Viruses as Tau-Parasites

Viral Hijacking of Host Tau-Address Machinery · Prions as Tau-Misfolding · CRISPR as Natural Tau-Address Editor · Antibiotic Resistance as Tau-Lattice Adaptation

Stephen Daubney

The Daubney Foundation

P-VIR-1 The FOT Classification of Pathogens

The Force of Time framework classifies pathogens by their relationship to the host's Tau-address machinery:

Pathogen type	FOT classification	Tau-address relationship
Virus	Tau-parasite	No independent Tau-address machinery; hijacks host Strand 1 without contributing Strand 2 regulation
Bacterium	Proto-Tau-organism	Possesses own Tau-address machinery (circular DNA = closed Tau-loop); competes with host register
Prion	Tau-misfolding agent	Protein in wrong Strand configuration; catalyses mis-registration of native proteins
Fungus	Tau-register squatter	Establishes competing G1 register within host tissue; does not hijack but displaces
Parasite (macro)	Tau-address thief	Uses host Tau-medium (water, nutrients) to maintain own Tau-address at host's expense

P-VIR-2 Viral Replication as Tau-Hijacking

A virus consists of a Tau-address (its genome — DNA or RNA) encased in a protein shell (the capsid — a Tau-address delivery vehicle). The virus has no Strand 2 regulatory machinery of its own. Upon entering a host cell, it injects its Tau-address into the host Strand 1 machinery, which executes the viral replication programme because it cannot distinguish the viral Tau-address from a legitimate host instruction set.

P-VIR-2

A virus is a Tau-parasite: a minimal Tau-address (genome) with no Strand 2 regulation, packaged for delivery into a host cell whose Strand 1 machinery it hijacks. The viral genome is not 'alive' in isolation — it has a Tau-address but no active Tau-register. It becomes active only inside a host G1 register.

This explains why viruses exist at the boundary of the living/non-living distinction: in FOT terms, a virus has a Tau-address but no Tau-register. Life requires both. A virus parasitises the host's register to execute its address — temporarily borrowing the G1 register to become a functional Tau-node.

P-VIR-3 RNA Viruses and Tau-Address Instability

RNA viruses (influenza, SARS-CoV-2, HIV) have much higher mutation rates than DNA viruses. In FOT, this is Tau-address instability: RNA is single-stranded (Strand 1 only), lacking the Strand 2 stabilisation provided by the DNA double helix. Without Strand 2, the RNA Tau-address is not held in precise {2,3,5,pi} lattice registration and drifts — producing mutations.

```
DNA virus mutation rate: ~10^-8 per base per replication RNA virus mutation rate: ~10^-4 per base per replication Ratio: 10^4 = (10^2)^2 — the Strand 2 stabilisation factor 10^4 = 2^4 x 5^4 (pure {2,5} lattice)
```

The factor 10^4 = 2^4 x 5^4 separating DNA and RNA mutation rates is a {2,5} lattice value. FOT predicts this exact ratio (not 10^3 or 10^5) as the Strand 2 stabilisation constant for the nucleotide Tau-address.

P-VIR-4 Prions as Tau-Misfolding Agents

Prions (misfolded proteins that catalyse the misfolding of native proteins) cause diseases including CJD, scrapie, and chronic wasting disease. In FOT, a prion is a protein locked in the wrong Strand configuration: instead of the native Strand 2 (folded, regulated) conformation, it is stuck in a Strand 1 (unfolded, unregulated) conformation that is thermodynamically stable but biochemically inert.

The prion's catalytic effect — it induces normally-folded proteins to adopt its misfolded configuration — is Tau-configuration propagation: the wrong Strand configuration spreads through the tissue Tau-field, progressively converting Strand 2 proteins to Strand 1 configuration. FOT prediction: prion propagation rate should follow a Tau-wave diffusion equation with diffusion constant set by the {2,3,5,pi} lattice of the affected protein's Tau-address.

P-VIR-5 CRISPR as Natural Tau-Address Editor

CRISPR-Cas9 — the bacterial immune system that cuts foreign DNA at specific sequences — is in FOT a natural Tau-address editor. Bacteria store fragments of viral Tau-addresses in their CRISPR arrays (the 'memory' of past infections), then use guide RNAs (Tau-address probes) to locate and cut matching sequences in incoming viral DNA.

The precision of CRISPR targeting — 20 nucleotide guide sequences — encodes a {2,3,5} value: 20 = 2^2 x 5. The guide length of 20 nt is not arbitrary but is the minimum Tau-address segment required for unique identification in a G1-register genome. FOT prediction: the optimal guide length for single-target specificity in any genome is 2^2 x 5 = 20 nucleotides, regardless of genome size.

P-VIR-6 Antibiotic Resistance as Tau-Lattice Adaptation

Antibiotic resistance evolves when bacteria alter the Tau-address targeted by the antibiotic. Antibiotics work by binding to specific bacterial Tau-addresses (ribosomal RNA, cell wall synthesis enzymes, DNA gyrase) and disrupting their function. Resistance mutations shift the Tau-address slightly — enough to prevent antibiotic binding while

retaining sufficient lattice precision for function.

FOT prediction on antibiotic resistance: resistance mutations are constrained to Tau-address shifts within the {2,3,5,pi} nearest-neighbour lattice. Non-nearest-neighbour shifts are not viable because they disrupt the target protein's own function. This predicts: (1) resistance mutations will cluster at specific lattice positions, (2) multi-drug resistance requires sequential nearest-neighbour steps, (3) antibiotics targeting the Tau-address core (not periphery) will have the fewest viable resistance routes.