FORCE OF TIME

Treatment Protocols — Internal Reference
Full Protocol Detail — All Conditions

Stephen Daubney · The Daubney Foundation · 2026

This document preserves the complete FOT treatment protocol details for all medical conditions addressed by the Force of Time framework. These are the specific modalities, frequencies, dosages, compounds, durations, and sequencing that were developed as part of the FOT medical series. This document is an internal reference for The Daubney Foundation. It is distinct from the published medical paper series, which describes the structural theoretical framework without specific clinical protocol prescriptions pending the outcome of medical trials.

Contents

Alzheimer's Disease

Class III

FOT Classification: Class III T-Address Calibration Divergence — progressive directional drift away from the 40 Hz consciousness ground state. Amyloid-beta and NFTs are downstream markers of T-address drift, not primary causes.

Full Four-Register Protocol (P-ALZ-4)

Component	Frequency / Compound	Duration	Target Register	Mechanism
1 — Temporal (40 Hz entrainment)	40 Hz LED flickering + 40 Hz acoustic tone delivered simultaneously	1 hour daily	T_λ + T_M (temporal/consciousness)	Re-entrains neural T-address to C_Earth/1000. Dua
2 — Mitochondrial (669.9709632 nm PBM)	669.9709632 nm (near-infrared, cytochrome c oxidase peak)	20 min daily (whole-body panel)	Mitochondrial G1 register	Activates cytochrome c oxidase (Complex IV). Rest
3 — Dimensional (486 nm H-beta)	486 nm = 2×3⁵ nm (Balmer H-beta, narrow-band LED)	10 min daily (surface exposed)	Dimensional lattice (cellular water TEQ)	Fundamental UFOT dimensional wavelength. Re-sy
4 — Molecular (Retinoid oral)	Oral retinol at maintenance dose (RAR/RXR pathway)	Daily oral supplementation	Molecular programme (Strand 2 re-inscription)	Activates RAR/RXR nuclear receptor superfamily. I

Panel coherence: 40 × 486 = 19,440 = $2^4 \times 3^5 \times 5$ [EXACT pure {2,3,5}]. The temporal and dimensional components are not independent — their product is a pure lattice value, confirming coherent lattice pairing and predicting super-additive combined effect.

Parkinson's Disease

Class II

FOT Classification: Class II Frozen T-Address — dopaminergic Tau-programme present but silenced at the substantia nigra T-node. NURR1/RXR = the re-inscription unit.

Five-Component Protocol

Modality	Compound / Frequency	Route	Notes
40 Hz acoustic stimulation	40 Hz = C_Earth/1000	Headphones or full-room audio	Re-synchronises basal ganglia gamma oscillation network
670 nm photobiomodulation	670 nm red light	Surface panel / transcranial	Targets cytochrome c oxidase — mitochondrial G1-registe
486 nm H-beta photobiomodulation	486 nm = 2×3⁵ nm	Surface LED exposure	Re-synchronises cellular water TEQ. Adenine resonance.
NURR1/RXR retinoid activation (oral/systemic)	IRX4204 (selective RXR agonist) or BRF110 (NURR1 activator) or 9-cis retinoic acid	Oral — non-invasive (characterised in pre-clinical and early clinical work)	IRX4204: restores TH, AADC, DAT gene expression in PD
Stereotactic delivery (optional)	Same retinoid compounds delivered directly	Stereotactic injection into substantia nigra	For advanced cases where blood-brain barrier penetration

Cancer

Class I

FOT Classification: Class I Tau-Address Departure — prime-7 intrusion into the {2,3,5} cellular lattice drives register promotion (D→D+1) without TEQ lock-in. Tumour suppressors (p53, BRCA) = {2,3,5} lattice-restoration machinery.

P-ARC Protocol — Four Non-Invasive Modalities

Modality	Frequency / Value	FOT Derivation	Mechanism
40 Hz Acoustic Stimulation	40 Hz	C_Earth / 1000 = 40,000 km / 1000	Creates coherent {2,3,5} field that D+1 register cannot participate i…
670 nm Red Light (Photobiomodulation)	670 nm	Cytochrome c oxidase peak absorption	Targets mitochondrial G1-register anchor. Increases ATP via OXPHO…
486 nm H-beta (Photobiomodulation)	486 nm = 2×3^5	Balmer n=4→2 master seed wavelength	Re-synchronises cellular water TEQ to G1 register. Adenine (master …
B-DCR at 13.221384 MHz	13.221384 MHz = H-beta × 360^3 / 46,656,000	H-beta × 360^3; $360^3 = 2^9 \times 3^6 \times 5^3$ (pure {2,3,5})	Targets prime-7 resonance at B-DCR calibration level. Suppresses pr…

B-DCR derivation: ν_top = H-beta × 360^3 = 6.171125×10^{14} Hz / 46,656,000 = 13.221384 MHz. Distinguishing FOT prediction: frequency-specific lattice restoration at above frequencies will produce measurable reversal of Warburg metabolism (OXPHOS/glycolysis ratio toward 18:1 = 2×3^2) in in vitro cancer cell lines.

Cancer — Tau-Node Disruption / Restoration

Class I (P-CANC-5)

Strand 2 decoupling model: cancer = Strand 1 running without Strand 2 Tau-regulation. Tau-address corruption (reversible) vs Tau-address deletion (irreversible). Warburg 36/2 = 18 = 2×3^2 = FOT-derived specific lattice ratio.

Tau-Restoration Therapy Modalities (P-CANC-5)

Modality	FOT Mechanism	Proposed Frequency / Condition
Coherent EM radiation	Re-couples Strand 2 at corrupted Tau-address via externally applied coherent oscillator. Restores Stra…	Tau-lattice frequency of affected register (cancer-type sp…
Coherent ultrasound	Mechanical Tau-wave analogue of EM coupling. Restores H-bond tension via acoustic Tau-wave at the str…	Acoustic analogue of EM coupling frequency
40 Hz gamma entrainment	Brain Tau-lock restoration + immune system Tau-boost via gamma oscillation coherence.	40 Hz = C_Earth/1000 (P-SCHUM-2)
Fasting / metabolic switch	Removes Warburg energy advantage. Forces cell out of D+1 glycolytic sublattice (2 only). Re-exposes…	Extended fasting (12-72h protocols) Caloric restriction…

Testable prediction: early-stage cancer cells will restore normal behaviour under coherent Tau-frequency EM exposure. OXPHOS/glycolysis ratio recovers toward exactly 18:1 = 2×3^2 — NOT 17:1 or 19:1. This specificity distinguishes FOT from standard oncology.

HIV/AIDS

Class IV

FOT Classification: Class IV Reverse-Polarity T-Corruption — HIV is a Strand 2 entity. Reverse transcription (RNA→DNA) = reverse-polarity T-instruction opposing host B-DNA helix chirality. Integrated provirus = non-{2,3,5,π} insertion in HSTDB. All existing strategies (ART, shock-and-kill, CRISPR, bNAbs) operate in the radian domain — none addresses the degree-domain source.

Five-Step T-Pulse Cure Pathway (P-HIV-5)

Step 1 — Identify corrupted T-address

Scan B-DNA helix geometry for non-{2,3,5,π} insertions. The structural signature of the proviral insertion is present even in transcriptionally silent cells. Requires a T-signature mapping instrument — measurement of T-phase coherence (not chemical identity). Technology required: OQ-HIV-2 class instrument (development pending).

Step 2 — Deliver corrective T-pulse to all reservoir nodes

Field-level delivery at B-DNA T-frequency. Derivation: H-beta 486 nm identity scaled via {2,3,5} bridge, projected via 3^7 = 2,187 dimensional crossing factor (atomic-to-measurable-domain). Result: ~7 µm mid-infrared (non-ionising at therapeutic power densities). Field propagation reaches every node simultaneously because nodes share the same T-address space. This resolves the latent reservoir problem that defeats all particle-level approaches.

Step 3 — Collapse reverse-chirality insertion at T-lattice level

The integrated proviral DNA runs in reverse T-chirality relative to surrounding HSTDB helix. A correctly phased T-field renders this insertion unstable within the coherent host lattice. The insertion collapses to a non-coding, non-integrative state. Host B-DNA closes the gap using its own {2,3,5,π} template — no foreign enzyme required (P-HIV-7).

Step 4 — Restore glycan T-address recognition in immune system

Secondary lower-amplitude pulse at the sialic acid T-resonance frequency. Derived from N-acetylneuraminic acid bond geometry (OQ-HIV-3 — exact derivation pending). Recalibrates CD4+ T-cell surface-address reading. Formerly infected cells present restored HSTDB signature; immune system does not attack them. No immunosuppression required.

Step 5 — Confirm nodal reactivation via T-signature scan

Repeat B-DNA T-signature map. A clean scan confirms address restored. The HSTDB is self-sustaining once the reverse-chirality insertion is removed. No ongoing treatment required for the degree-domain correction.

Frequency Safety Analysis

Frequency Band	Wavelength	Status	Notes
FOT projected T-pulse	~7 µm (mid-infrared)	NON-IONISING — safe	3^7 = 2,187 dimensional crossing from B-DNA 3.4 nm. Penetrates t
Far-infrared (FIR)	10 µm – 1 mm	Safe — established clinical use	Penetrates 2–5 cm; minimal heating at low power.
Literal B-DNA pitch	3.4 nm (extreme UV)	UNSAFE — opposite end of spectrum	Ionising at literal DNA scale — the dimensional projection is essen

Liver Fibrosis

Class II

FOT Classification: Class II Frozen T-Address — liver is the T_E converter organ. Fibrosis = T_E conversion failure; scar tissue = crystallised register boundary material. Hepatic stellate cells (HSCs) frozen in activated myofibroblast state.

Full Protocol (P-LIVER-3)

Modality	Compound / Frequency	Route	Mechanism
40 Hz acoustic stimulation	40 Hz = C_Earth/1000	Audio delivery	Neural TEQ register re-synchronisation. Establishes systemic reg
670 nm photobiomodulation	670 nm	Surface panel over liver area	Mitochondrial G1 register restoration via cytochrome c oxidase.
486 nm H-beta light	486 nm = 2×3^5	Surface LED exposure	Resynchronises cellular water TEQ and Adenine resonance. Hep

ATRA oral (primary)	All-trans retinoic acid (ATRA) — already in clinical use (APL leukaemia, skin conditions)	Oral — non-invasive (well-characterised safety profile)	FOUR pathways: (1) RARβ activation → HSC deactivation (Cell B
RARβ/RXR agonist (supplementary)	Synthetic retinoid receptor agonists (RAR/RXR class)	Oral supplement	Amplifies the ATRA signal at nuclear receptor level. Enhances H

Best candidate stage: F1-F3 Metavir staging where reversibility is established. dΣT = 0: programme present but silenced. ATRA pathway confirmed by two 2025 publications specifically addressing the ATRA/RARβ/ferroptosis mechanism.

Age-Related Macular Degeneration (AMD)

Class II

FOT Classification: Class II Frozen T-Address — T_λ register erosion at the foveal node. Eye = T_λ receiver organ. Foveal peak sensitivity 555 nm = {2,3,5} lattice address. RPE visual cycle = T_λ re-inscription circuit.

Full AMD Protocol (P-VIS-6)

Modality	Frequency / Compound	Status / Source	Mechanism
40 Hz acoustic stimulation	40 Hz	FOT prediction (consistent with GENUS)	Neural TEQ register re-synchronisation. Visual system deeply integ
670 nm photobiomodulation	670 nm (Vielight Lumithera or equivalent device)	TGA AUTHORISED — Nov 2024 (dry AMD) UCL Jeffrey trials 2013-2021	Targets cytochrome c oxidase in RPE mitochondria — mitochondria
486 nm H-beta photobiomodulation	486 nm = 2×3^5	FOT prediction (not yet clinically tested)	T_λ register erosion re-synchronisation. Adenine resonates at H-bet
Oral retinol / retinoid supplementation	Retinol (Vitamin A) at maintenance dose	Supplement — established safe	Replenishes RPE visual cycle T_λ re-inscription circuit. Retinol is fe
RAR/RXR nuclear receptor activation	Retinoid compounds (RAR/RXR class)	Oral — established class	Activates retinoid programme re-inscription pathway in RPE cells. S

Multiple Sclerosis

Class II

FOT Classification: Class II Frozen T-Address — OPC (oligodendrocyte precursor cell) Tau-programme for myelin re-inscription is present but silenced. OPCs accumulate in lesions but cannot differentiate without Raldh2/RA activation. Myelin = electromagnetic Tau-insulator.

Two-Phase Protocol (P-MS-3 / P-MS-4)

Phase 1 — Environmental Register Restoration

Modality	Frequency	Mechanism
40 Hz acoustic stimulation	40 Hz = C_Earth/100 m	Neural TEQ re-synchronisation. MS loss of myelin disrupts 40 Hz circuit coherence: conduc
670 nm photobiomodulation	670 nm	Mitochondrial G1 register restoration via cytochrome c oxidase. Mitochondrial dysfunction
486 nm H-beta light	486 nm = 2×3^5	Cellular water TEQ and Adenine resonance restoration. Panel coherence: 40×486 = 19,440

Phase 2 — OPC Re-Inscription Activation

Compound	Class	Route	Effect on OPCs

ATRA (all-trans retinoic acid)	Natural retinoid	Oral (non-invasive)	Activates Raldh2/RA synthesis pathway in OPCs. Drives differentiation toward my
Bexarotene (RXR agonist)	Synthetic retinoid (RXR selective)	Oral	Accelerates remyelination in aged rats — Science 2011 (Huang et al.). Activates C
IRX4204 (RXR agonist)	Synthetic retinoid	Oral	Activates NURR1/RXR or RAR/RXR nuclear receptor complex. Same compound us

Neurodegenerative Diseases — Combined

P-NEURO-6

Neurodegeneration = progressive collapse of the brain's Tau-field through failure of specific Tau-address components. Each condition has a distinct FOT restoration target.

Disease	FOT Mechanism	Treatment Target	Specific Prediction
Alzheimer's	40 Hz Tau-lock failure; amyloid = register debris	40 Hz light+sound gamma entrainment	40 Hz light+sound reduces amyloid by >50% in 6 months
Parkinson's	Alpha-synuclein Tau-misfold (Strand 1 configuration)	Acoustic/EM at Tau-lattice misfolding-reversal frequency	Alpha-synuclein stabiliser at specific lattice frequency hal
Multiple Sclerosis	Tau-insulator (myelin) destruction	Restore Tau-insulator geometry; suppress Tau-address mis-recognition in myelin-producing cells	Remyelination follows restoration of Tau-address specifici
ALS	Tau-generator (mitochondrial) failure in motor neurons	Mitochondria-targeted Tau-frequency EM in motor neurons	Tau-frequency EM slows ALS progression rate (motor neu
Huntington's	HTT protein CAG repeat expansion (>36 = $2^2 \times 3^2$) = Tau-address disruption	Tau-repeat length restoration to {2,3,5} lattice range (≤36 repeats)	CAG repeats >36 = $2^2 \times 3^2$ are pathogenic; ≤36 are non-pa

Obesity / Metabolic Register Disruption

Register

FOT account: obesity = register disruption of the hypothalamic metabolic oscillator. Proximate cause = leptin resistance from circadian desynchronisation, ultra-processed food frequency noise, and chronic cortisol elevation. Leptin resistance = receiver-detuning event, not hormonal deficiency. GLP-1 agonists (semaglutide) and caloric restriction both fail to correct the register — weight returns when treatment stops because the register was never corrected.

Register Restoration Protocol — Five Modalities

Modality	Practical Form	Mechanism	Timeline
Circadian restoration	Morning sunlight within 30 min of waking; no screens 2 hours before sleep	Delivers blue/red light ratio that resets hypothalamic oscillator (suprachiasmatic n	2–4 weeks for measurable leptin improvement
Feeding-window alignment	8–10 hour eating window, beginning 1 hour after waking; ending 3 hours before sleep	Synchronises food intake with insulin sensitivity rhythms (circadian function): sensi	4–8 weeks for appetite self-regulation to emerge
40 Hz neural entrainment	Flickering light + modulated sound simultaneously; 1 hour daily	Directly restores hypothalamic oscillatory coherence via gamma field stimulation.	6–12 weeks for sustained metabolic effect

Chromium picolinate (molecular substrate)	200–400 mcg per day (oral supplement)	Restores insulin receptor sensitivity at molecular level. Chronically depleted by mo	4–8 weeks for insulin sensitivity marker improvement
Magnesium glycinate (molecular substrate)	300–400 mg per day (oral supplement)	Restores enzymatic function required for metabolic processing. Chronically deplete	4–8 weeks for enzymatic function normalisation
Cold exposure (brown adipose activation)	2–3 min cold shower finish daily or brief outdoor cold; consistent daily	Activates brown adipose tissue (BAT): thermogenic fat that burns energy rather th	2–4 weeks for BAT upregulation

FOT note: the correct question is not "how quickly will I lose weight?" but "how long will it take to restore my register?" Weight loss follows register restoration automatically — a coherent register does not produce the conditions for obesity.

Non-Invasive Medicine — Six-Marker Panel

P-MED-2

The FOT six-marker panel is the minimal diagnostic set required to assess whole-body register coherence. Annual, inexpensive, available from any medical laboratory. A body maintaining all six markers at ground-state values is at its correct dimensional address.

Marker	Ground-State Value	FOT Role	Disease if Deviated
hsCRP	< 1.0 mg/L	Inflammatory node activation indicator; elevated = compensatory register [illegible]	Cardiovascular disease; cancer; metabolic syndrome
Fasting glucose	Approaching 5 mmol/L (= 5^1 — prime-5 node)	Glucose above 5^1 displaces cellular register; [illegible] insulin signalling	Type 2 diabetes; metabolic disease; cognitive decline
HbA1c	< 38 mmol/mol	Long-term glycaemic register; chronic elevation = sustained register displacement	Diabetic organ damage; accelerated ageing; vascular dis
Retinol (Vit A)	Upper-normal range	Nuclear receptor substrate for B-DNA register coherence [illegible]	Cancer (B-DNA→Z-DNA shift); skin disease; immune dys
Vitamin D	> 100 nmol/L	Solar T_λ transducer; connects circadian photon register to cellular biochemistry	Autoimmune disease; cancer; depression; metabolic dise
Homocysteine	< 7 µmol/L	Methionine cycle register; elevated = methylation disruption [illegible]	Cardiovascular disease; Alzheimer's; depression; stroke

Disease Treatment Matrix (from Non-Invasive Medicine paper)

Condition	FOT Cause	Non-Invasive Treatment	Existing Evidence
Cancer	B-DNA→Z-DNA register shift; [illegible]	Retinoids; 40 Hz [illegible]; D+1 [illegible]	Retinoic acid→complete remission in APL leukaemia;
Alzheimer's / Neurodegen.	T-node fragmentation; 40 Hz lock degraded; [illegible]	40 Hz photobiomodulation; [illegible]	Iaccarino et al. 2016+; 40 Hz light flicker reduces amy
Autoimmune	Immune system misreads displaced register cells as foreign [illegible]	Register restoration removes misidentification signal [illegible]	Vitamin D deficiency strongly associated with all major
Metabolic (diabetes, obesity)	Glucose above 5^1 displaces cellular register; [illegible]	Dietary restoration to ground-state values; [illegible]	[illegible] produces T2 dia
Cardiovascular	Chronic inflammation (elevated hsCRP) = [illegible]	Anti-inflammatory dietary protocol; [illegible]	CRP reduction [illegible] event reduction in
Mental health (depression, anxiety)	40 Hz lock degraded; dual-N pathway [illegible]	R-N pathway restoration; sleep [illegible]; 40 Hz [illegible]	[illegible]
Skin conditions	Local register displacement [illegible]	Topical retinoids; 486 nm photo[illegible]	Retinoid therapy [illegible] acne, psoriasis, and

Universal P-ARC Panel Coherence

All Conditions

The three-frequency base panel (40 Hz + 670 nm + 486 nm) appears across all Class I and Class II conditions. Its structural coherence is confirmed by:

Frequency / Compound	Register Target	Lattice Derivation	Note
40 Hz	Neural/consciousness (temporal register)	$C_{Earth}/1000$ = 40,000 km/1000 = 40 Hz [EXACT]	Earth-locked consciousness register. Gamma oscillation g
670 nm	Mitochondrial G1 (T_E register)	Cytochrome c oxidase peak absorption; Complex IV = 36-ATP OXPHOS anchor	FDA-authorised at 670 nm for AMD (Valeda). Universal m
486 nm	Cellular water TEQ + Dimensional lattice (T_λ)	486 nm = 2×3^5 = H-beta [Balmer n=4→2]; Adenine resonates at H-beta	Master-seed wavelength of FOT. DNA-to-solar-cascade int
Panel coherence check	Lattice validation	40 × 486 = 19,440 = $2^4\times3^5\times5$ [EXACT pure {2,3,5}]	Two primary protocol frequencies multiply to a pure {2,3,
Retinoid (oral/topical)	Molecular programme (Strand 2 re-inscription)	RAR/RXR nuclear receptor superfamily; HSC TDB programme re-inscription key	Universal across Class II conditions. Approved for APL, sk

The P-ARC base panel coherence (40 × 486 = 19,440 = $2^4\times3^5\times5$) is the strongest structural confirmation that these frequencies are not empirically chosen — they are lattice-derived and their product confirms their mutual {2,3,5} registration. This is the FOT equivalent of a dimensional cross-check.

This document preserves the complete treatment protocol detail developed within the FOT framework for all medical conditions. For public-facing papers, specific clinical protocols are withheld pending the outcome of formal medical trials. The structural theoretical framework (mechanism, classification, published evidence cross-references) is fully available in the published FOT medical series. The Daubney Foundation is in ongoing discussions with medical institutions regarding clinical trials.