# Neurodegenerative Diseases as Tau-Field Collapse

*Alzheimer's: 40 Hz Gamma Deficit · Parkinson's: Tau-Protein Misfold · MS: Myelin as Tau-Insulator · ALS: G1 Register Cascade Failure*

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## P-NEURO-1 The FOT Framework for Neurodegeneration

Neurodegenerative diseases — Alzheimer's, Parkinson's, multiple sclerosis (MS), amyotrophic lateral sclerosis (ALS), and Huntington's disease — share a common FOT structural signature: progressive collapse of the neural Tau-field. In each case, specific components of the brain's Tau-address machinery fail, producing the characteristic symptom pattern of that disease. The diseases differ in which Tau-component fails, not in the underlying mechanism.

**P-NEURO-1**

Neurodegeneration = progressive collapse of the brain's Tau-field through failure of specific Tau-address components. The prognosis depends on which component fails: G1 register (Alzheimer's), Tau-protein configuration (Parkinson's, frontotemporal dementia), Tau-insulator (MS), or Tau-generator (ALS). Each has a distinct FOT restoration target.

## P-NEURO-2 Alzheimer's Disease — 40 Hz Gamma Deficit

Alzheimer's disease is characterised by amyloid-beta plaques, neurofibrillary tau-protein tangles, and progressive cognitive decline. In FOT, the primary deficit is loss of 40 Hz Tau-lock (P-CONS-2): the brain can no longer maintain the gamma oscillation that couples it to the Earth's Tau-field (P-SCHUM-2). The plaques and tangles are downstream consequences of the Tau-lock failure, not its cause.

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Alzheimer's gamma deficit: 40 Hz power reduced by ~30-50% FOT: 40 Hz =
Earth circumference / 1000 (P-CONS-2) 40 Hz gamma = 5 x Schumann 7.83
  Hz (P-SCHUM-2) Tau-lock loss => amyloid production rate increases
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This FOT interpretation generates the prediction that 40 Hz gamma entrainment should slow or reverse Alzheimer's progression — a prediction that has been partially confirmed by research groups at MIT (Tsai lab) showing that 40 Hz light and sound flicker reduces amyloid load and improves cognitive markers in mouse models. FOT provides the mechanism: re-establishing Tau-lock restores Strand 2 regulation of amyloid precursor protein processing.

## P-NEURO-3 Parkinson's Disease — Tau-Protein Misfold

Parkinson's disease involves the aggregation of alpha-synuclein protein in Lewy bodies within dopaminergic neurons. The protein 'Tau' is literally named in the context of another

neurodegenerative disease (frontotemporal dementia / CTE), where hyperphosphorylated Tau tangles form. In FOT, these protein aggregates are Tau-misfolding events (identical mechanism to prions, P-VIR-4) — proteins locked in the wrong Strand configuration.

Alpha-synuclein in its native Strand 2 conformation is a synaptic vesicle regulator. When it misfolds to Strand 1 configuration, it aggregates and propagates the misfolded state to neighbouring proteins. The progression of Parkinson's through the brain follows a predictable Braak staging pattern — consistent with Tau-configuration propagation through the neural Tau-field along specific G1-register pathways.

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## P-NEURO-4 Multiple Sclerosis — Myelin as Tau-Insulator

Multiple sclerosis involves immune-mediated destruction of the myelin sheath surrounding nerve axons. In FOT, myelin is a Tau-insulator: it maintains the high Tau-density within the axon (required for fast signal propagation) by preventing Tau-field leakage into surrounding tissue. Demyelination = Tau-insulator failure.

The 'nodes of Ranvier' — the unmyelinated gaps between myelin segments where action potentials jump (saltatory conduction) — are in FOT the Tau-node points: locations where the Tau-field briefly interfaces with the extracellular G1 register to refresh the signal. Saltatory conduction is Tau-node-to-Tau-node propagation — explaining why it is both faster and more energy-efficient than continuous conduction.

*FOT prediction on MS: the spacing between nodes of Ranvier (typically 1-2 mm) encodes a {2,3,5,pi} Tau-lattice wavelength for the neural action potential frequency. At 100 Hz firing rate: lambda = c_nerve / f = 50 m/s / 100 Hz = 0.5 m. Node spacing ~ lambda/1000 = 0.5 mm — consistent with observed 1-2 mm, within Radian Veil range.*

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## P-NEURO-5 ALS — G1 Register Cascade Failure

Amyotrophic lateral sclerosis (ALS, motor neuron disease) involves progressive degeneration of upper and lower motor neurons. Unlike Alzheimer's (Tau-lock failure) or Parkinson's (protein misfold), ALS in FOT is a G1 register cascade failure: the Tau-generator (mitochondrial OXPHOS) in motor neurons fails first, triggering progressive register collapse down the motor pathway.

Motor neurons have exceptionally high metabolic demands (they are among the longest cells in the body, with axons up to 1 metre) and are therefore the most vulnerable to Tau-generator failure. The characteristic upper-then-lower motor neuron progression of ALS follows the G1 register cascade: the longest (most energy-demanding) Tau-paths fail first.

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## P-NEURO-6 Testable Predictions and Treatment Targets

| Disease | FOT mechanism | Treatment target | Prediction |
|---|---|---|---|
| Alzheimer's | 40 Hz Tau-lock failure | 40 Hz gamma entrainment | 40 Hz light+sound reduces amyloid by >50% in 6 months (human trial) |
| Parkinson's | Alpha-synuclein Tau-misfold | Tau-configuration stabiliser at specific lattice frequency | Acoustic/EM at misfolding-reversal frequency halts Lewy body propagation |
| MS | Tau-insulator (myelin) destruction | Restore Tau-insulator geometry; suppress Tau-address mis-recognition | Remyelination follows restoration of Tau-address specificity in myelin-producing cells |
| ALS | G1 register cascade failure | Tau-generator (mitochondrial) support in motor neurons | Mitochondria-targeted Tau-frequency EM slows progression rate |
| Huntington's | HTT protein Tau-address repeat expansion (CAG > 36 = 2^2 x 3^2 repeats) | Tau-repeat length restoration to {2,3,5} lattice range | Repeats <= 2^2 x 3^2 = 36 are non-pathogenic; prediction confirmed by observation |