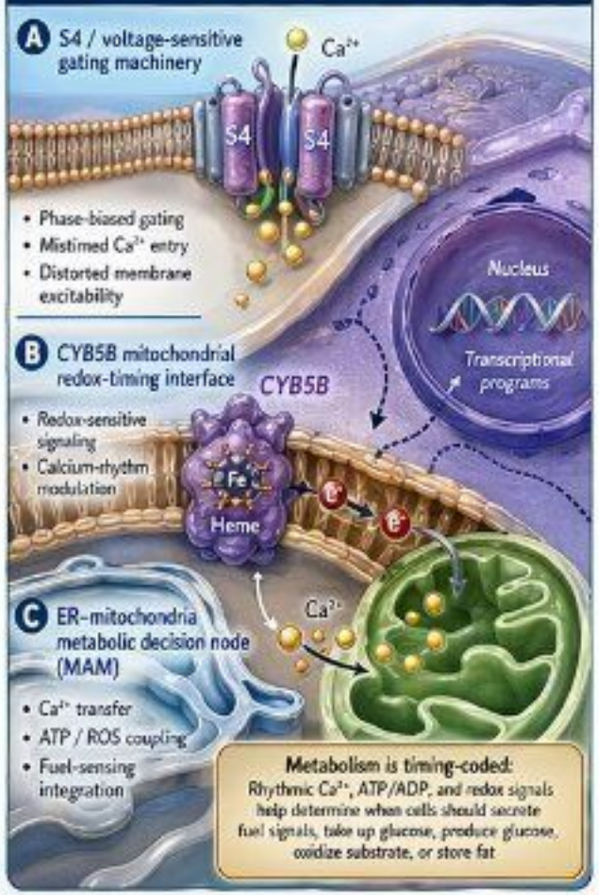
Proposed Bioelectric Dissonance Pathway to Metabolic Dysfunction

Non-native EMF exposure → S4/CYB5B/ER-mitochondria timing disruption → insulin pulse distortion, impaired glucose uptake, excess hepatic glucose output, and metabolic-syndrome drift

Proposed mechanistic integration
A biologically grounded synthesis, not settled proof.

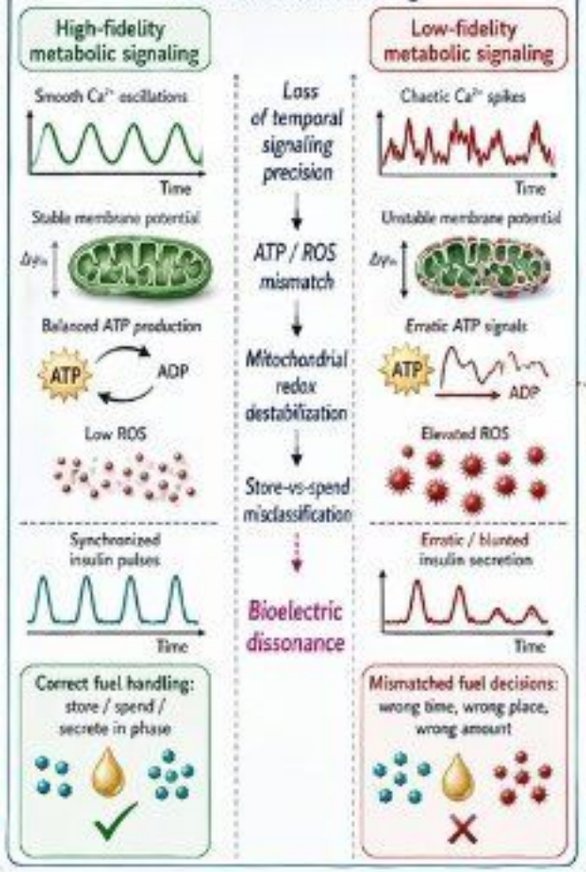
1. Non-native EMF / ELF Exposure

2. Metabolic decision hardware

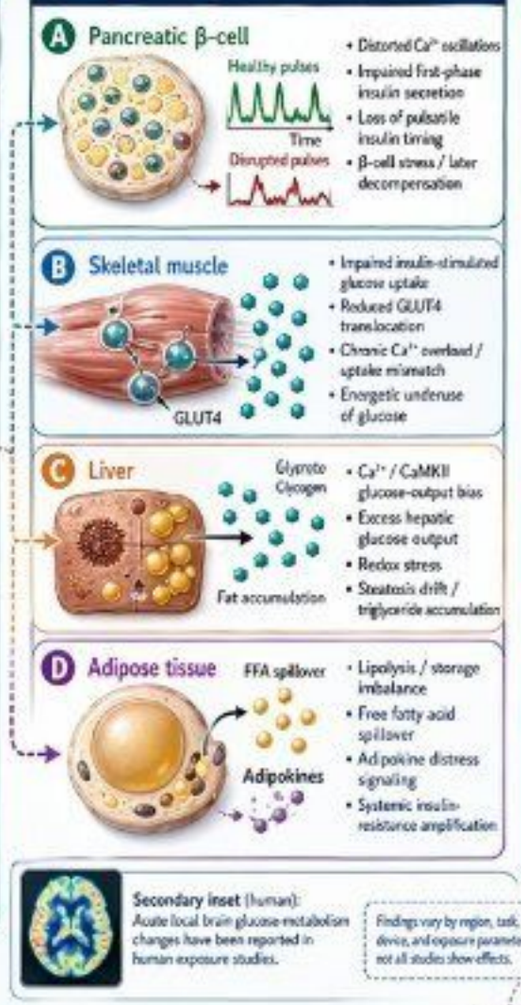


3. Metabolic desynchronization

Timing noise turns coordinated fuel handling into metabolic mis-coding



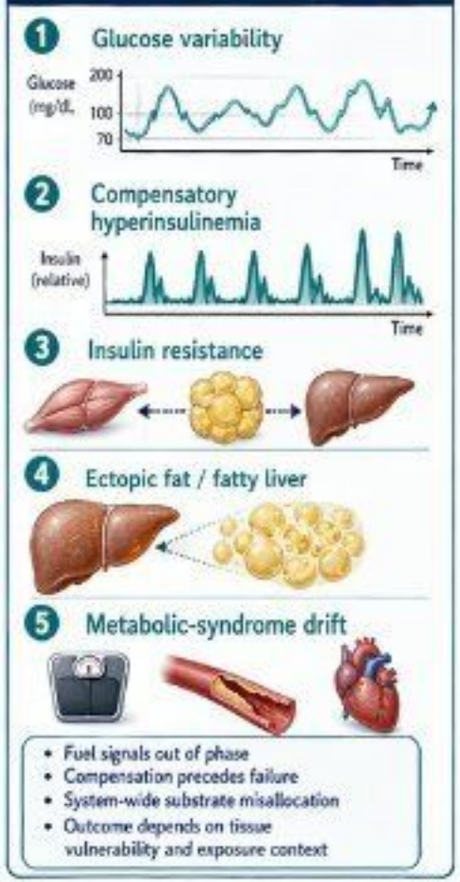
4. Tissue-specific consequences



Secondary inset (human): Acute local brain glucose-metabolism changes have been reported in human exposure studies.

Findings vary by region, task, device, and exposure parameters; not all studies show effects.

5. Metabolic dysfunction phenotype



Ca2+ Calcium (amber) · ATP / Energy · ROS / Redox · Glucose · Fatty acids / Lipids · Insulin / Granules

LEGEND: Arrow code

- Solid → established biology
- Dashed - - → proposed bridge
- Dotted ····→ downstream interpretation

PATHWAY SUMMARY

Non-native EMF → S4/CYB5B/MAM timing disruption → Ca2+ / ATP / redox mis-coding → insulin-pulse distortion + impaired glucose uptake + excess hepatic glucose output + adipose spillover → systemic metabolic dysfunction

SELECTED EVIDENCE ANCHORS (Complementary lines of support)

Human PET: acute local brain glucose-metabolism shift reported

Rat islets: hyperglycemia, oxidative stress, impaired insulin secretion reported

Established physiology: β-cell Ca2+ pulsatility, liver Ca2+ control, muscle Ca2+-glucose coupling, MAMs in insulin resistance

Results depend on exposure context, biology, intensity, and timing.