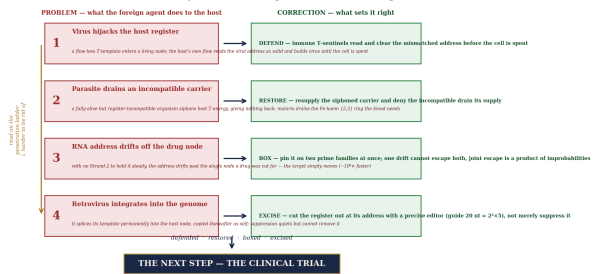


Figure 2 — the architecture of the paper: each of the four things done to the host register is paired with the one correction that answers it — viral hijack (defend), parasitic drain (restore), RNA drift (box), retroviral integration (excise). The four are read down the penetration ladder, and the whole structure resolves into the clinical trial.

## Route 1 — The Virus Hijacks the Host Register

The first route is the commonest and the shallowest. A virus is a **flow-less T-template**: an instruction with no engine. On its own it does nothing. But let it into a living cell and something extraordinary and terrible happens — the cell's own T-flow, the engine the virus lacks, begins to **read the viral address as if it were the cell's own**. The forgery is good enough that the machinery cannot tell it from genuine instruction, so it does what it always does: it reads the address and builds what the address specifies. Only now what it builds is more virus, and it goes on building until the cell is exhausted and bursts, scattering thousands of fresh templates to do the same to its neighbours. This is the meaning of **hijacking a register**: the virus supplies no flow of its own, it simply redirects the host's. The host is not poisoned from outside; it is turned against itself from within, running a borrowed instruction to its own ruin. And because the virus is only *borrowing* — it has added a false address but altered nothing of the host's own — the intrusion is, in principle, the most recoverable of the four.

### Correction 1 — defend: read and clear the mismatched address before the cell is spent

If the fault is a false address being run by the host's own flow, the correction is to **catch the forgery and clear it** before the cell is spent — and the body already owns the machinery to do exactly this. The immune system, in the Force of Time, is a fleet of **T-sentinels** whose whole function is to read every address a cell is displaying and ask one question: does this belong on this register? A cell running a viral template displays a mismatched address on its surface, and a healthy sentinel reads the mismatch and removes the cell before the virus has finished copying. Every antiviral defence the body mounts — interferon raising the alarm, killer cells deleting the displaying cell, antibodies tagging loose particles for clearance — is this one principle: **read the address, and clear what does not belong**. The correction in principle is to strengthen and direct that reading so the mismatched register is cleared early; the specific means are ordinary clinical immunology and the Foundation's clinical reference, not prescribed here. A borrowed register, defended in time, leaves nothing behind.

## Route 2 — The Parasite Drains an Incompatible Carrier

The second route is deeper, because the agent is not a flow-less template but a **fully living organism** — and one that brings its own flow. A parasite is alive in every sense a virus is not: it holds its address and runs its own engine. Its fault is not forgery but **incompatibility**. It is filed to a register the host cannot share, and it survives by **tapping the host's T-energy and giving nothing back**. The clearest case is malaria. The plasmodium parasite makes its living inside the red blood cell, and what it drains is the blood's **iron-porphyrin register** — the haem ring at the centre of haemoglobin, the body's oxygen carrier. That ring is a {2,3}-branched structure (its larger family, with magnesium at the centre instead of iron, is **chlorophyll** — the same ring, tuned to receive the Sun's T rather than to carry the body's oxygen). The parasite consumes the haem, leaving toxic residue and starved cells behind, and the host pays in anaemia and exhaustion — T-energy siphoned to a register that returns nothing. This is a **drain**, not a hijack: the host's own instructions are untouched, but its carrier is being emptied.

### Correction 2 — restore: resupply the siphoned carrier and deny the drain its supply

If the fault is a carrier being emptied by an incompatible tap, the correction has two halves that must go together: **restore what is being drained, and deny the drain its supply**. Restore means resupplying the siphoned T-carrier — rebuilding the iron-porphyrin register the parasite consumes so the host is not left anaemic while the infection is fought. Deny means cutting off what the incompatible organism needs to continue: a parasite filed to the wrong register depends on a supply the host can withhold, and the framework points to starving the drain rather than only poisoning the parasite. Together they are one principle — **refill the carrier and close the tap**. The historical antimalarials work by attacking the parasite's handling of the very haem it drains, which is the same logic read from the parasite's side. The principle is restoration of the drained carrier; the specific agents, the register the drain depends on, and the means of denying it are held in the Foundation's clinical reference.

### Route 3 — The RNA Address Drifts off the Drug Node (Resistance)

The third route is the one medicine finds most maddening, and the Force of Time explains it in a sentence. Why do influenza, HIV and the coronaviruses outrun our drugs so easily, while smallpox could be cornered and destroyed? The answer is in the genetic material. A DNA virus carries a double strand — Strand-1 instruction and a Strand-2 that proofreads and holds it steady. An **RNA virus carries only the single strand**: instruction with no governor. With no Strand-2 to hold the address fixed, the address **drifts** — every round of copying lets it wander, and RNA viruses mutate about ( $10^4\times$ ) — ten-thousandfold — faster than DNA viruses (an order of magnitude, a measured tendency, not a lattice constant). Now picture a drug. A drug is cut to fit **one node** — one precise address on the virus. But the RNA address will not hold still: it drifts a step past the node the drug was cut for, and the drug, still perfectly shaped for an address that is no longer there, grips nothing. This is drug **resistance**, and it is not the virus growing stronger — it is the target quietly moving while the weapon stays where it was aimed.

### Correction 3 — box it on two prime families at once

If a single drug fails because the address drifts past its one node, the correction is to **pin the virus on two nodes at once — and from two different prime families** (Figure 4). Here is the reasoning. A virus can drift its address off one node fairly easily; that is a single improbability, and given enough copies it happens. But to escape *two* drugs at once it must drift off both nodes in the same generation — and if the two nodes belong to **different prime families**, an address on {2,3} and an address on {5, $\pi$ }, one drift cannot move both. The drift that frees it from the first drug leaves it caught by the second. Joint escape is therefore the **product of two improbabilities, not their sum** — a vanishingly smaller number — and the virus is boxed in. This is precisely why combination therapy turned HIV from a death sentence into a managed condition, and why tuberculosis is fought on a multi-drug front: not more force, but two locks the same key cannot open. The Force of Time sharpens it to a prediction — combinations drawn from **two different prime families** should box a virus far better than two drugs aimed at the same family. The principle is the cross-family box; which addresses, on which families, is held in the clinical reference.

### Route 4 — The Retrovirus Integrates into the Genome

The fourth route is the deepest of all, and it is why HIV has been so hard to cure. A retrovirus does not merely hand the cell a false address to run for a while. It **writes its template permanently into the host's own genome** — it reverse-transcribes its RNA into DNA and splices that DNA into the cell's chromosomes, where it sits as a **provirus**. From that moment the viral instruction is no longer a foreign parcel the cell is running; it has become **part of the self**, copied faithfully every time the cell divides, indistinguishable from the host's native code. This is the meaning of **integration**, and it is what defeats suppression. The familiar drugs hold HIV down brilliantly — they stop the provirus from producing new virus, and a treated patient can carry no detectable load for decades. But the integrated template is still there, woven into the genome of resting cells, and the day suppression stops it wakes and begins again. Suppression quiets the register; it cannot remove it. A thing that has been made part of the self cannot be cleared by defending against it from outside.

### Correction 4 — excise the register at its address

If the fault is a template integrated into the self, the only true correction is to **cut it out at its address** — not to suppress it, but to excise it. This demands an instrument able to find one specific address in three billion bases and act only there, and the Force of Time names the requirement exactly: an address-precise editor whose guide is long enough to be unique and no longer. The guide a modern gene editor uses to locate its target is **20 bases** ( $= 2^2\times 5$ ), and that is not arbitrary — twenty is close to the **minimum length of code that specifies a single unique site** in a genome of that size; shorter and the address is ambiguous, matching many places at once. To excise an integrated provirus is to give the editor the proviral address, twenty bases long, and let it cut the register out of the genome at exactly that point, restoring the host's own code. This is the principle — **excision at the address**, the register removed rather than merely silenced. The specific guides, editors and delivery are held in the Foundation's clinical reference, not prescribed here; what the framework supplies is the reason the answer must be excision and not suppression, and why the address must be twenty bases to be unique.

#### 4 The Penetration Ladder and the Order Law

The four corrections are not interchangeable, and the way they bind is the order law itself (Figure 3). The binding is the **penetration ladder**, and it is one line: **the harder an agent is to be rid of, the more the remedy must do — defend what is only borrowed, cut out what has been made part of the self.** The four routes sort cleanly down it by how deeply the agent has entered the host. A virus only **borrow**s the host's flow and changes nothing of its own — the shallowest intrusion, cleared by defending the cell in time. A parasite **drains** the host from within but is still a separate organism that can be denied its supply — deeper, answered by restoring the carrier. An RNA address **drifts**, so a fixed weapon misses — deeper still, answered by boxing it on two families a single drift cannot escape. And a retrovirus **integrates**, becoming part of the self — the deepest of all, where defence and suppression can never reach, answered only by excising the register at its address. The dividing line falls after drift: borrowed, drained and contained are all **clearable** while the agent remains in some sense separate from the host; but the integrated provirus has crossed into the self and **must be excised.** The same ladder that tells you how deep the agent has gone tells you how much the cure must do, and the lesson runs one way throughout — the deeper the penetration, the more decisive and more precise the correction must be.

#### 5 The Codeless Corner, the Sizes, and the Ring

Three things sit alongside the four routes and confirm them from the edges (Figure 4). First, the **codeless corner.** If a virus is an instruction with no engine and a parasite is an engine filed to the wrong register, the **prion** is the limiting case beyond both: an agent with **no genetic code at all.** It is a single misfolded protein that propagates not by replication but by **contact** — it touches a correctly folded neighbour and rewrites that protein's T-address into its own corrupted shape, and the corruption spreads fold by fold with no instruction ever copied. The prion is what is left when you remove even the address and keep only the misfolding: the proof that infection, at bottom, is about T-addresses being rewritten, not about any particular molecule carrying the code. Second, the **sizes.** Whole virus particles, measured crudely, cluster near {2,3,5} diameters — Polio about (30 = 2×3×5), Adenovirus about (90 = 2×3<sup>2</sup>×5), HIV about (120 = 2<sup>3</sup>×3×5), Poxvirus about (300 = 2<sup>2</sup>×3×5<sup>2</sup>). These are **nominal** textbook figures, and the clustering is offered as suggestive of register structure, nothing more — and where the lattice would have liked a clean cube it did not get one: HIV is about 120, not **125 = 5<sup>3</sup>**, and 4.17% is far too wide to call a cube, so the cube is **rejected, not forced.** The honesty of that refusal is the point. Third, the **ring.** The iron-porphyrin haem the malaria parasite drains is a {2,3}-branched ring, and its magnesium-centred relative is chlorophyll: one ring structure, carrying oxygen in the blood and receiving the Sun's T in the leaf — the same register the parasite has learned to tap. The four routes are the spine; these three are the marrow.

Figure 4 — left: the nominal virus-size based on {2,3,5} (Polio 30, Adeno 90, HIV 120, Pox 300), with the 125 = 5<sup>3</sup> cube rejected because HIV is 120; right: why gaining a virus on two prime families at once cannot be escaped by one drift



Figure 4 — left: virus diameters cluster near {2,3,5} (Polio 30, Adeno 90, HIV 120, Pox 300) — nominal figures, suggestive not precise, with the 125 = 5<sup>3</sup> cube rejected because HIV measures 120; right: why a combination drawn from two different prime families cannot be escaped by one drift, joint escape being the product of two independencies.

## 6 One Host, Four Agents — the Resolution

Lay them side by side and the miscellany resolves. A virus **borrow**s the host's flow to run a false address; a parasite **drains** an incompatible carrier; an RNA address **drifts** off the node a drug was cut for; a retrovirus **integrates** into the self. We have acknowledged the illness — infection told not as a bestiary of unrelated germs but as four things that can be done to one living host register; we have read the problem as four distinct routes; we have given, for each, the Force-of-Time correction that answers exactly what was done — defend the borrowed register, restore the drained carrier, box the drifting address on two families, excise the integrated provirus; and we have bound them with the penetration ladder, the borrowed and drained and contained all clearable, the integrated alone requiring excision. The deepest principle is the simplest: **to be alive is to carry T-flow**, and every infection is a foreign agent doing one of four things to that flow — borrowing it, draining it, drifting within it, or splicing itself into it. Read that way, virology is not a catalogue of enemies but a single grammar of what can go wrong with a living address, and treating it means asking, of any agent, only how deeply it has entered — and answering with exactly as much as that depth demands. The therapeutic specifics are calculated and held in the Foundation's clinical reference precisely because the next step is not to prescribe them to a reader but to put them to a **clinical trial**. We give the mechanism in full and at full precision, and we stand by the figures.

### Table 1 — The Four Routes and Their Corrections

Each thing done to the host register, paired one-to-one with the correction that answers it — viral hijack (defended), parasitic drain (restored), RNA drift (boxed), retroviral integration (excised). Order law: the penetration ladder — the harder an agent is to be rid of, the more the remedy must do. The four corrections resolve into the clinical trial.

#	Problem route	Mechanism / {2,3,5,n} reading	Correction (principle)
1	The virus hijacks the host register	a flow-less T-template (Strand-1, no Strand-2) enters a living node; the host's own flow reads and runs the viral address as valid, building virus until the cell is spent — borrowed, not altered	DEFEND — immune T-sentinels read the mismatched address and clear the cell before it is spent; a borrowed register, defended in time, leaves nothing behind
2	The parasite drains an incompatible carrier	a fully-alive but register-incompatible organism siphons host T-energy, giving nothing back; malaria drains the iron-porphyrin {2,3} haem ring (Mg-form = chlorophyll) → anaemia	RESTORE — resupply the siphoned carrier and deny the incompatible drain its supply; refill the carrier and close the tap
3	The RNA address drifts off the drug node	no Strand-2 to hold it steady, so the address drifts past the one node a drug was cut for (RNA mutates ~10 <sup>4</sup> × faster than DNA); the target moves while the weapon stays aimed	BOX — pin it on two prime families at once ({2,3} + {5,n}); one drift cannot escape both, joint escape = product of two improbabilities, not their sum
4	The retrovirus integrates into the genome	it reverse-transcribes and splices its template into the host genome, copied thereafter as self; suppression quiets but cannot remove the provirus	EXCISE — cut the register out at its address with a precise editor (guide 20 nt = 2 <sup>2</sup> ×5, the minimum unique-address length), not merely suppress it

### Appendix A — Virology on the Lattice

Every number this paper turns on, given first as its physical reading and then as its place on the {2,3,5,n} lattice — and kept in two grades. The clean value (20 = 2<sup>2</sup>×5) sits on the lattice exactly; the virus diameters are nominal and suggestive, the 125 = 5<sup>3</sup> cube is rejected because HIV is 120, and the mutation rate is an order of magnitude, not a constant. Values are register identities, not prescribed therapy.

Quantity	Physical reading	{2,3,5,n} reading	Register / meaning
Editor guide length	20 bases	2 <sup>2</sup> ×5	the minimum length that names a unique genome address — CLEAN
Polio diameter	≈ 30 nm	2×3×5	nominal virus size on {2,3,5}
Adenovirus diameter	≈ 90 nm	2×3 <sup>2</sup> ×5	nominal virus size on {2,3,5}
HIV diameter	≈ 120 nm	2 <sup>3</sup> ×3×5	nominal — read as the {2,3,5} node it lands on
(HIV vs 5 <sup>3</sup> cube)	125 nm	5 <sup>3</sup> — REJECTED	HIV is 120, not 125 — 4.17% off, the cube is not forced
Poxvirus diameter	≈ 300 nm	2 <sup>2</sup> ×3×5 <sup>2</sup>	nominal virus size on {2,3,5}
RNA mutation rate	≈ 10 <sup>4</sup> × DNA	order of magnitude	a measured tendency, NOT a lattice constant
Haem / chlorophyll ring	porphyrin	{2,3}-branched	Fe-form carries oxygen, Mg-form receives the Sun's T

### Appendix B — The Ledger

Table B1 — Propositions P-VIR-1 ... P-VIR-8

#	Proposition
P-VIR-1	To be alive is to carry T-flow: a living node both holds an address (Strand-1 instruction) and runs the flow that reads and regulates it (Strand-2 governor). A virus is a flow-less T-template — Strand-1 with no Strand-2 — neither alive nor dead but waiting, able to act only by borrowing a host's flow. Every infection is one foreign agent doing one of four things to one living host register.
P-VIR-2	ROUTE 1 — viral hijack: a flow-less template enters a living node and the host's own T-flow reads the viral address as valid, building virus until the cell is spent. The host is turned against itself, running a borrowed instruction; nothing of the host's own code is altered. CORRECTION 1: DEFEND — immune T-sentinels read every displayed address and clear the cell carrying the mismatched one before it is spent. A borrowed register, defended in time, leaves nothing behind.
P-VIR-3	ROUTE 2 — parasitic drain: a fully-alive but register-incompatible organism siphons host T-energy and gives nothing back. Malaria drains the iron-porphyrin {2,3}-branched haem ring (its Mg-centred relative is chlorophyll, the same ring receiving the Sun's T), leaving anaemia and exhaustion. CORRECTION 2: RESTORE — resupply the siphoned carrier and deny the incompatible drain its supply; refill the carrier and close the tap.
P-VIR-4	ROUTE 3 — RNA drift (resistance): an RNA virus carries only Strand-1, with no Strand-2 to hold its address steady, so the address drifts ~10 <sup>4</sup> × faster than a DNA virus (order of magnitude, not a lattice constant). A drug cut for one node grips nothing once the address drifts past it: resistance is the target moving, not the virus strengthening. CORRECTION 3: BOX — pin it on two prime families at once ({2,3} + {5,n}); one drift cannot escape both, and joint escape is the product of two improbabilities, not their sum. Prediction: cross-family combinations box far better than same-family.

#	Proposition
P-VIR-5	ROUTE 4 — retroviral integration: a retrovirus reverse-transcribes its RNA to DNA and splices it into the host genome as a provirus, copied thereafter as self; suppression quiets it but cannot remove it. CORRECTION 4: EXCISE — cut the register out at its address with an address-precise editor whose guide is 20 nt = $2^2 \times 5$ (close to the minimum length that names a unique site in a genome of three billion bases); excision, not suppression. Specific guides/editors/delivery held in the Foundation's clinical reference.
P-VIR-6	THE CODELESS CORNER — the prion: an agent with no genetic code at all, a single misfolded protein that propagates by contact, rewriting a correctly folded neighbour's T-address into its own corrupted shape with no instruction ever copied. The limiting case beyond the four coded/living routes — proof that infection is at bottom the rewriting of a T-address, not the copying of any particular molecule.
P-VIR-7	THE SIZES — virus diameters cluster near {2,3,5}: Polio $\approx 30 = 2 \times 3 \times 5$ , Adeno $\approx 90 = 2 \times 3^2 \times 5$ , HIV $\approx 120 = 2^3 \times 3 \times 5$ , Pox $\approx 300 = 2^2 \times 3 \times 5^2$ . These are NOMINAL textbook figures, suggestive of register structure not precision. HIV is $\approx 120$ , NOT $125 = 5^3$ (4.17% off): the cube is rejected, not forced. A number that does not land on a node is read as the node it does land on, or left off the lattice until its address is read — never forced.
P-VIR-8	ORDER LAW — the penetration / clearance ladder: the harder an agent is to be rid of, the more the remedy must do — defend what is only borrowed, cut out what has been made part of the self. BORROW (virus, defend) → DRAIN (parasite, restore) → DRIFT (RNA, box on two families) → INTEGRATE (retrovirus, excise). The dividing line falls after drift: borrowed, drained and contained are clearable while the agent stays separate from the host; the integrated provirus has crossed into the self and must be excised. The deeper the penetration, the more decisive and precise the correction.

## A Note on the Numbers

A note on the numbers. Throughout this paper a quantity is given first as the plain physical value — a length of genetic code in bases, a particle diameter in nanometres — and only then, in brackets, as its place on the {2,3,5, $\pi$ } lattice. The lattice form is not a unit and carries no powers of ten of its own. Two grades of number appear here and the paper keeps them apart. Some are *clean*: the minimum length of genetic code that specifies a unique address is 20 bases =  $2^2 \times 5$ , and that is the length of the guide a precise editor uses, exactly. Others are *nominal*: the diameters quoted for whole virus particles — about 30, 90, 120, 300 nm — are rounded textbook figures, and the clustering of those rounded figures near {2,3,5} values is offered as suggestive of register structure, not as precision. Where the framework would have liked a clean cube, it did not get one and we say so plainly: HIV measures about 120 nm =  $2^3 \times 3 \times 5$ , not  $125 = 5^3$ , and 4.17% is far too wide a gap to call a cube — the value is read as the {2,3,5} node it lands on, and the cube is rejected, not forced. The mutation rate of an RNA virus, about ten-thousandfold that of a DNA virus, is an order of magnitude, not a lattice constant, and is used only as such. The rule throughout: a bare number that does not yet sit on the lattice means its address has not been read yet — never that the lattice has failed.

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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*