Proposed Bioelectric Dissonance Pathway to Autoimmunity

Non-native EMF exposure → S4/CYB5B timing disruption → immune mis-coding, sterile danger signaling, and breakdown of self-tolerance

Proposed mechanistic integration
Biologically grounded synthesis, not settled proof

1. Non-native EMF / ELF Exposure

Smartphone
Pulsed electromagnetic signals
Carrier + low-frequency modulation

Wi-Fi Router
Pulsed electromagnetic signals
Carrier + low-frequency modulation
Time-structured environmental input

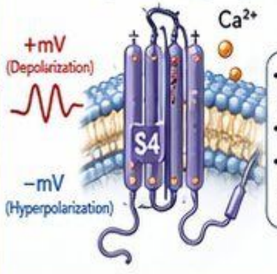
Wearable / Bluetooth
Pulsed electromagnetic signals
Carrier + low-frequency modulation

Cell Tower / Ambient Wireless Field
Carrier + low-frequency modulation

Phase dynamics may bias voltage-sensitive gating

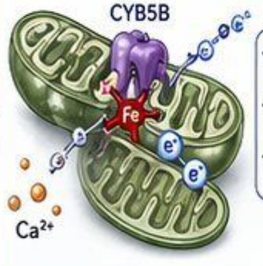
2. Immune decision hardware

A. Membrane target
S4 / voltage-sensitive gating



- Phase-sensitive gating bias
- Mistimed Ca^{2+} entry
- Distorted polarization transients

B. Mitochondrial target
CYB5B mitochondrial EMF-responsive mediator



- Redox-sensitive signaling interface
- Calcium timing modulation
- Mitochondrial stress signaling

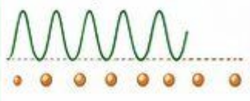
Immune cells decode timing: Ca^{2+} frequency, duty cycle, and membrane-potential transients help determine tolerance vs activation vs inflammation

3. Immune timing-code corruption

Small persistent timing errors may mimic distress or impair tolerance

High-fidelity immune signaling

Ca^{2+} timing



Duty cycle



Polarization transients



Mitochondrial state



- Stable redox
- Efficient ATP
- Low ROS

Downstream interpretation
tolerance / controlled activation

Loss of signaling precision

Bioelectric dissonance

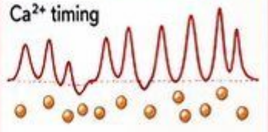
Mitochondrial redox destabilization

Sterile danger signaling

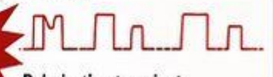
False context decoding

Low-fidelity immune signaling

Ca^{2+} timing



Duty cycle



Polarization transients



Mitochondrial stress



- High ROS
- Impaired ATP
- Redox imbalance

Sterile danger signals / DAMPs

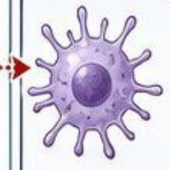


Downstream interpretation
danger / impaired tolerance

Timing code → misread as danger

4. Three converging autoimmune lanes

A. False danger encoding



- NFAT / NF-κB / AP-1 misactivation
- Inflammatory cytokine bias
- Sterile alarm without pathogen
- Aberrant costimulation

IL-1β | IL-6 | TNF-α | Type I IFN

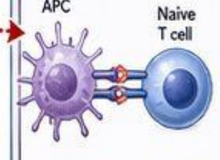
B. Self-antigen distortion



- Oxidized proteins and lipids
- Neoepitope generation
- ATP / mtDNA / HMGB1 release
- Enhanced antigen availability



C. Breakdown of self-tolerance



- Reduced Treg induction / function
- Th17 / Th1 skew
- Inflammatory antigen presentation
- Failure of peripheral tolerance

Treg ↓ | Th17 ↑ | Th1 ↑

Loss of self-tolerance

5. Autoimmune escalation

1. Autoreactive lymphocyte expansion



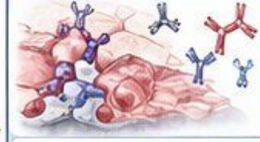
- Self-reactive clonotypes expand
- Memory formation
- Tissue homing

2. Autoantibody production



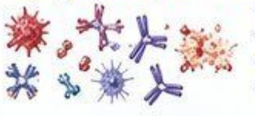
- B cell activation
- Plasma cell differentiation
- Autoantibodies

3. Tissue-targeted immune attack



- Autoantibody binding
- Immune complexes
- Complement activation
- Cellular infiltration
- Tissue injury

4. Epitope spreading and chronicity



- Spreading to new epitopes
- Chronic inflammatory loop
- Persistent tissue damage
- Organ-specific or systemic autoimmunity



Outcome pattern depends on antigen context and host susceptibility

Self-reinforcing autoimmune loop



Legend (arrow coding)

- Solid = established biology
- Dashed = proposed bridge
- Dotted = downstream interpretation

Pathway summary

Non-native EMF → S4/CYB5B timing disruption → Ca^{2+}/ polarization mis-coding → *sterile danger signaling + self-antigen distortion + tolerance failure* → *autoreactive lymphocytes and autoantibodies* → *autoimmune tissue injury*

Evidence framing

Conceptual synthesis: immune cells interpret temporal signaling patterns; disturbed calcium/redox timing is shown here as a proposed upstream contributor to inflammatory misclassification and loss of tolerance.