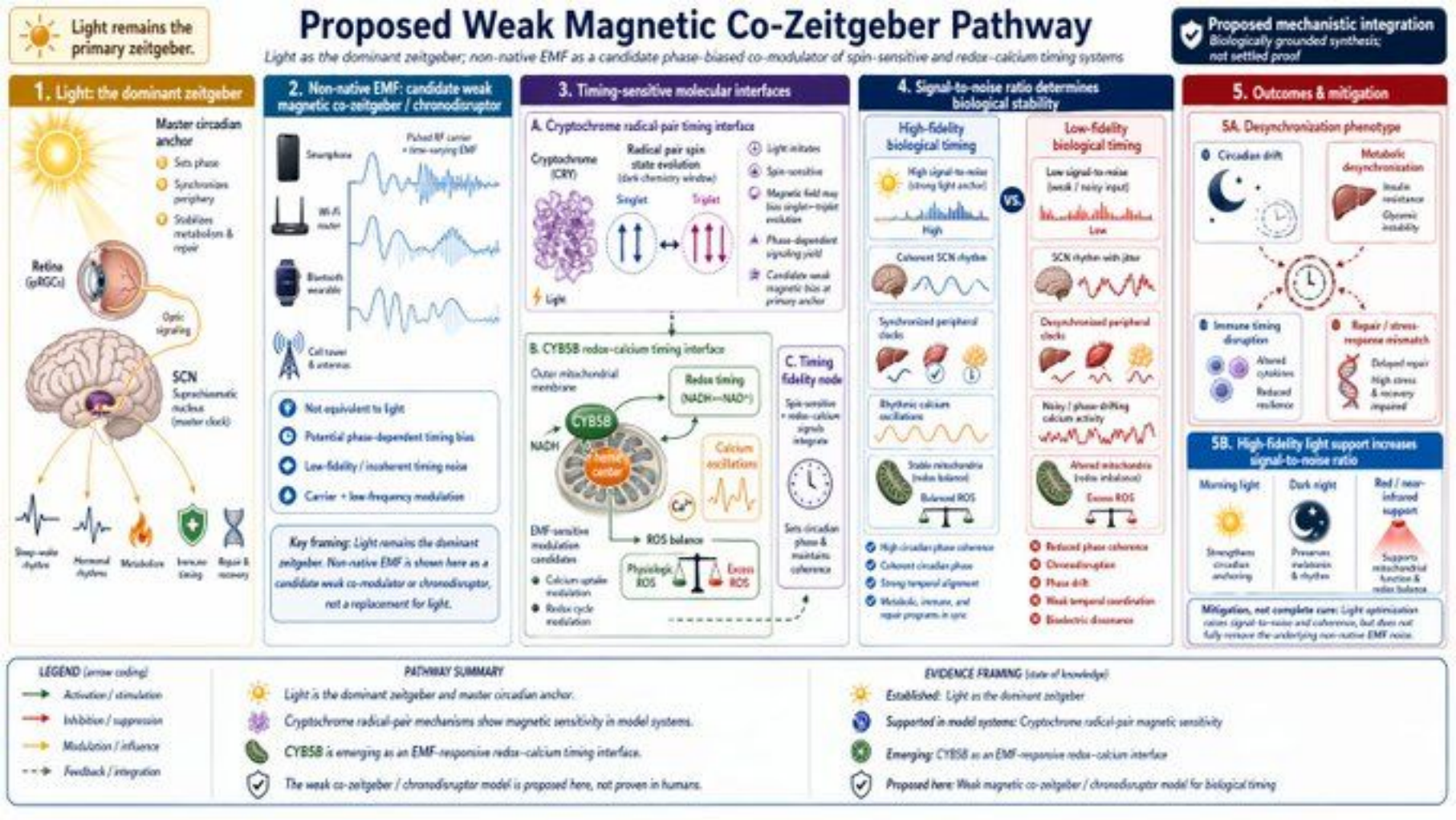
Light remains the primary zeitgeber.
Proposed Weak Magnetic Co-Zeitgeber Pathway
Light as the dominant zeitgeber; non-native EMF as a candidate phase-biased co-modulator of spin-sensitive and redox–calcium timing systems
Proposed mechanistic integration
Biologically grounded synthesis; not settled proof
1. Light: the dominant zeitgeber
Master circadian anchor
Sets phase
Synchronizes periphery
Stabilizes metabolism & repair
Retina (ipRGCs)
Optic signaling
SCN
Suprachiasmatic nucleus (master clock)
Sleep–wake rhythm
Hormonal rhythms
Metabolism
Immune timing
Repair & recovery
2. Non-native EMF: candidate weak magnetic co-zeitgeber / chronodisruptor
Smartphone
Wi-Fi router
Bluetooth wearable
Cell tower & antennas
Pulsed RF carrier + time-varying EMF
Not equivalent to light
Potential phase-dependent timing bias
Low-fidelity / incoherent timing noise
Carrier + low-frequency modulation
Key framing: Light remains the dominant zeitgeber. Non-native EMF is shown here as a candidate weak co-modulator or chronodisruptor, not a replacement for light.
3. Timing-sensitive molecular interfaces
A. Cryptochrome radical-pair timing interface
Cryptochrome (CRY)
Radical pair spin state evolution
(dark chemistry window)
Singlet
Triplet
Light
Light initiates
Spin-sensitive
Magnetic field may bias singlet–triplet evolution
Phase-dependent signaling yield
Candidate weak magnetic bias at primary anchor
B. CYB5B redox–calcium timing interface
Outer mitochondrial membrane
Redox timing (NADH⇌NAD+)
CYB5B
NADH
Heme center
Calcium oscillations
EMF-sensitive modulation candidates
Calcium uptake modulation
Redox cycle modulation
ROS balance
Physiologic ROS
Excess ROS
C. Timing fidelity node
Spin-sensitive + redox-calcium signals integrate
Sets circadian phase & maintains coherence
4. Signal-to-noise ratio determines biological stability
High-fidelity biological timing
High signal-to-noise (strong light anchor)
High
Coherent SCN rhythm
Synchronized peripheral clocks
Rhythmic calcium oscillations
Stable mitochondria (redox balance)
Balanced ROS
High circadian phase coherence
Coherent circadian phase
Strong temporal alignment
Metabolic, immune, and repair programs in sync
VS.
Low-fidelity biological timing
Low signal-to-noise (weak / noisy input)
Low
SCN rhythm with jitter
Desynchronized peripheral clocks
Noisy / phase-drifting calcium activity
Altered mitochondria (redox imbalance)
Excess ROS
Reduced phase coherence
Chronodisruption
Phase drift
Weak temporal coordination
Bioclectric dissonance
5. Outcomes & mitigation
5A. Desynchronization phenotype
Circadian drift
Metabolic desynchronization
Insulin resistance
Glycemic instability
Immune timing disruption
Altered cytokines
Reduced resilience
Repair / stress-response mismatch
Delayed repair
High stress & recovery impaired
5B. High-fidelity light support increases signal-to-noise ratio
Morning light
Strengthens circadian anchoring
Dark night
Preserves melatonin & rhythm
Red / near-infrared support
Supports mitochondrial function & redox balance
Mitigation, not complete cure: Light optimization raises signal-to-noise and coherence, but does not fully remove the underlying non-native EMF noise.
LEGEND (arrow coding)
Activation / stimulation
Inhibition / suppression
Modulation / influence
Feedback / integration
PATHWAY SUMMARY
Light is the dominant zeitgeber and master circadian anchor.
Cryptochrome radical-pair mechanisms show magnetic sensitivity in model systems.
CYB5B is emerging as an EMF-responsive redox–calcium timing interface.
The weak co-zeitgeber / chronodisruptor model is proposed here, not proven in humans.
EVIDENCE FRAMING (state of knowledge)
Established: Light as the dominant zeitgeber
Supported in model systems: Cryptochrome radical-pair magnetic sensitivity
Emerging: CYB5B as an EMF-responsive redox–calcium interface
Proposed here: Weak magnetic co-zeitgeber / chronodisruptor model for biological timing