Fever and Immune Response as Tau-Field Retuning

Body Temperature 37°C = 307 FOT-K · Fever Threshold 40°C = 310 FOT-K · Immune Response as Strand 2 Restoration · Inflammation as Register Boundary Reset

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P-FEVR-1 Body Temperature as a Tau-Lattice Value

Normal human body temperature is 37.0°C. In the FOT temperature scale, where absolute zero = -270°C (rather than -273.15°C), temperature in FOT-Kelvin is T_FOT = T_Celsius + 270. Body temperature in FOT-K is:

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T_body (FOT) = 37.0 + 270 = 307.0 FOT-K 307 is a prime number in the
 {2,3,5,pi} lattice context T_fever max (FOT) = 40.0 + 270 = 310.0
      FOT-K 310 = 2 x 5 x 31 ({2,5} component with prime 31)
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The fever threshold of 40°C = 310 FOT-K = 2 x 5 x 31 is a {2,5} lattice value in its leading factors. The {2,5} sub-lattice governs the Hayflick limit (50 = 2 x 5^2), the Schumann excitation rate (50/s), and now the fever threshold. FOT predicts that the boundary between physiological and pathological temperature is set by the {2,5} prime-lattice intersection, not by arbitrary biochemical kinetics.

P-FEVR-2 Fever as Tau-Field Thermal Retuning

When pathogens disrupt Tau-addresses in the immune register, the organism's response is to raise the ambient thermal energy of the G1 register — increasing Tau-density uniformly across all tissues. This elevated Tau-density has two effects: it disrupts the lower-Tau-density pathogens (whose Tau-addresses are optimised for lower-register temperatures) and it accelerates the immune system's Tau-restoration machinery.

P-FEVR-2

Fever is not a side effect of immune activation. It is the primary Tau-retuning mechanism: raising the G1 register temperature toward 310 FOT-K = 2 x 5 x 31 disrupts pathogen Tau-addresses (optimised for lower register) while maintaining host Tau-address integrity (already calibrated to 310 FOT-K). The fever is the treatment, not the symptom.

This reframes the clinical debate on fever suppression. FOT predicts that suppressing fever below 40°C (310 FOT-K) before pathogen clearance removes the primary Tau-retuning mechanism and prolongs infection. The conventional recommendation to suppress fever above 40°C is consistent with FOT: above 310 FOT-K the retuning overshoots the {2,5} threshold and begins disrupting host Tau-addresses.

P-FEVR-3 The Immune System as Tau-Restoration Army

The immune system in FOT is the organism's Tau-address restoration service. White blood cells are mobile Tau-validators: they patrol the G1 register, detect Tau-address mismatches (non-self antigens = foreign Tau-addresses), and eliminate or quarantine the mismatch. The immunological 'self/non-self' distinction is a Tau-address recognition problem.

Autoimmune disease in FOT is Tau-address mis-recognition: the immune system incorrectly flags native Tau-addresses as foreign, mounting a restoration response against the organism's own register. The autoimmune trigger is a Tau-address collision — where a native address is structurally close enough to a pathogen address that molecular mimicry occurs. FOT predicts autoimmune triggers should always have Tau-address proximity to the affected tissue's native addresses.

P-FEVR-4 Inflammation as Register Boundary Reset

Inflammation — the cardinal signs of heat, redness, swelling, pain, and loss of function — represents a local G1 register boundary reset. When Tau-addresses in a tissue are disrupted, the organism isolates the disrupted zone (swelling = Tau-boundary establishment), increases local Tau-density (heat, redness = register elevation), and suspends normal register function (pain, loss of function = standby mode) while restoration proceeds.

Chronic inflammation occurs when the reset cannot complete — the Tau-address disruption is too widespread or too deeply corrupted for the restoration process to converge. FOT predicts that chronic inflammatory conditions are characterised by register boundary instability: the Tau-field cannot establish a stable G1 boundary at the inflammation site, producing oscillating rather than resolving inflammation.

P-FEVR-5 Vaccination as Tau-Address Preloading

Vaccination exposes the immune system to a pathogen Tau-address (attenuated or fragmented) without the pathogen's full Strand-1 replication programme. The immune system learns to recognise the Tau-address pattern and prepares Tau-restoration responses. Vaccination is Tau-address preloading: the immune register is pre-calibrated to the pathogen's Tau-address.

mRNA vaccines in FOT: the mRNA encodes Strand 1 instructions for producing a partial Tau-address fragment (the spike protein). The cell's Strand 2 regulation remains intact, so the fragment is produced at controlled levels, processed, and used to pre-load the immune Tau-register. The mRNA is destroyed after use — it does not alter the host Tau-address (DNA) because Strand 2 prevents reverse transcription in normal G1-register cells.

P-FEVR-6 Testable Predictions

Prediction	Measurement	FOT claim
Fever suppression below 40°C prolongs infection duration	RCT: fever suppression vs permissive fever in bacterial infection	Suppression prolongs by factor proportional to Tau-density deficit
Autoimmune trigger antigens have Tau-address proximity to affected tissue native antigens	Structural comparison of autoimmune triggers vs tissue antigens	Proximity in {2,3,5,pi} lattice, not merely sequence similarity
Chronic inflammation oscillates at Tau-field boundary frequency	Time-series analysis of inflammatory markers	Oscillation period = Tau-boundary reset time for affected register
40 Hz gamma entrainment boosts immune response via Tau-lock	Monitor immune markers during 40 Hz gamma exposure	Predicts quantifiable NK cell and T-cell activity increase

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