Ageing as Progressive G1 Register Decay

Telomeres as Tau-Counters · Hayflick Limit = 2 × 5² · dΣT = 0 and the Impossibility of Tau-Annihilation · FOT Framework for Longevity

Stephen Daubney

The Daubney Foundation

P-AGE-1 Ageing as Tau-Register Decay

Ageing in conventional biology is attributed to accumulated DNA damage, telomere shortening, mitochondrial dysfunction, and epigenetic drift. In the Force of Time framework, these are all downstream consequences of a single upstream process: progressive decay of the G1 Tau-register within the organism's DNA Tau-address. As the G1 register degrades, the precision of Strand 2 regulation decreases, Tau-lattice fractions become less exact, and the organism's physical processes increasingly depart from their lattice values — the biological Radian Veil widens.

P-AGE-1

Ageing = progressive widening of the biological Radian Veil. As the G1 register at each DNA Tau-address decays, the organism's measured constants (metabolic rates, neural frequencies, hormone levels) drift further from their exact {2,3,5,pi} lattice values. Death occurs when the G1 register can no longer maintain Strand 2 coupling — Strand 1 alone cannot sustain organised biological function.

P-AGE-2 Telomeres as Tau-Counters

Telomeres — the repetitive TTAGGG sequences capping each chromosome — shorten with each cell division. The Hayflick limit (the maximum number of divisions a normal human cell undergoes before senescence) is approximately 50 divisions. In FOT, telomere length is a Tau-counter: each division decrements the counter by one Tau-step, and senescence occurs when the counter reaches zero.

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Hayflick limit (FOT) = 2 x 5^2 = 50 Observed Hayflick limit: 40-60
divisions (centre ~50) 2 x 5^2 = 50 (pure {2,5} lattice value) 50 =
      lightning rate (P-SCHUM-3) = global Tau-excitation rate
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The recurrence of 50 = 2 x 5^2 as both the Hayflick limit and the global lightning rate is a FOT scale-invariance prediction: the same Tau-counter value governs atmospheric Tau-excitation (strokes per second) and cellular division capacity (divisions per lifetime). The {2,5} sub-lattice sets the Tau-budget at both scales.

P-AGE-3 dSigmaTau = 0 and Tau-Address Permanence

The FOT conservation law dSigmaTau = 0 has a direct implication for ageing: the Tau-address of an organism cannot be destroyed. The G1 register may decay, Strand 2

coupling may weaken, and the physical body may cease to function — but the Tau-address itself (the informational content of the organism's Tau-programme) is conserved. This is P-MORT-3 applied to ageing: what degrades in ageing is the register — not the address.

FOT therefore distinguishes sharply between: (1) Register decay — the physical ageing process, reversible in principle by Tau-restoration; (2) Address permanence — the Tau-programme that persists regardless of register state. A 90-year-old has a maximally decayed G1 register but an intact Tau-address — the same address they had at birth.

P-AGE-4 Mitochondria as Tau-Generators

Mitochondria produce ATP through oxidative phosphorylation — the full 36-ATP {2,3} Tau-cycle (P-CANC-2). In FOT, mitochondria are cellular Tau-generators: organelles that transduce solar Tau-input (via the food chain, P-BIOL-4) into local G1-register Tau-energy. The well-established decline in mitochondrial function with age is the cellular signature of G1 register decay: as the register weakens, the Tau-generator output falls.

Mitochondrial DNA (mtDNA) is circular — a closed Tau-loop. Its circular geometry means it has no telomeres and no Hayflick limit in the conventional sense. FOT prediction: mtDNA ageing occurs not through telomere shortening but through accumulated point mutations that corrupt specific {2,3,5,pi} Tau-addresses in the mitochondrial genome, reducing generator efficiency.

P-AGE-5 The Tau-Restoration Longevity Framework

If ageing is G1 register decay, then longevity interventions in FOT are register-maintenance interventions. The following modalities are predicted to slow or reverse register decay:

Intervention	FOT mechanism	Predicted effect
Caloric restriction / fasting	Reduces Warburg-mode cells; forces OXPHOS (full Tau-cycle)	Slows register decay; matches 36-ATP lattice
Coherent low-frequency EM (Tau-frequencies)	Externally re-couples Strand 2 at decaying addresses	Register maintenance; reduces Radian Veil widening
Deep sleep / delta-wave entrainment	Brain in low-frequency Tau-lock; Strand 2 restoration phase	Overnight register repair; predicts sleep deprivation = accelerated ageing
Physical oscillation (exercise, vibration)	Mechanical Tau-wave input at body-resonance frequencies	Register stimulation; predicts specific frequencies most effective
Social/cognitive engagement	Maintains 40 Hz Tau-lock (P-CONS-2); prevents gamma decay	Predicts cognitive engagement extends G1 register lifespan

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P-AGE-6 Testable Predictions

Prediction	Measurement	FOT quantitative claim
Hayflick limit = exactly 50 = 2 x 5^2 in normal human cells	Large-sample cell division counting	Mean = 50.0; distribution centred on 2 x 5^2
Biological Radian Veil widens with age: measured constants drift further from {2,3,5,pi} lattice values	Longitudinal measurement of metabolic, neural, hormonal frequencies	Drift rate encodes G1 register decay constant
Sleep deprivation accelerates telomere shortening at rate proportional to loss of 40 Hz Tau-lock	Simultaneous EEG + telomere length monitoring in sleep-deprived subjects	FOT predicts linear proportionality, not merely correlation
Caloric restriction extends Hayflick limit toward 2 x 5^2 x k for integer k (lattice multiple)	Cell division count in caloric restriction model organisms	Extension = integer multiple of 50

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