The Vibrant Longevity Summit

Longevity Foundations

Redefining Oxidative Stress and Cellular Health

Session 1

Dr. Halland Chen, MD

Session 2

Dr. Thomas Sult, MD, IFMCP

Session 3

Dr. Marcos de Andrade, MD

Session 4

Dr. Jay Goodbinder, ND, DC, DABCI

Longevity Foundations

Redefining Oxidative Stress and Cellular Health

Session 1

Dr. Halland Chen, MD

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Longevity Foundations:

Redefining Oxidative Stress and Cellular Health

Meet Your Speaker

Dr. Halland Chen, *MD*

Dr. Halland Chen, MD, is a double board-certified physician, recognized for his innovative approach to accelerating healing and enhancing well-being. His expertise spans longevity, NAD and stem cell research, anti-aging therapies, and non-invasive approaches to pain and injury rehabilitation. He optimizes health through performance enhancement, inflammation reduction, and the integration of advanced regenerative therapies, evidence-based physiological interventions, and targeted nutritional supplementation.

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Objectives

- Identify & Interpret:
  - Key biomarkers of oxidative stress, mitochondrial function, and methylation to detect early cellular decline
- Integrate Frameworks:
  - Apply toxic load, detoxification, and biotransformation principles to guide targeted clinical interventions
- Implement Case-Based Protocols:
  - Translate biomarker data into personalized nutrition, supplementation, and lifestyle plans to improve healthspan outcomes

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Section 2:
Framing the Problem

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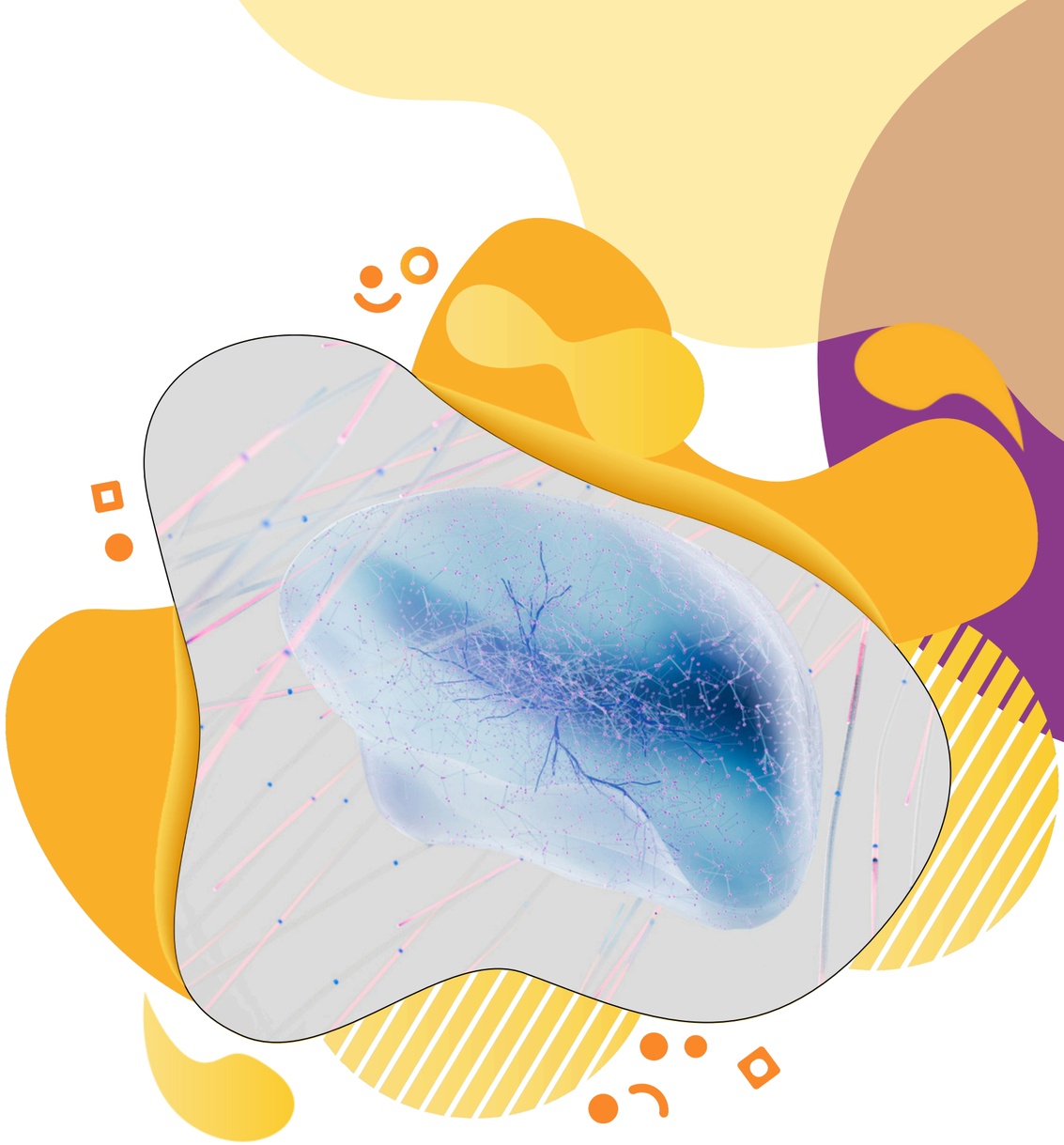
Drivers of Biological Aging

- Oxidative Stress
- Mitochondrial Dysfunction
- Inflammaging
- Detox/Methylation Impairment



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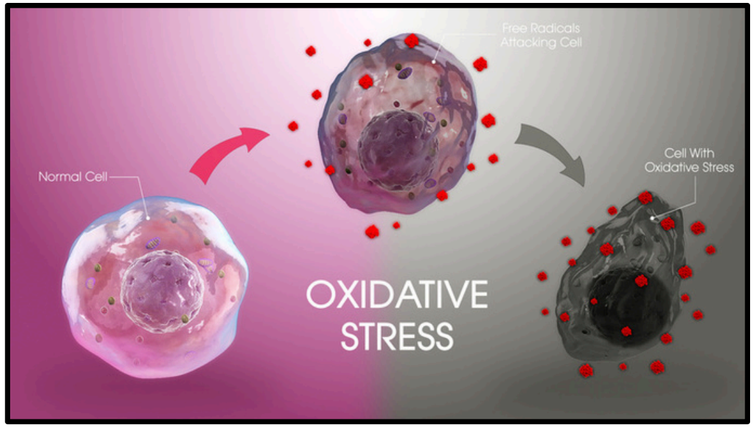
What is Oxidative Stress?



Oxidative stress is one part of our total allostatic load aka the ‘wear and tear’ that accumulates from **toxins, stress, poor nutrition, and environment**

High oxidative stress + low antioxidants → ***faster biological aging***

What Gets Damaged: DNA, proteins, cell membranes

Why It Matters:
Accelerates aging, depletes energy, fuels inflammation
Defenses depend on glutathione, selenium, zinc, magnesium, NAD → micronutrient testing is key

Key Biomarkers:
8-OHdG • F2-isoprostanes • Oxidized LDL
Use biomarkers to guide interventions & confirm progress

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The Oxidative Stress ↔ Mitochondria Loop

GOAL = BREAK THE LOOP

- Oxidative stress damages mitochondria → ↓ ATP → ↑ ROS
- Creates a vicious cycle → accelerates aging
- Positive feedback loop → accelerates aging, fatigue, brain fog, inflammation
- Breaking the loop is key:
  - Rebuild **antioxidant** defenses (glutathione, selenium, NAC, CoQ10)
  - Support **mitochondrial biogenesis** (exercise, fasting, PQQ, red light)
  - **Optimize elimination** (hydration, magnesium citrate, sweating) to clear byproducts
  - **Restoring NAD+** is key to “breaking the vicious cycle” of mitochondrial decline

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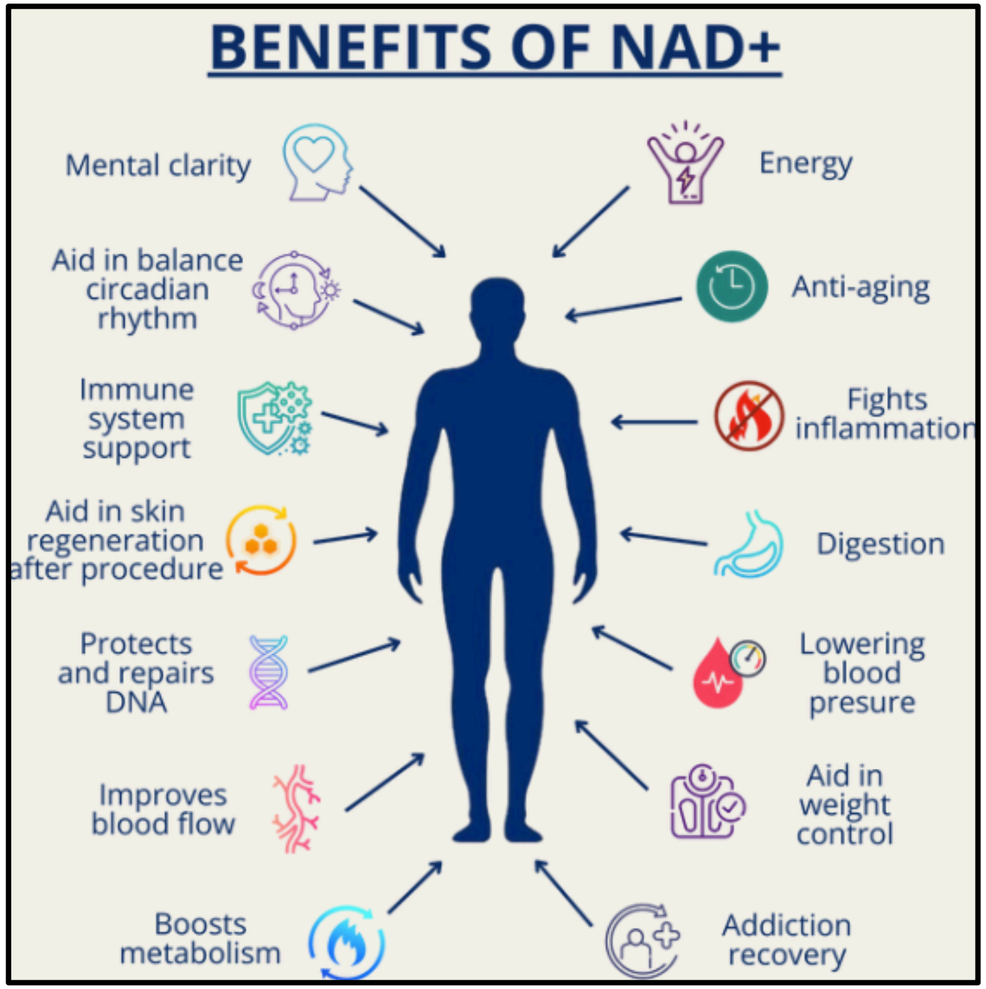
The Role of NAD+ in Cellular Repair and Energy

NAD+ is a coenzyme essential for energy production, DNA repair, and cellular resilience. As we age, NAD+ levels drop, which weakens our ability to respond to stress and recover. Supporting NAD+ is foundational to slowing cellular aging.

Key functions of NAD+:

- Supports mitochondrial ATP production
- Activates sirtuins involved in longevity
- Facilitates DNA repair enzymes (PARPs)



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NAD+ & Cellular Repair

- **↓ NAD+ = slower repair, ↓ energy, ↑ susceptibility to oxidative stress**
- **Contributes to fatigue, brain fog, slower recovery, and inflammaging**

How to Rebuild NAD+

- **Precursors:** NR, NMN, niacin, tryptophan
- **Cofactors:** B2, Mg, B6 to support the NAD+ pathway
- **Lifestyle Boosters:**
  - Exercise & movement (activates AMPK → ↑ NAD+)
  - Fasting/time-restricted eating (stimulates autophagy & NAD+ production)
  - Heat & cold therapy (sauna, cold plunge) → mitochondrial biogenesis
  - Red light therapy for mitochondrial support
  - Optimize circadian rhythm: evening blue-light blockers, morning sun exposure, mouth tape to improve sleep & oxygenation

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Chronic Inflammation the Silent Accelerant

Low-grade inflammation = ***inflammaging***
It damages tissues, disrupts hormone signaling, and raises risk for nearly every chronic disease including atherosclerosis, neurodegeneration, hormone imbalance

- **Drivers of Inflammaging:**
  - Redox stress + toxins (heavy metals, mold, PFAS)
  - Gut dysbiosis, poor diet, chronic infections
  - Sleep disruption, psychosocial stress

Key biomarkers:

- hs-CRP (high sensitivity C-reactive protein)
- IL-6 (interleukin 6)
- TNF-alpha (tumor necrosis factor-alpha)

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Section 3:
Toxic Load & Susceptibility

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Toxic Load & Susceptibility

Total Toxic Load = Exposure × Susceptibility

- **Exposure:** PFAS, pesticides, heavy metals, mycotoxins, solvents
- **Susceptibility:** micronutrient status, gut dysbiosis (β-glucuronidase), Phase I/II capacity
- **Allostatic Load Amplifiers:** prenatal stress, ACEs, geography, occupation, disasters

When toxic load > clearance capacity → chronic inflammation, oxidative stress, and accelerated biological aging

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Common Drivers of Impaired Detox & Biotransformation

- **Overwhelming Total Toxin load** – exogenous + endogenous
- **Poor Elimination** – Constipation, sluggish GI transit, or renal dysfunction
- **Deficiency of detoxifying substances** (antioxidants, cofactors, conjugating agents).
- **Bacterial uncoupling of conjugation** (ie increased Glucuronidase in GI tract)
- **Dysregulation of enzymes** (Phase 1>Phase II>

Key Contributors to Toxic Load

- Endocrine disruptors in plastics, cosmetics, and household cleaners that interfere with hormonal balance
- Heavy metals like mercury, lead, and arsenic that impair mitochondrial function and detox pathways
- Air pollution, pesticides, herbicides
- Alcohol, nicotine, drugs, processed food
- Contaminated air, water, soil
- Psychological stress → ↓ detox efficiency

- EWG
- Think Dirty app

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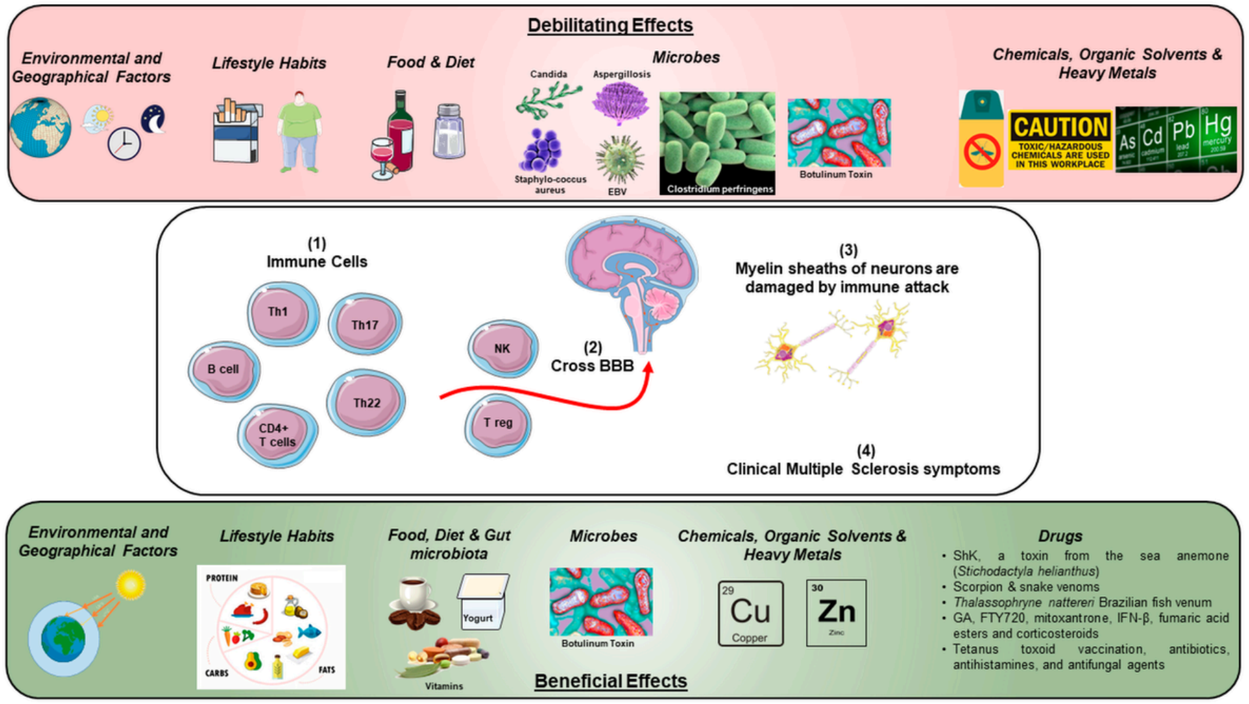
Reduce Exposure → Support Elimination

- Clean water (spring), HEPA air filter
- Check local water supply (EWG.org), use RO filter, avoid tap water
- Eat organic when possible (Dirty Dozen list)
- Optimize bowel regularity (hydration, fiber, magnesium citrate)
- sweating (sauna, exercise)
- binders (citrus pectin, chlorella)
- Sulforaphane; induces phase 2 detoxification enzyme and also inhibiting phase 1 metabolizing enzymes (mostly cytochrome P450)
- Ozone/EBOO therapy



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Section 4:
Biomarkers & Testing

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How Symptoms Show Up Before Disease

By the time disease is diagnosed, cellular health has often been declining for years.

Fatigue, slow recovery, mood shifts, and poor sleep are early clues.

Learn to listen to the body before it breaks down.

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“Normal” to Optimal Health

Why We Test — Not Guess

Functional testing looks beyond “**normal**” ranges to find early imbalances, using **optimal** ranges to guide targeted plans and track progress before disease develops.

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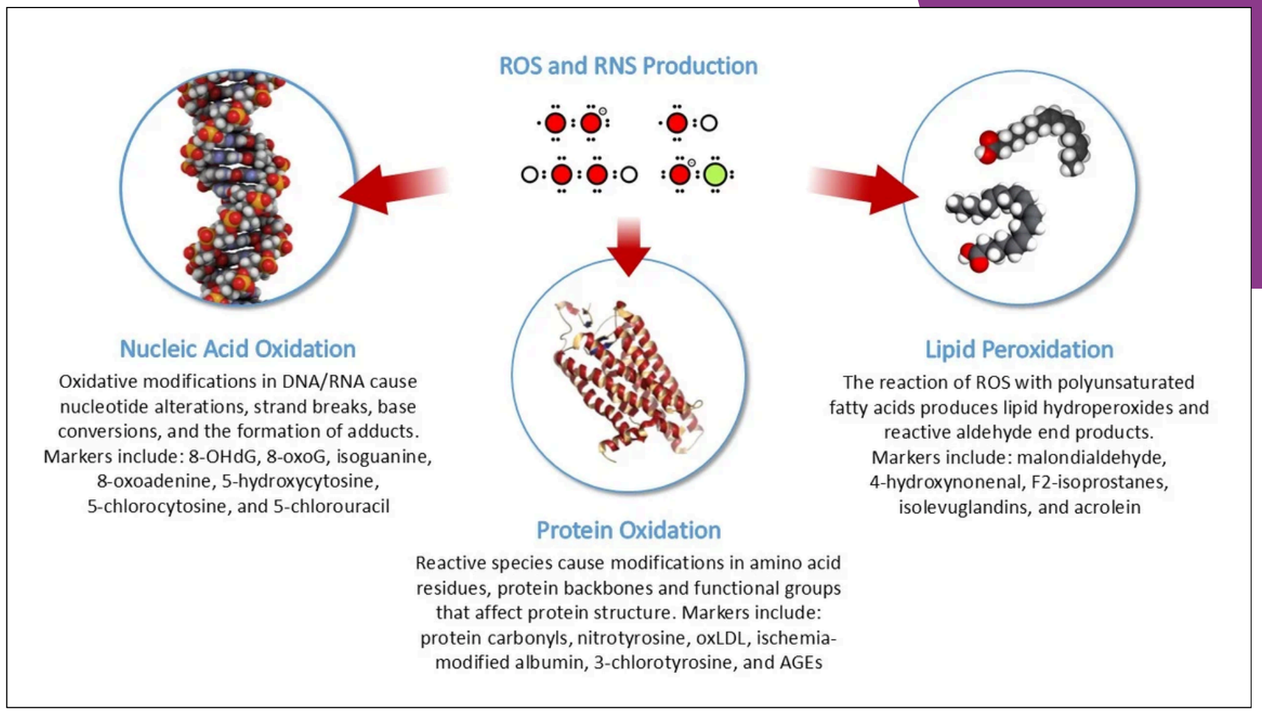
Measuring the Burden of Oxidative Damage

We can directly measure oxidative stress with markers like:

- 8-OHdG (DNA oxidation)
- F2-isoprostanes (lipid peroxidation)
- Glutathione status (reduced vs oxidized GSH)

These markers correlate with CVD, Alzheimer's, and chronic fatigue. Even if cholesterol or CBC is normal, this panel can reveal silent oxidative damage.



THE POWER OF FUNCTIONAL MEDICINE!

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Cellular Health Functional Biomarkers

8-OHdG
Glutathion

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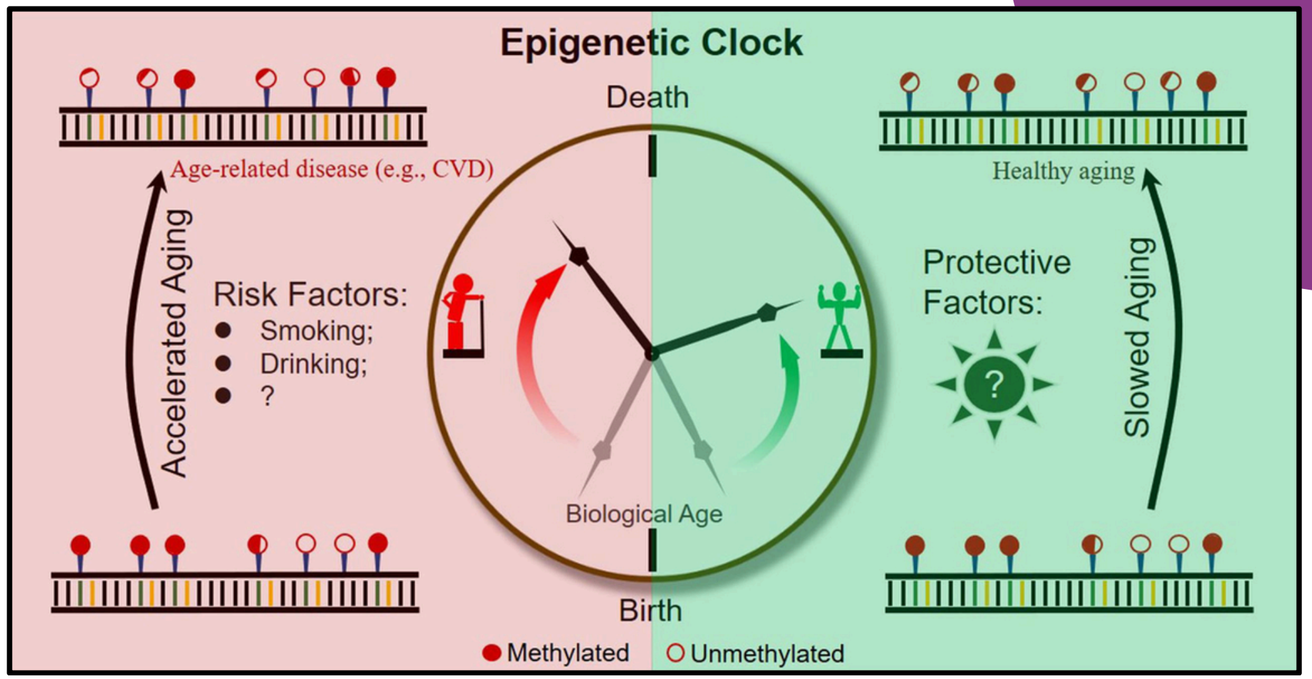
Methylation and its Impact on Aging

Methylation is a biochemical process that affects detoxification, DNA stability, mood, and gene expression. When methylation falters, the body struggles to adapt to stress and clear toxins, both of which contribute to aging.



Critical methylation markers:

- Homocysteine
- SAMe to SAH ratio
- MTHFR gene variants
- Folate
- Vitamin B12

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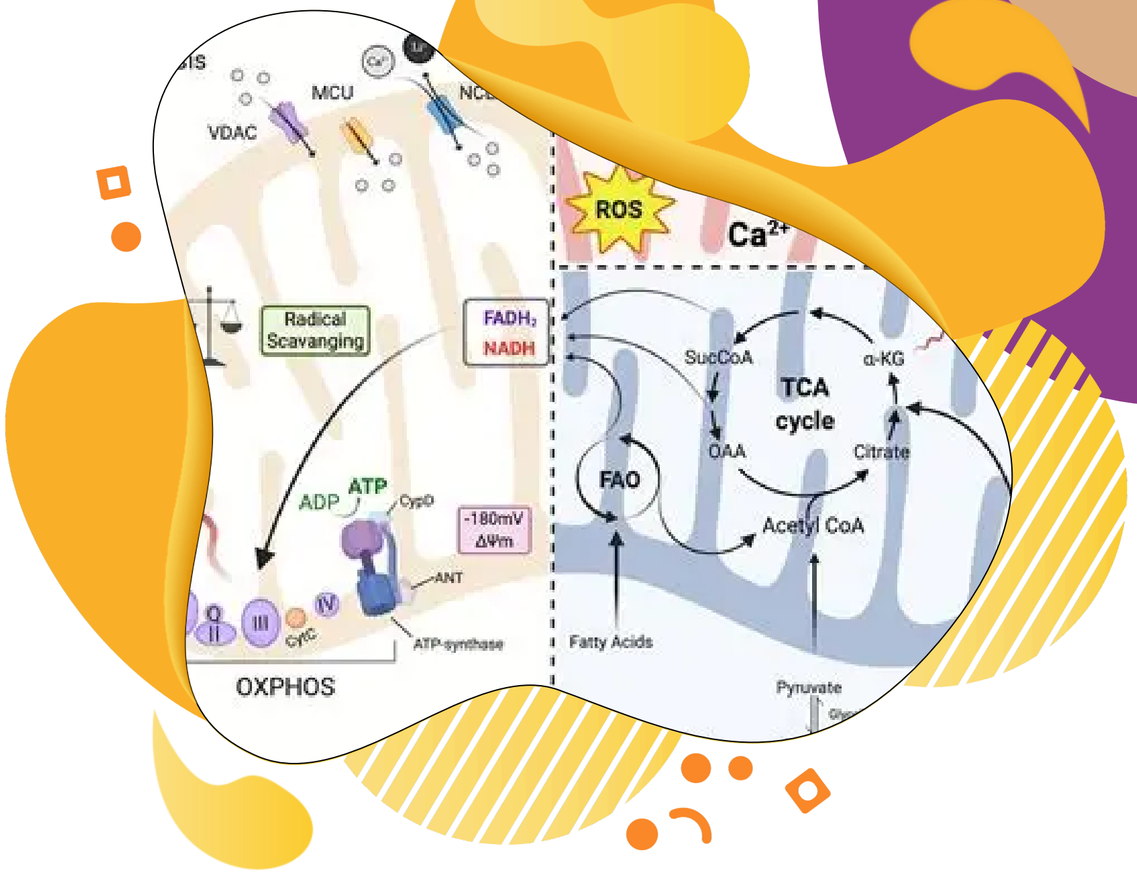
Measuring Mitochondrial Function

Mitochondria are the power plants of your cells. When they slow down or become less efficient, energy drops, and you start to feel it.

Fatigue, brain fog, poor recovery after exercise or stress—these are common signs that your mitochondria are not keeping up.



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Mitochondrial Function Tools

How We Measure Cellular Energy

- Glutathione (GSH): Master antioxidant; low = poor detox & ↑ oxidative stress
- 8-OHdG: Urinary marker of DNA damage → predictor of accelerated aging
- CoQ10: Mitochondrial energy cofactor; low = fatigue, statin effects, CV strain
- Homocysteine: Methylation & CV risk marker; goal <8 µmol/L
- Lipid Peroxides / OxLDL: Show oxidative damage to cell membranes & CVD risk
- Organic Acids: Reveal mitochondrial "traffic jams" (high lactate/pyruvate) → energy inefficiency
- Mitochondrial inefficiency and nutrient cofactor depletion.

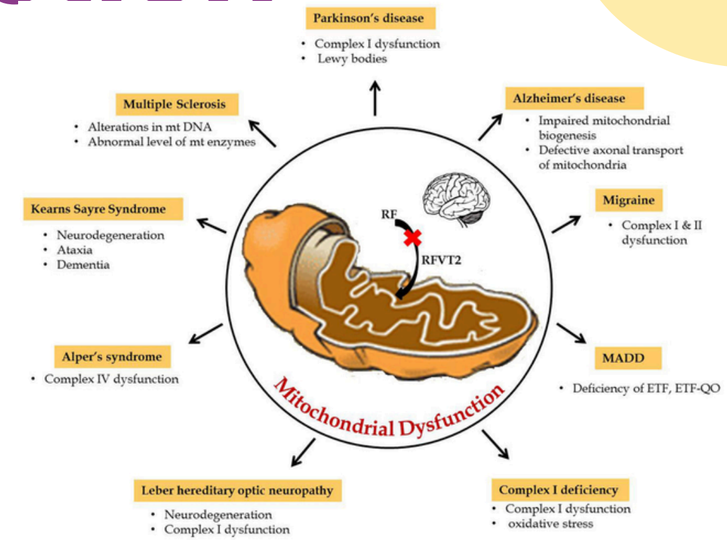
Mitochondria Dysfunction

Case:

A 58-year-old man on a statin presents with new fatigue and mild muscle pain. CBC/CMP WNL.

Findings:

- **Organic Acids:** Elevated lactate + pyruvate (mitochondrial inefficiency)
- **Micronutrients:** Low CoQ10
- **Oxidative Stress Profile:** Elevated lipid peroxides



Interpretation:

Low CoQ10 from statin use impaired the electron transport chain → increased electron leakage → more ROS → muscle symptoms and fatigue.

Intervention:

- Supplemented with ubiquinol (CoQ10) 200 mg/day
- Added antioxidants: vitamin C, alpha-lipoic acid, NAC
- Lifestyle: mitochondrial support (interval training, sauna)

Outcome:

Within 8 weeks, lactate normalized, ROS markers dropped, and fatigue resolved.

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Section 5:

Micronutrients & Food–First: Why genes are not your destiny

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Epigenetics and Lifestyle-Triggered Aging

Your genes are not your fate.

Epigenetics is how your environment and choices influence the way your genes behave. It explains why two people with the same genetic profile can age very differently.

Everyday inputs, like nutrition, sleep, stress, and toxin exposure, can either up-regulate protective genes or activate those tied to inflammation and decline.

Factors that shape gene expression:

- What you eat and how well you methylate
- How you respond to and recover from stress
- Quality of sleep and alignment with your circadian rhythm
- Micronutrients: B vitamins, zinc, and magnesium directly influence methylation and gene expression.

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Nutrient Deficiencies that Disrupt the Equation

Even subtle nutrient deficiencies can quietly impair critical cellular processes—long before any symptoms appear on the surface.

These gaps in micronutrient support can lower resilience, slow detoxification, and disrupt energy production at the mitochondrial level.

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Key Nutrients to Evaluate

Micronutrients are co-factors that turn genes “on” or “off.”
Optimizing them is foundational for methylation, detox, and cellular resilience.

Methylation Support

- B12, B6, Folate: DNA repair, neurotransmitter synthesis, methylation, detox
- Deficiency → ↑ homocysteine, cognitive decline

Minerals & Cofactors

- Magnesium: >300 enzymatic reactions (ATP, Methylation); low levels → muscle tension, anxiety, poor sleep
- Zinc: Immune & antioxidant defense; deficiency slows wound healing
- Iron: Low iron ↑ cadmium absorption, raises lead body burden

Antioxidant Protection

- Selenium: Protects mitochondria, supports thyroid function; buffers mercury & lead
- Vitamin C & E: Regenerate glutathione & CoQ10; deficiency ↑ oxidative stress
- Glutathione: Master antioxidant & Phase II conjugation support

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Micronutrients as Epigenetic Switches

Micronutrients are co-factors for enzymes that turn genes ‘on’ or ‘off’, Optimizing them is foundational for methylation, detox, and resilience.

- ***Magnesium*:**
  - *Critical for >300 enzymatic reactions, including methylation and ATP production.*
  - *Labs: RBC magnesium*
  - *Clinical dosing range: 400–1200 mg/day depending on bowel tolerance and clinical need.*
- ***Selenium***:
  - *Essential for glutathione peroxidase and thyroid enzyme activity.*
  - *Deficiency worsens mercury and lead toxicity, increases oxidative stress, and contributes to hypothyroidism, CV risk, and immune dysfunction.*
  - *Labs: RBC selenium; supplementation: 100–300 mcg/day.*
  - *Food sources: Brazil nuts (1–2 nuts/day), halibut, tuna, sardines, cod.*
- ***Iron***:
  - *Iron deficiency increases gastrointestinal absorption of cadmium and raises lead body burden.*
  - *Assess ferritin, iron saturation; replete cautiously to optimal range.*
- ***Glutathione & Phase II Conjugation***:
  - *Endogenous glutathione declines with age.*
  - *Support with N-acetylcysteine (NAC), glycine, selenium to restore detox capacity and reduce oxidative stress.*

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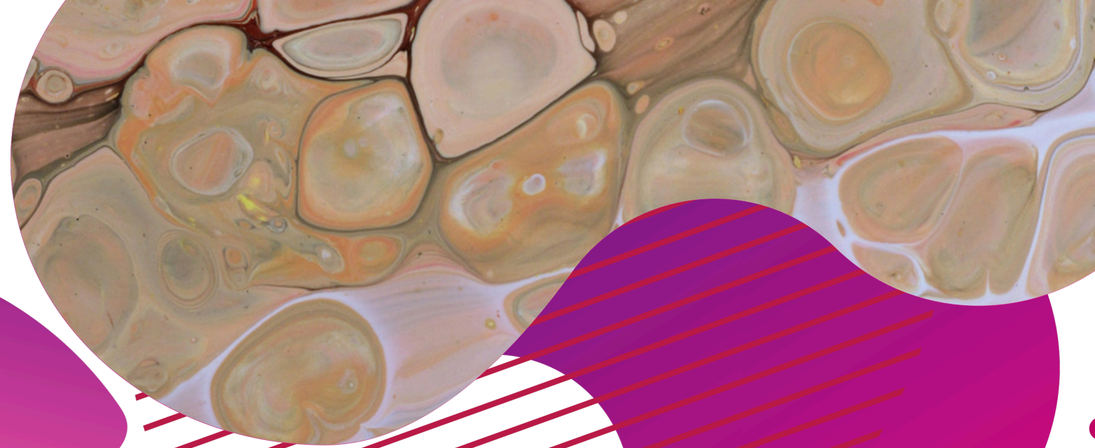
Food-First Detox Foundations

- **Protein sufficiency** – supports Phase II conjugation & tissue repair
- **Cruciferous vegetables** & broccoli sprouts – activate NRF2 → boost detox enzymes
- **Phytonutrient diversity** – aim for ≥30 plant varieties per week
- **Reduce inflammatory inputs** – limit sugar, refined oils, and gluten/dairy
- **Clean air, clean water** – use Reverse Osmosis or spring water; HEPA filter at home



Always optimize Phase II nutrients (protein, glycine, sulfur compounds) before adding binders or aggressive detox therapies.

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Toxic Burden and Micronutrient Testing

Case: **A 44-year-old woman with fatigue, brain fog, and constipation following amalgam removal.**
Findings:

- Total Tox Panel: Elevated mercury
- Micronutrients: Low RBC selenium
- Clinical symptoms: sluggish detox, brittle hair, decreased energy
  Interpretation:
  Low selenium reduced glutathione peroxidase activity → impaired mercury detox → increased oxidative stress and thyroid strain.
  Intervention:
- Selenium repletion: 200 mcg/day (selenium methionine or 2 Brazil nuts/day)
- Binders: chlorella + modified citrus pectin (2h away from meds/supps)
- Supportive care: optimize hydration (2.5–3 L/day), magnesium citrate for bowel motility, cruciferous vegetables (sulforaphane for Phase II), sauna 2–3x/week
  Outcome:
  At 12 weeks: mercury levels decreased, energy and mental clarity improved, bowel regularity restored.

Interventions & Tools

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Mitochondrial Rescue: Where to Begin

1. Reduce Mitochondrial Stress

- Lower toxic burden (e.g., heavy metals, environmental toxins)
- Reduce chronic inflammation and oxidative stress
- Improve sleep and circadian alignment

2. Rebuild the Energy System

- CoQ10 or Ubiquinol: Supports the electron transport chain and ATP synthesis
- Alpha Lipoic Acid (ALA): Recycles other antioxidants and improves insulin sensitivity
- L-Carnitine: Transports fatty acids into mitochondria for fuel
- Magnesium & B vitamins: Essential cofactors for mitochondrial enzymes

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NAD+ Supplementation

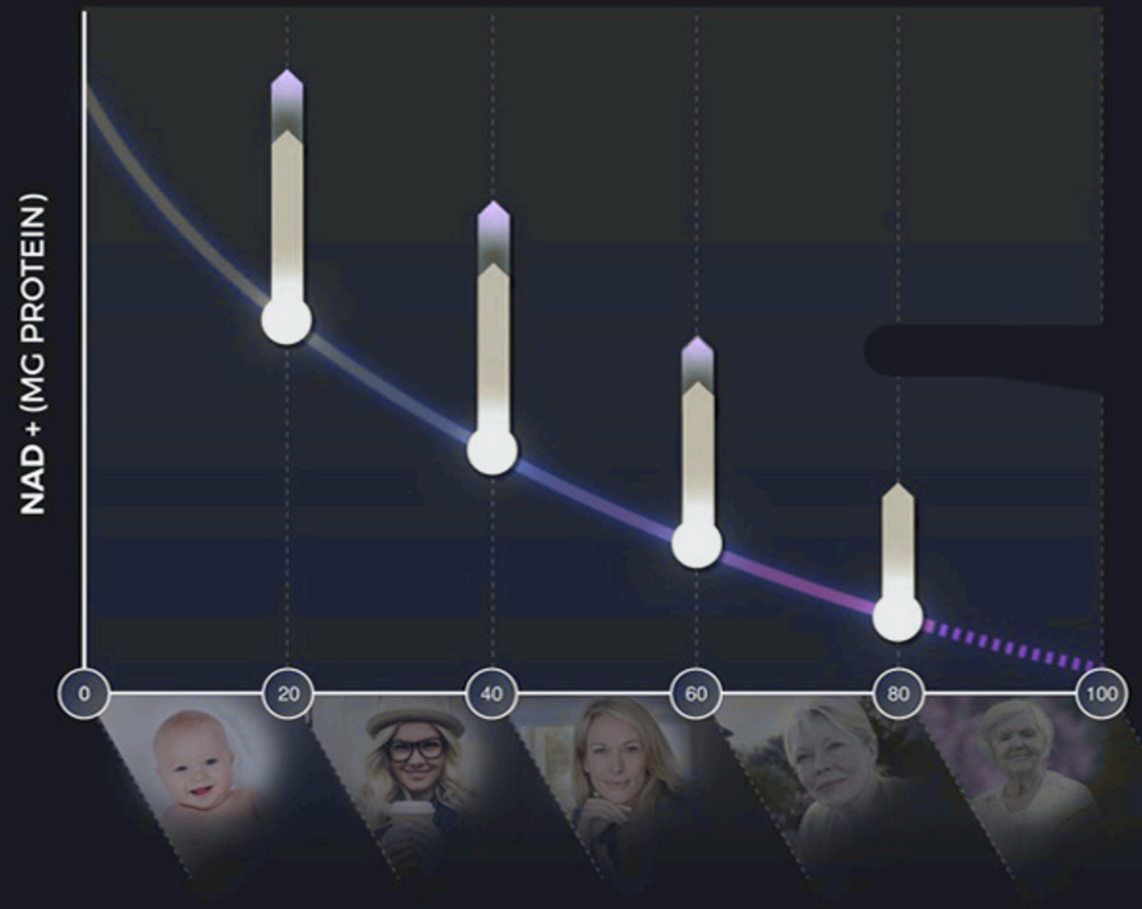
Why it matters:

- NAD+ is a coenzyme central to redox reactions, facilitating ATP production via oxidative phosphorylation
- It fuels sirtuins and PARPs: enzyme families linked to DNA repair, inflammation modulation, and genomic stability
- As levels decline with age, we see a cascade of dysfunction: poor energy output, increased oxidative stress, impaired cellular repair, and inflammatory activation

science:

NAD decline with age

Every 20 Years, NAD+ Levels Drop By 50%



Typical NAD+ Level Loss during aging (NAD+ Levels Decline approx. 50% every 20 years of life)

NAD+ Support

1. Precursors & Cofactors

- NR / NMN / Niacin → boost NAD+ pools
- B2 (riboflavin), Mg → required for NAD+ recycling

2. Supplementation Forms (clinical pearls)

- Nicotinamide Riboside (NR): RCT-supported, raises muscle & brain NAD+
- NMN: Improves insulin sensitivity & vascular health (preclinical)
- IV NAD+: Rapid systemic repletion – supports detox & cognition
- Liposomal NAD+: Gut-absorbed, practical alternative to IV

3. Lower NAD+ Drain

- Reduce CD38 activity (quercetin, apigenin)
- Address chronic inflammation & infections

4. Lifestyle Synergy

- Exercise, sauna, cold exposure → boost NAD+ biosynthesis
- Fasting / TRE → preserves NAD+ & activates sirtuins

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NAD+ Applications

Intravenous delivery for rapid cellular restoration

IV NAD+ provides the most complete and fast-acting method of restoration. It enters the bloodstream directly and reaches target tissues with high bioavailability.

Typical protocol:

- Dose range between 500 and 750 milligrams
- Administered slowly under clinical supervision

Considerations:

- Monitoring is required during infusion
- Nausea or shortness of breath may occur
- Often used as an initial loading dose to saturate depleted systems

Maintenance:

After IV therapy, NAD+ levels can be supported using oral or transdermal options to sustain benefits over time.

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The Synergy of Systems

Cellular aging doesn’t happen in silos. Oxidative stress, mitochondrial decline, inflammation, detox inefficiency, and nutrient depletion are all interconnected, and so are the solutions.

To restore mitochondrial function, we start by removing barriers and supplying core nutrients.

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The Synergy of Systems

- No single supplement or test creates resilience. *It's the synergy between systems.*
- When we reduce toxic burden, restore energy production, support NAD^+ and antioxidants, and correct methylation, we enable the body to become far more capable of repair and adaptation.
- Longevity begins by understanding that cellular systems speak to each other. The goal isn't to fix one piece, it's to restore communication across the whole network.

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Redefining Longevity—From Intervention to Optimization

Living longer is not the only goal. The aim is to extend the number of years lived with energy, clarity, strength, and purpose.

We're moving away from waiting for disease to show up. Instead, we're designing protocols that preserve function and build resilience.

By watching the right markers, we can take action long before symptoms take hold.

This is how we create a personalized, proactive roadmap, rooted in how the body repairs, adapts, and thrives.

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Final Thoughts

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Thank You!

To learn more about regenerative medicine visit www.DrHalland.com

Join Our Waitlist: Register for a complimentary 1 month supply of NAD+ product.

CELL THEORY

Longevity Foundations

Redefining Oxidative Stress and Cellular Health

Session 2

Dr. Thomas Sult, MD, IFMCP

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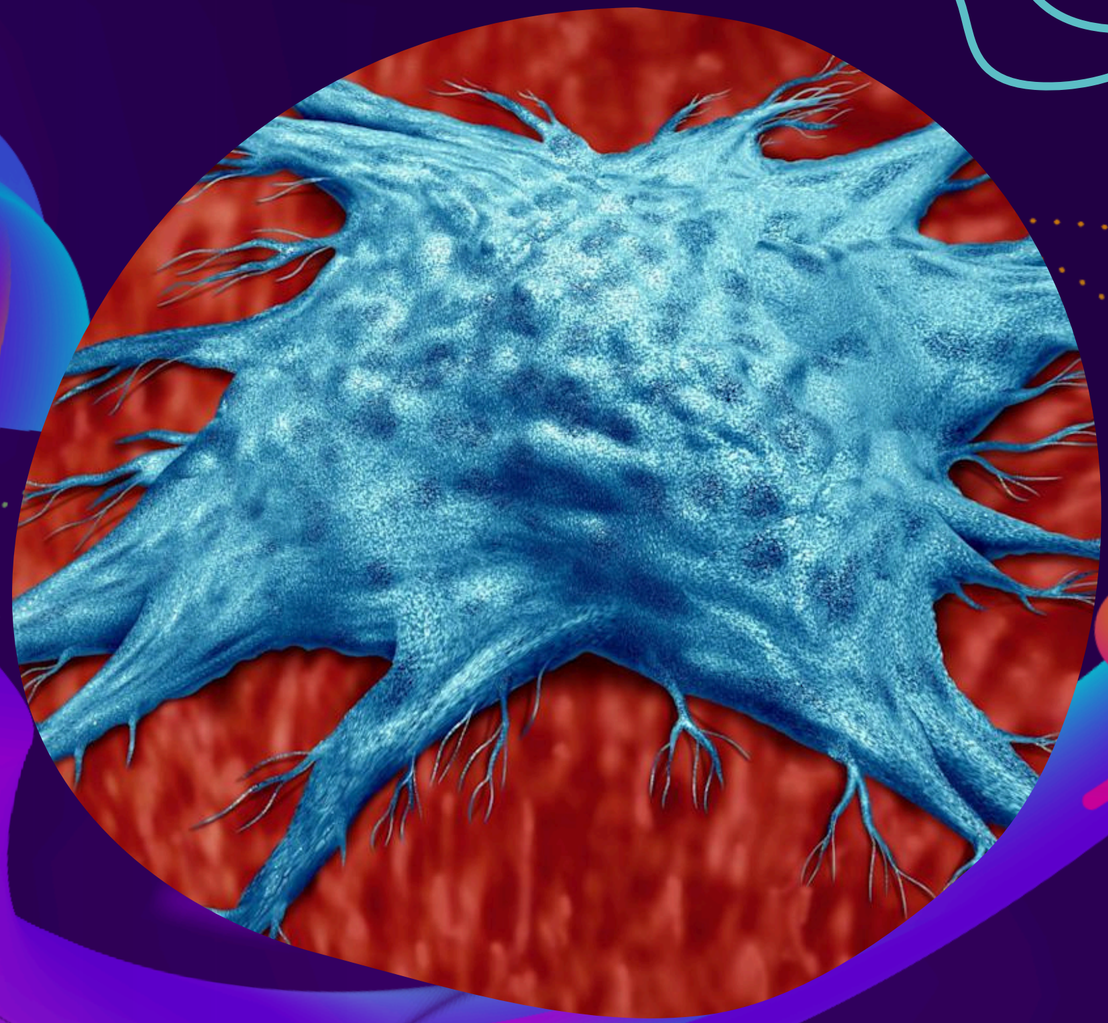
Calming The Zombie Apocalypse Within



Precision Age Management and the Science of Senescent Cell Reversal

Let's meet Tom

Dr. Thomas Sult

Dr. Thomas A. Sult is a graduate of the UCLA School of Medicine (MD), a Fellow of the American Academy of Family Physicians (FAAFP), a Diplomate of the American Board of Family Medicine (ABFM), a Diplomate of the American Board of Physician Specialties in Integrative Medicine (ABIM), and a Diplomat of the American Board of Integrative Holistic Medicine (ABIHM). He is also an Institute for Functional Medicine (IFM) Certified Practitioner and part of its faculty. As Faculty he teaches Both nationally and internationally in the Applying Functional Medicine in Clinical Practice (AFMCP) program, The Immune and GI Modules. He has been practicing Functional Medicine for nearly 30 years. Dr. Sult has 2 adult sons, with associated wonderful daughter-in-laws, 5 grandchildren. He lives with 2 Newfoundland dogs, Elizabeth, and her 3 boys.

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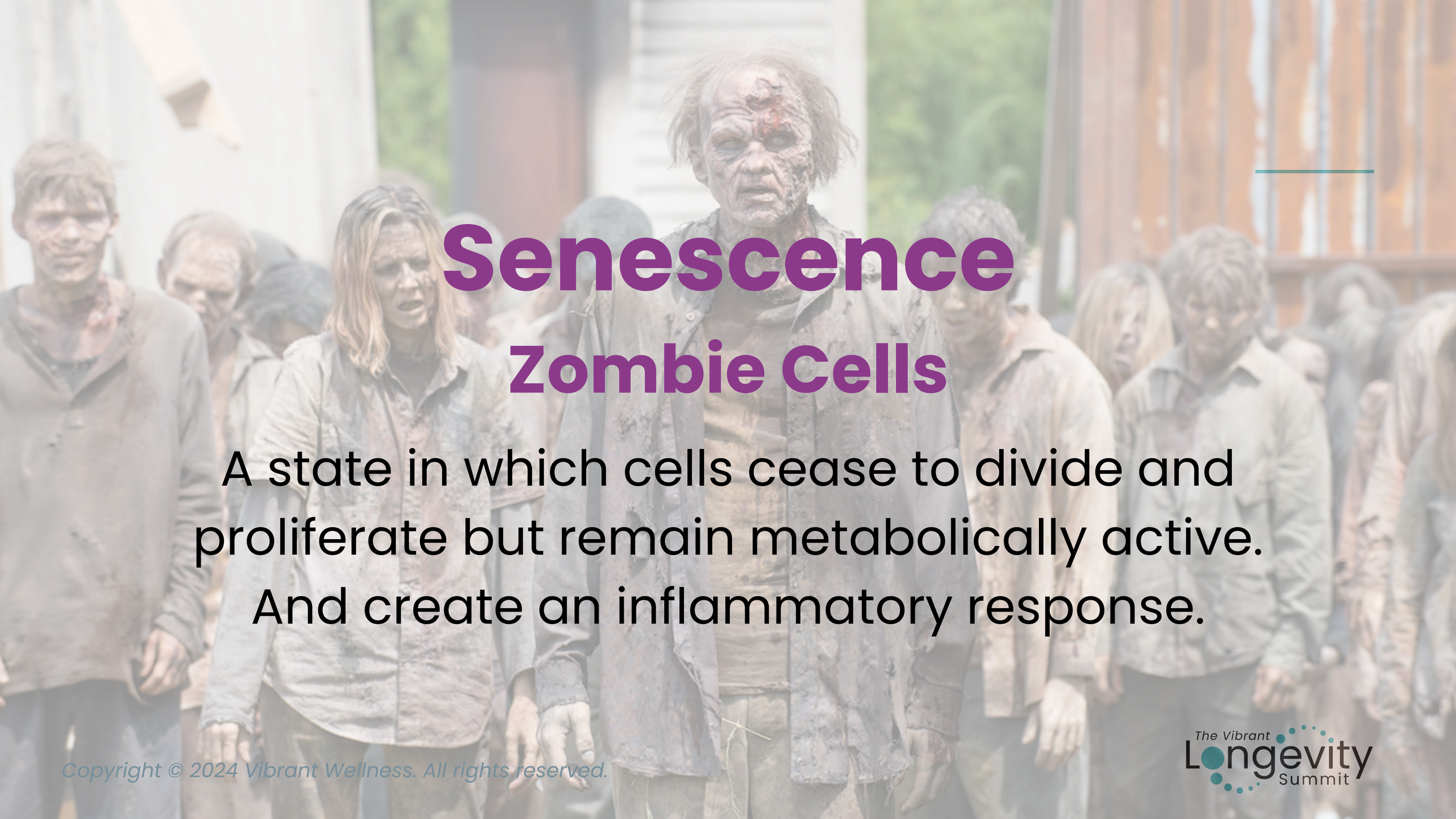

Senescence

Zombie Cells

A state in which cells cease to divide and proliferate but remain metabolically active. And create an inflammatory response.

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Estimated Senescent Cell Burden by Age

Age Range (Years)	Estimated Senescent Cells (% of total cells)	Notes
40–49	~1–3%	Noticeable increase in pro-inflammatory signaling in tissues like fat, muscle, joints
50–59	~3–5%	More senescence-linked dysfunction appears: osteoarthritis, insulin resistance, immune aging
60–69	~5–8%	Accelerated accumulation in chronic disease states or high oxidative burden
70–79	~8–12%	“Zombie” cells increasingly drive frailty, cognitive decline, and inflammation
80+	~10–20%	High burden in many tissues; senolytic therapies may have the greatest impact here

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Zombie Cells

- Why Are Zombie Cells Harmful?
- Secretion of Harmful Molecules (SASP): Senescence-Associated Secretory Phenotype.
- Chronic Inflammation:
- Disruption of Tissue Function:
- Induction of Senescence in Neighboring Cells:

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Senescence-Associated Secretory Phenotype (SASP) is a powerful pro-inflammatory secretome produced by senescent cells.

It includes:

- **cytokines:**
  - **IL-6, IL1B, TNF-a, IL-8**
- **chemokines:**
  - **CCL2, CXCL1, CXCL10, CCL5**
- **growth factors:**
  - **VEGF, TGF-B, IGFBPs, GM-CSF**
- **proteases:**
  - **MMP-1, MMP-3, MMP-9, PAI-1, Cathepsins**
- **extracellular vesicles:**
  - **miR-21, miR-146a, Exposome-packaged Cytokines, mtDNA, oxidized lipis,**

All of which can disrupt tissue structure, promote inflammation, and alter the behavior of nearby cells.

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Zombie Cells

- Contributing to Diseases:
  - Zombie cells are implicated in:
    - **Cardiovascular diseases:** Promoting atherosclerosis and heart failure.
    - **Neurodegenerative diseases:** Contributing to Alzheimer's and Parkinson's through neuroinflammation.
    - **Cancer:** Creating tumor-promoting microenvironment through chronic inflammation.
    - **Diabetes:** Worsening insulin resistance and pancreatic beta-cell dysfunction.
    - **Fibrosis:** Driving tissue scarring in organs like the lungs, kidneys, and liver.
- Prevention of Normal Tissue Renewal:

Elevated senescent cells "zombie cells”
may be as important or even more fundamental than traditional risk factors in the development of cardiovascular disease (CVD), neurodegeneration, cancer, and type 2 diabetes (DM2).

Suda et al. – Cells (2023)
Title: Senescent Cells: A Therapeutic Target in Cardiovascular Diseases
Summary: Detailed review explaining how accumulation of senescent cells—particularly in endothelial, smooth muscle, and immune cells—promotes atherosclerosis, fibrosis, and vascular dysfunction via SASP factors, independent of conventional risk factors. The authors highlight preclinical improvements in vascular health using senolytics
jci.org+4pmc.ncbi.nlm.nih.gov+4mdpi.com+4mdpi.com+1pubmed.ncbi.nlm.nih.gov+1.
DOI: 10.3390/cells12091296

Childs, Durik, Baker & van Deursen – Nature Medicine (2015)
Title: Cellular senescence in aging and age-related disease: from mechanisms to therapy
Summary: Seminal review establishing senescent cells as central actors in age-related diseases. They demonstrate that transplanting senescent cells induces multi-organ dysfunction—even in the absence of traditional risk factors—and that senescent cell removal restores tissue health across cardiovascular, metabolic, and neurodegenerative models mdpi.com+1frontiersin.org+1.
DOI: 10.1038/nm.4000

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Health Span Panel

VibrantWellness

Healthspan Assessment Panel Markers List

Anemia
- Ferritin
- Iron
- UIBC
- TIBC
- Transferrin
- Transferrin Saturation

Nutrition
- Folate
- Vitamin D, 25-OH
- Vitamin B12

Hormones
- Estradiol
- FSH
- DHEA-S
- LH
- SHBG
- Cortisol
- Testosterone, Total
- Free Testosterone
- Progesterone
- Parathyroid Hormone
- Prolactin
- Dihydrotestosterone
- Pregnenolone

Rheumatoid Arthritis
- RF IgM
- Anti-CCP3 IgG and IgA
- hs-CRP

ANA IFA Panel

Thyroid
- T3 - Triiodothyronine
- T4 - Thyroxine
- Free T3
- Free T4
- TSH
- Anti-TPO
- Reverse T3
- Anti-TG

CBC, Differential, & Platelets
- WBC
- RBC
- Hemoglobin
- Hematocrit

CBC, Differential, & Platelets, continued
- MCV
- MCH
- MCHC
- RDW - SD
- RDW - CV
- Platelet Count
- Neutrophils
- Lymphocytes
- Monocytes
- Eosinophils
- Basophils
- Immature Granulocytes
- Neutrophil Count
- Lymphocyte Count
- Monocyte Count
- Eosinophil Count
- Basophil Count
- Immature Granulocyte Count
- MPV
- Nucleated RBC Count
- Nucleated RBC %

Reticulocytes
- Reticulocyte Count
- Reticulocyte (%)
- IRF (Immature Reticulocyte Fraction)
- Reticulocyte Hemoglobin

CMP
- Sodium
- Potassium
- Chloride
- Carbon Dioxide
- Glucose (Renal)
- BUN
- Creatinine
- eGFR
- eGFR (African American)
- BUN/Creatinine Ratio
- Calcium
- Albumin
- ALT
- AST
- Bili, Total
- Protein, Total
- Alkaline Phosphatase
- Serum Osmolality

Lipids
- Cholesterol, Total
- LDL Calculation
- HDL Direct
- Cholesterol/HDL Ratio
- Triglycerides

LDL Direct

Apolipoproteins
- Apo A-1
- Apo B
- Apo B: Apo A-1

Inflammation
- PLAC
- Homocysteine
- ox-LDL
- MPO
- hs-CRP

Mycocardial Stress
- NT-proBNP

LipoProtein Markers
- sdLDL
- Lp(a)

Glycemic Control
- Glucose (Diabetes)
- Hemoglobin A1C
- Glycated Serum Protein

Insulin Resistance
- Adiponectin
- Ferritin

Beta Cell Function
- Insulin

Other Markers
- Total IgG
- Total IgM
- Total CK
- Uric Acid
- Human IGF-1
- Cystatin C
- GGT
- LDH
- Leptin

Revised 05/07/2025

Health Span Panel

Anemia
Nutrition
Hormones
Rheumatology
Thyroid
CBC
CMP
Lipids Expanded
Inflammation
Myocardial Stress
Glycemic Control
Insulin Resistance
Beta Cell Function
Immune Marker

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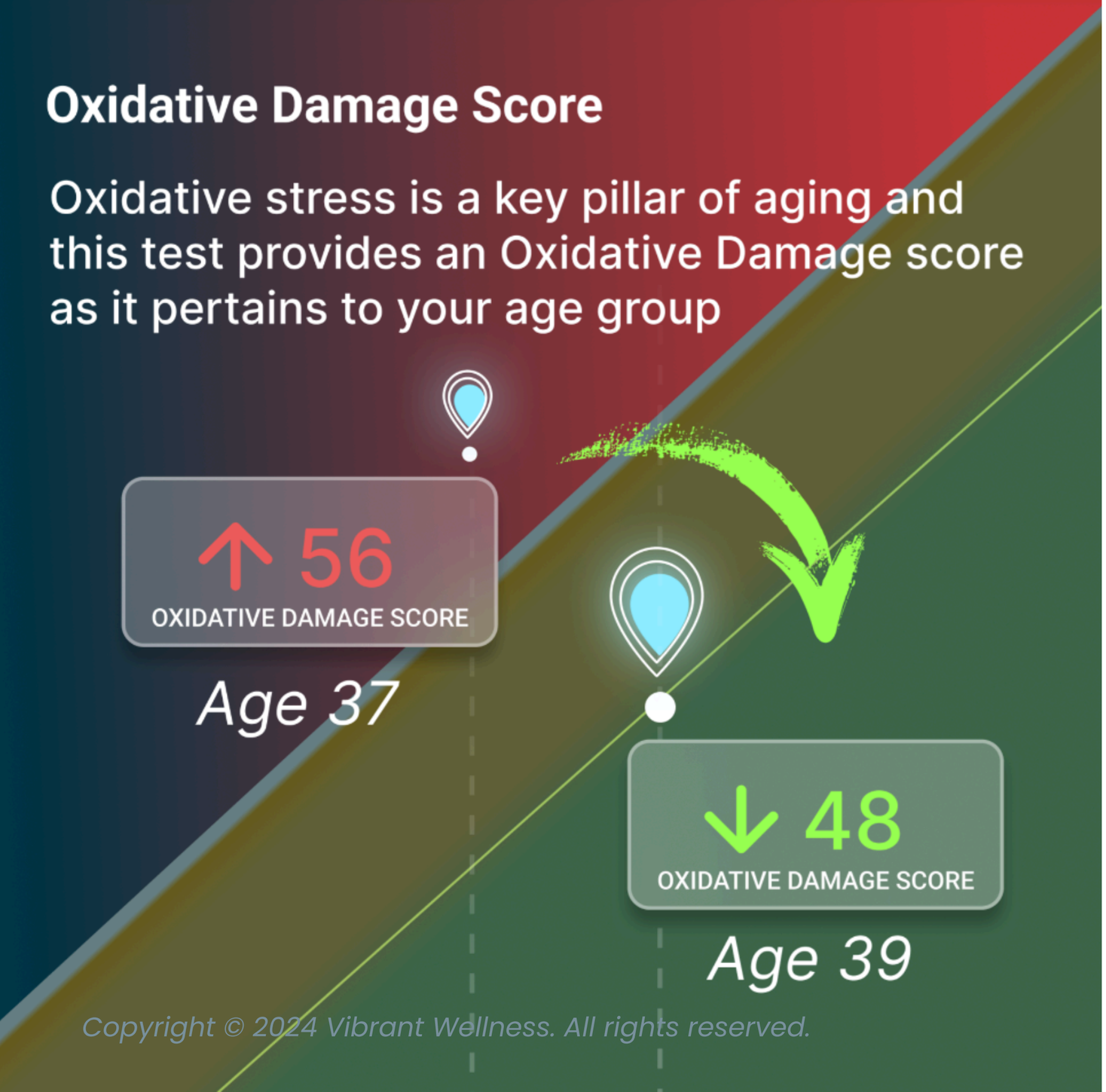
Oxidative Stress Profile



What is Measured:

- 29 Genetic Markers & Enzymatic Antioxidant Response Markers
- 11 Oxidative Damage & Cellular Stress Markers
- 5 Nitrative & Glycation Stress Markers

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Jackson R.

49 year old

Background:

- High-performing biohacker
- Startup founder.
- Fit, focused, and metabolically lean.
- Seeks Precision Age Management insights to extend healthspan.

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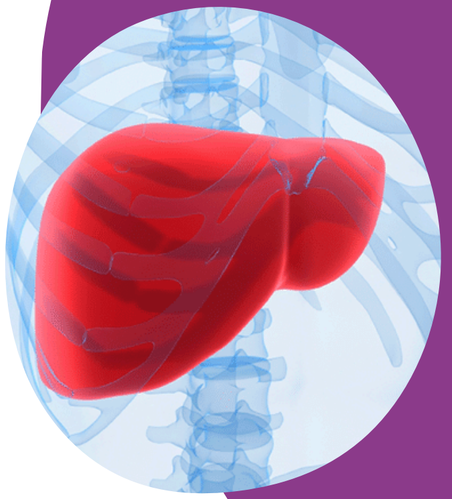
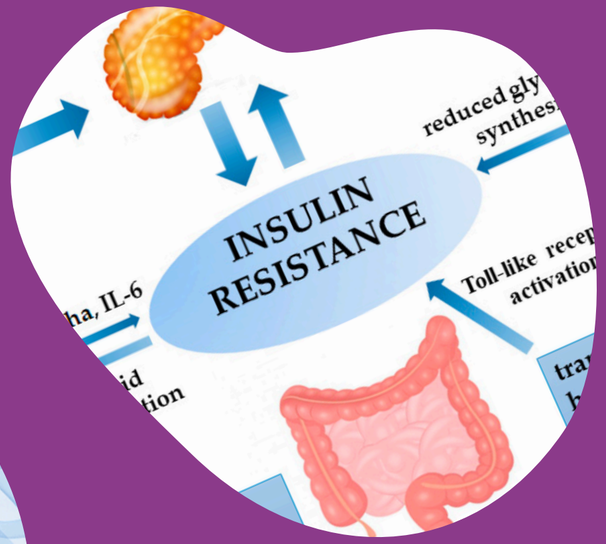
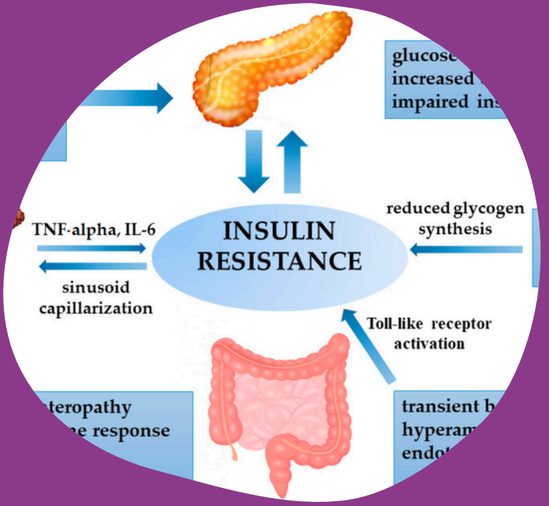
Healthspan Assessment Panel:

- *hs-CRP: 2.7 mg/L – SASP cytokine load*
- *ox-LDL: elevated – redox-modified lipoproteins → immune activation*
- *Ferritin: 310 ng/mL – redox-active iron, inflammatory mimic*
- *Adiponectin: low-normal – impaired insulin sensitivity marker*







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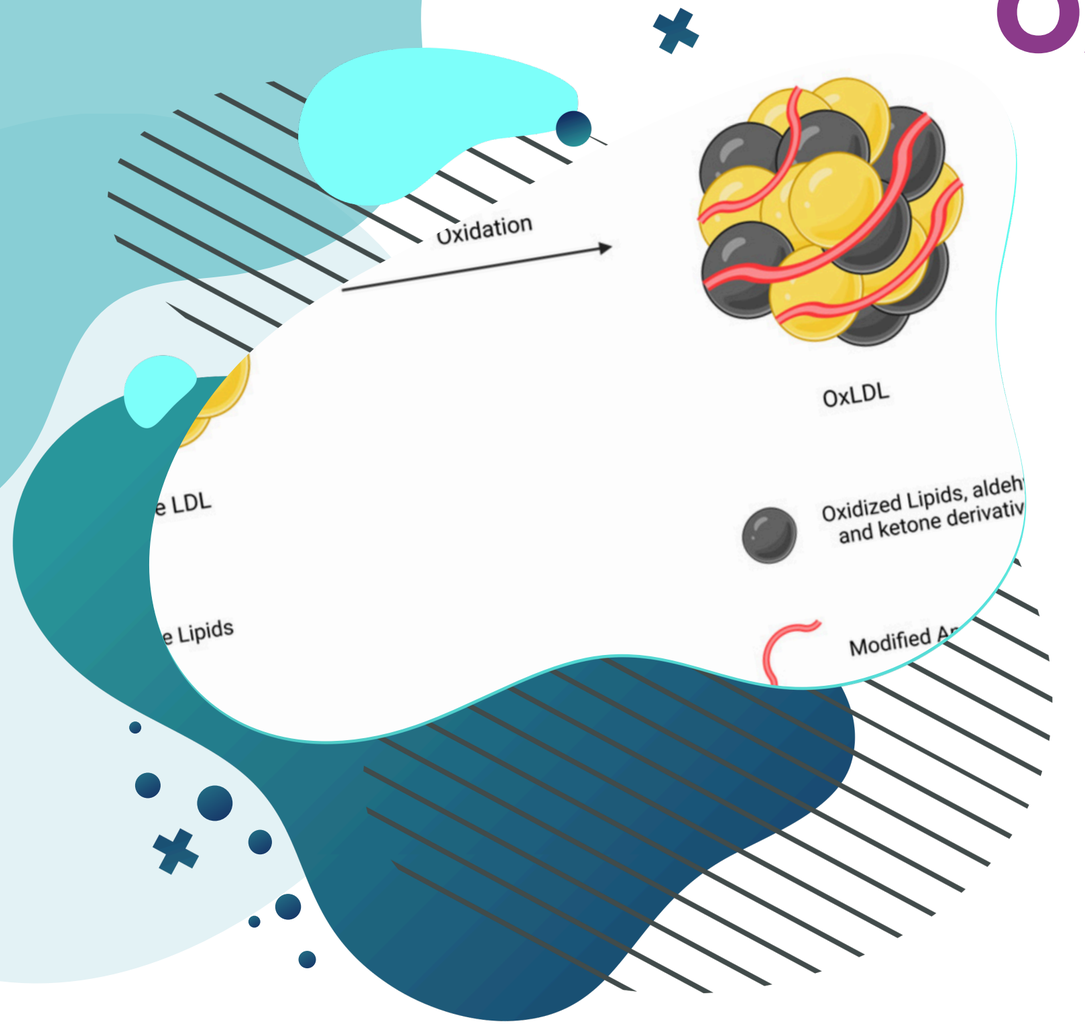
Oxidative Stress Panel



- ↑ 8-OHdG – DNA oxidation → cellular senescence
- ↑ MDA (Malondialdehyde) – lipid peroxidation → inflammatory signaling
- ↑ Nitrotyrosine – nitrative stress → mitochondrial dysfunction
- SOD2 SNP (rs4880 Ala16Val) – reduced mitochondrial resilience

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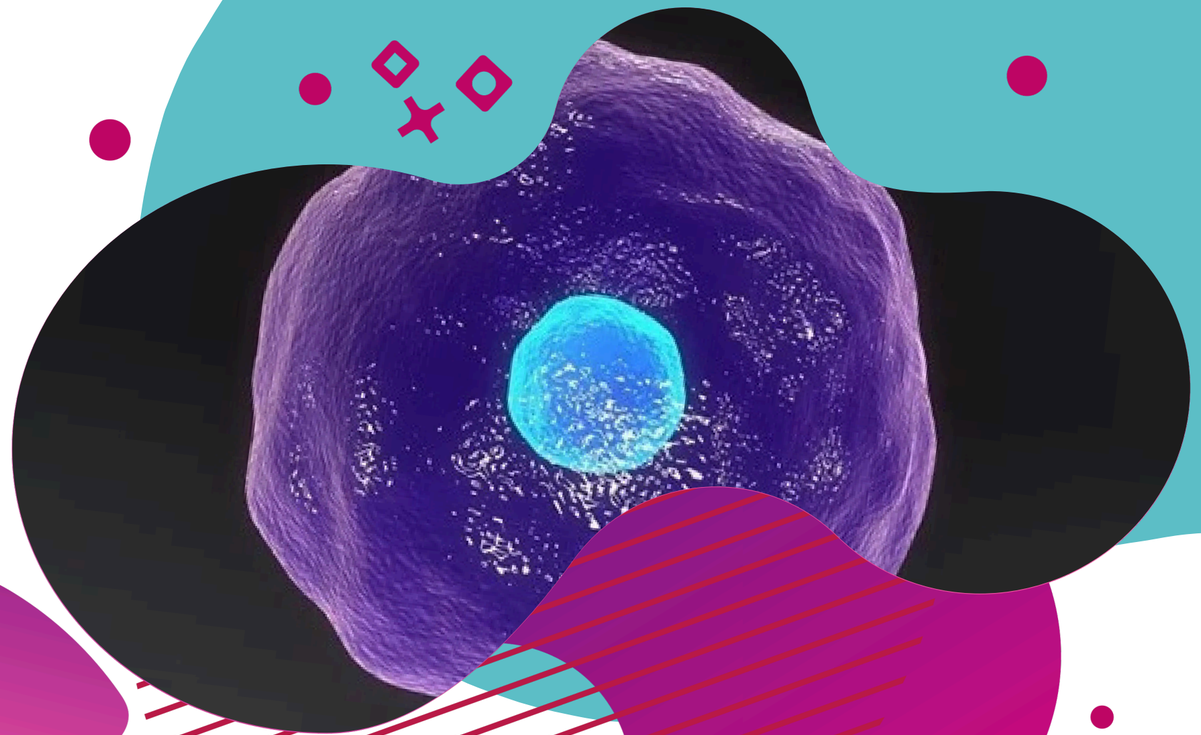
Cellular Cleanup Crew

- Autophagy clears damaged proteins, prevents SASP buildup
- Mitophagy removes dysfunctional mitochondria producing ROS
- Decline in both leads to accumulation of senescent cells



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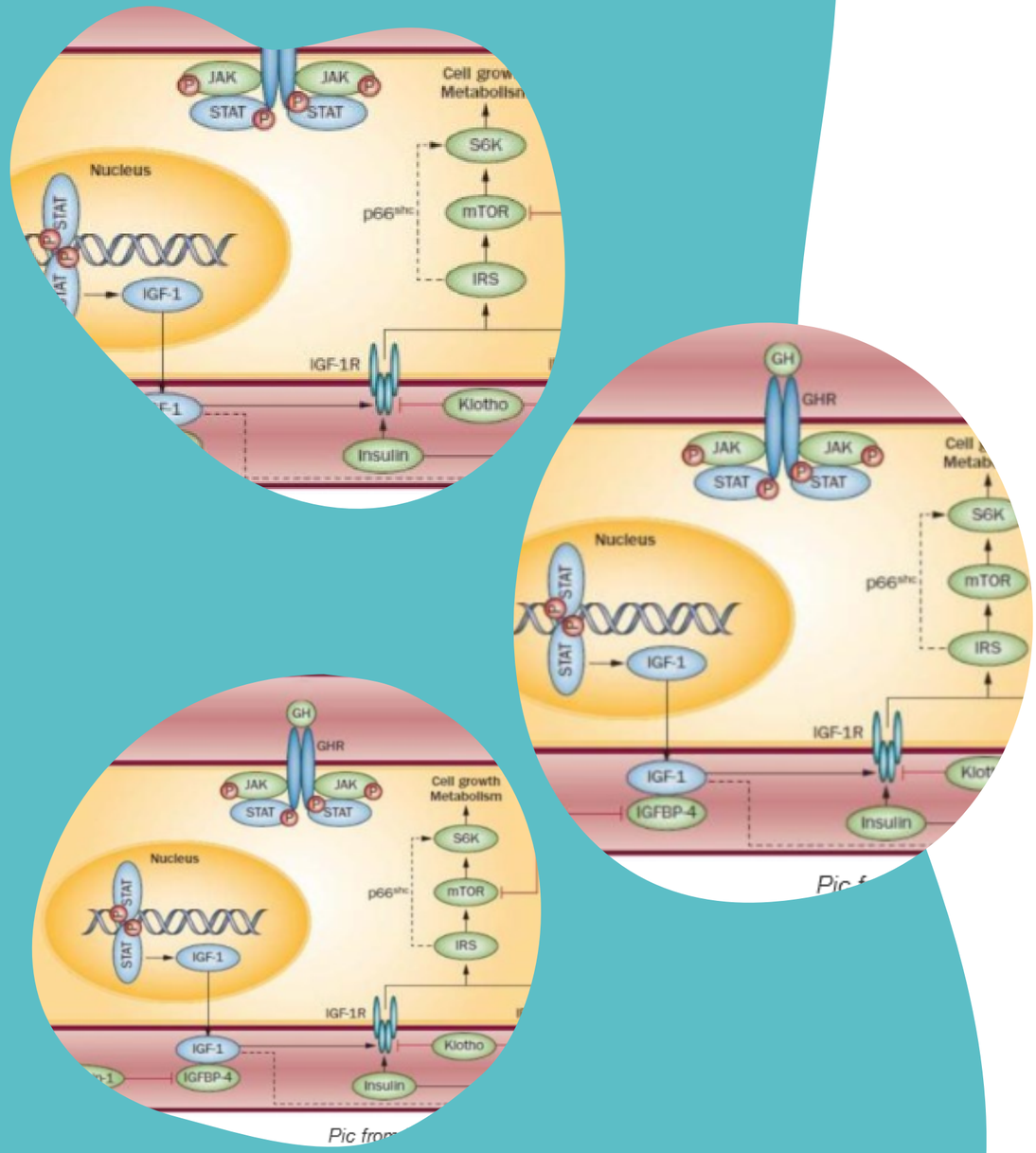

Anti-SASP Strategy: Dietary & Lifestyle Interventions

Daily (Baseline):

- 16:8 Time-Restricted Eating
  - → Eating window: 12:00 pm–8:00 pm
  - → Supports autophagy, glycemic control, and mitochondrial repair

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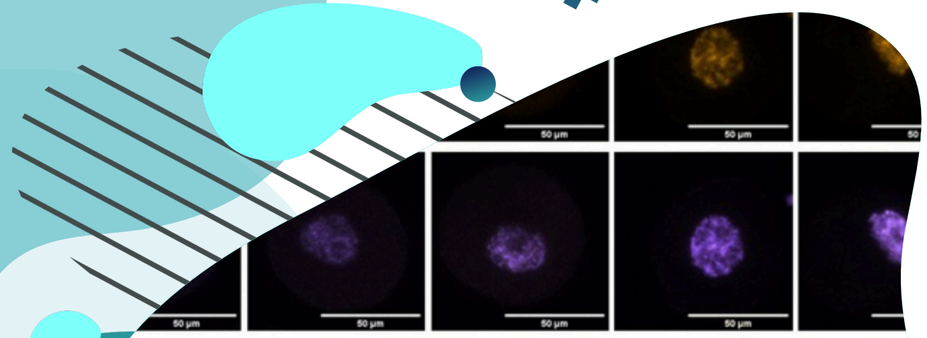

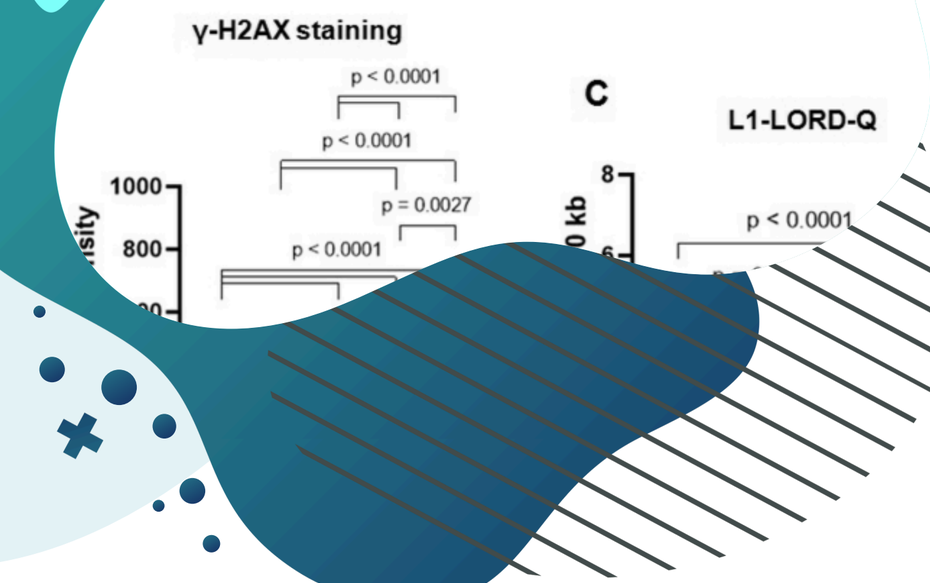

Anti-SASP Strategy: Dietary & Lifestyle Interventions

- Mediterranean-Paleo hybrid diet
  - → 75% plants (polyphenol-rich) + 25% protein
  - Healthy Fats to qs Calories
  - → Avoid seed oils, AGEs, ultra-processed foods

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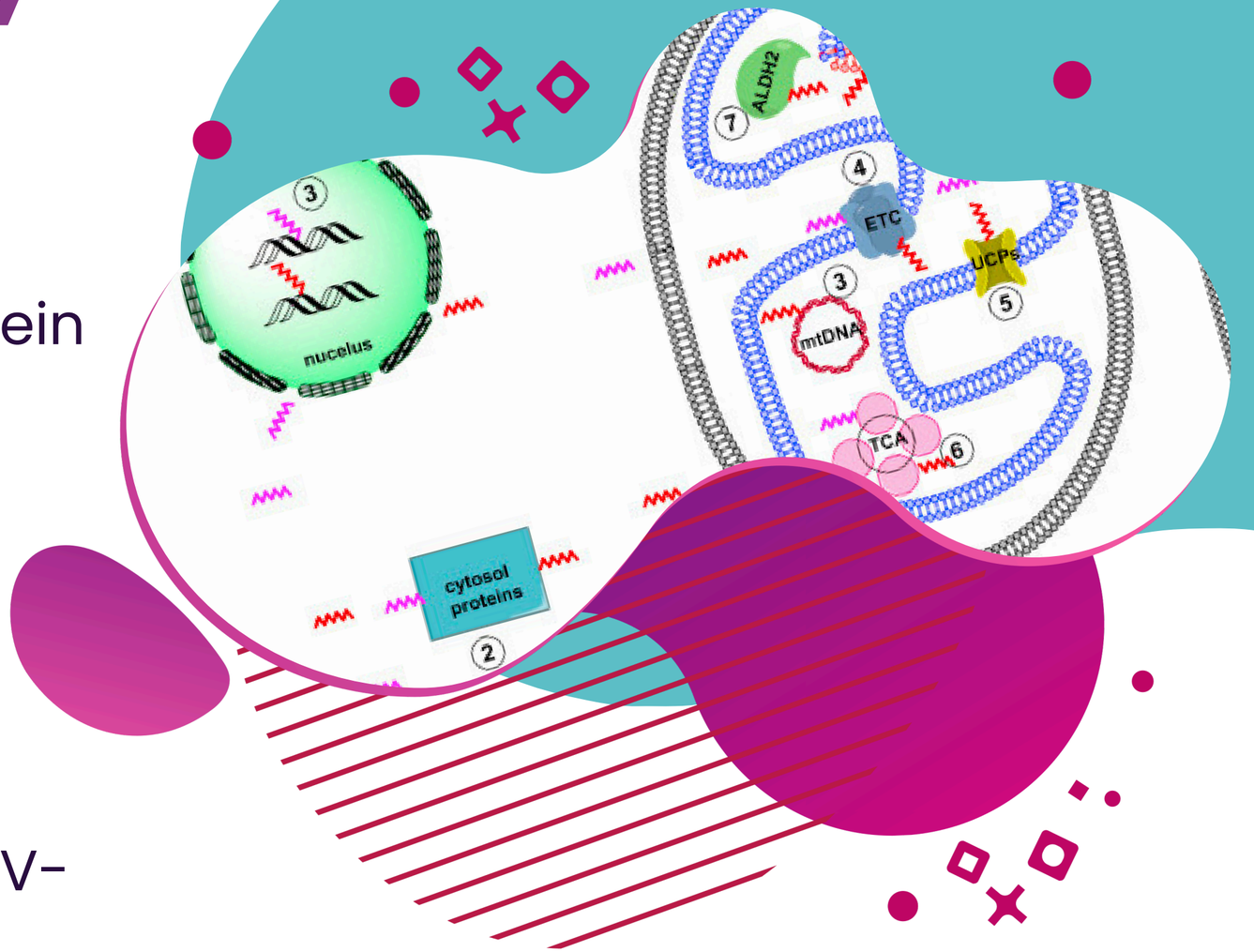
Anti-SASP Strategy: Dietary & Lifestyle Interventions

- Sauna 3x/week (heat shock protein induction)
- Cold plunge 2x/week (Nrf2 activation + mitochondrial hormesis)
- Zone 2 cardio: 45 min, 3x/week
- ReHIT 1 or 2 times per week
- Breathwork/Meditation daily (HRV-based training)



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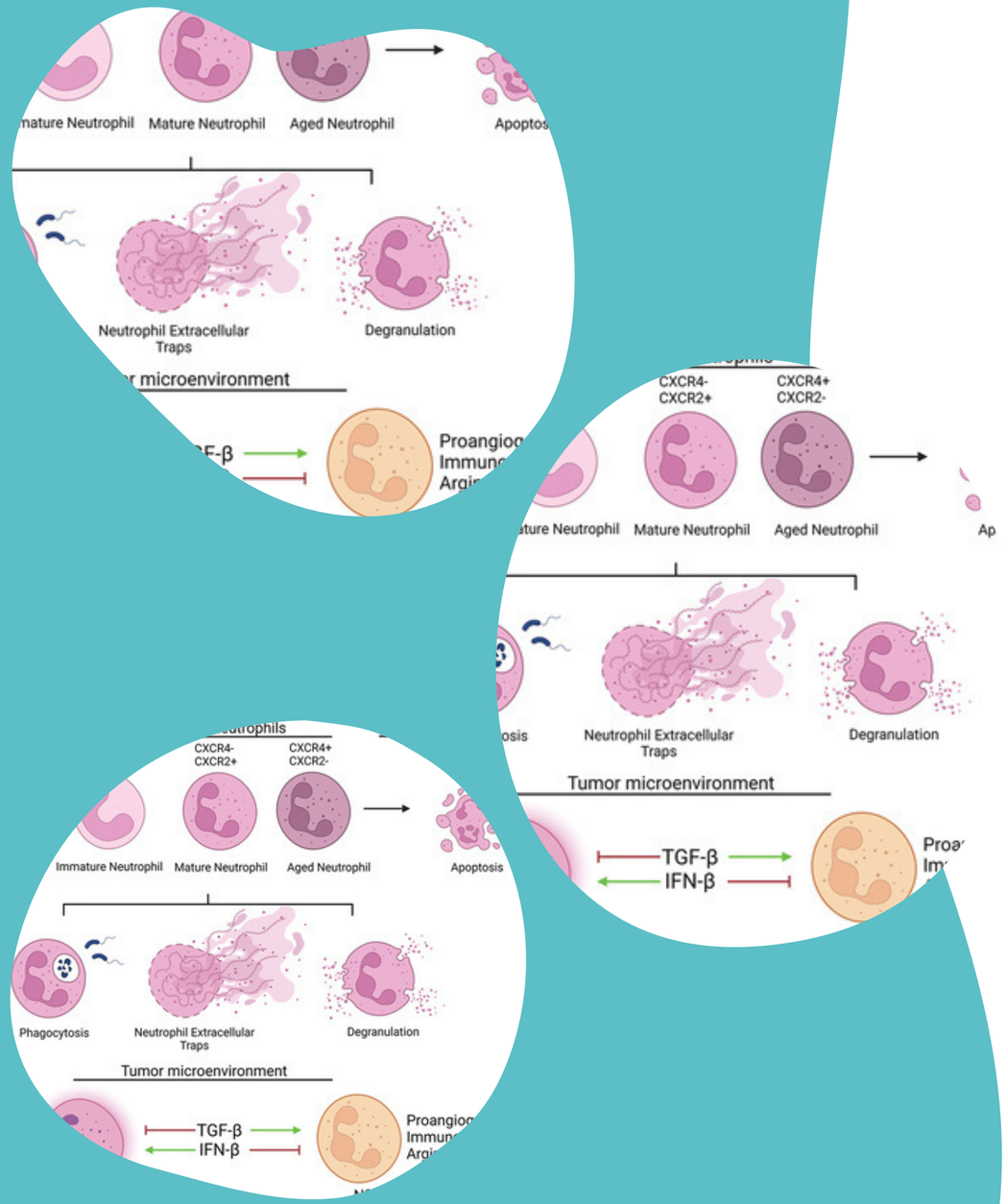

Anti-SASP Strategy: Dietary & Lifestyle Interventions

- Monthly:
- 3-Day Protein-Sparing Modified Fast (PSMF)
- keeping to his 16:8 TRE program
- → 1.5 g/kg lean mass protein only (no carbs/fats)
- → Water, electrolytes, and supervised clinical supplements only
- → Promotes autophagy, senescent cell clearance, and metabolic reset

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Targeted Supplements

Liposomal Glutathione – 500 mg/day

Why:

- *Master intracellular antioxidant*
- *Direct scavenger of ROS (reactive oxygen species).*
- *Senescent cells lead to*
  - *mitochondrial dysfunction*
  - *SASP activation.*

    Liposomal delivery enhances absorption, crosses cellular membranes and BBB.

    Goal: Improve detoxification, support mitochondrial efficiency, and reduce oxidative load contributing to SASP.

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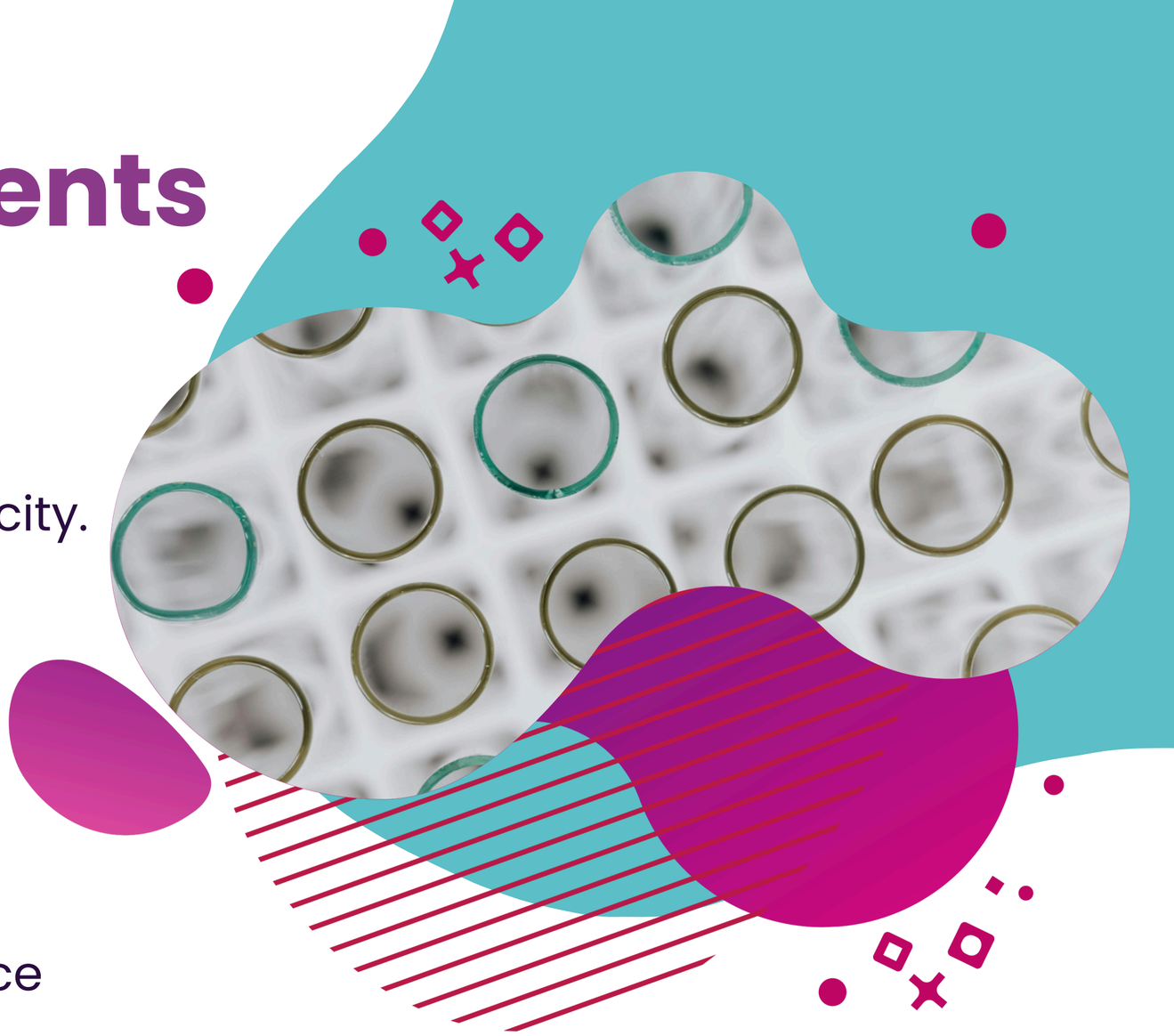
Targeted Supplements

N-Acetyl Cysteine (NAC) – 600 mg BID

Why:

- NAC is a precursor to glutathione
- Restore intracellular antioxidant capacity.
- Disrupts disulfide bonds in damaged proteins
- Regulates the redox-sensitive transcription factor NF-κB,
  - key regulator of SASP cytokines like IL-6 and TNF-α.

    Goal: Rebuild redox defenses, reduce SASP cytokine expression, and enhance glutathione recycling.



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Targeted Supplements

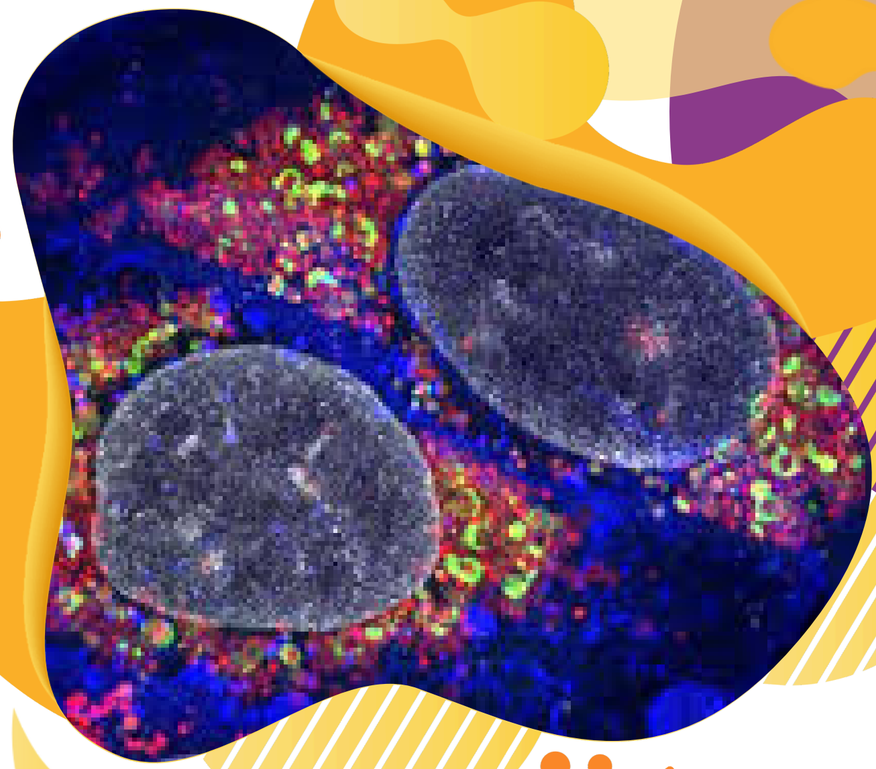
Curcumin (Phytosomal) – 500–1000 mg/day

Why:

Curcumin is a potent anti-inflammatory polyphenol that

- inhibits NF-κB,
- reduces COX-2 activity
- downregulates inflammatory prostaglandins elevated in SASP.
- Phytosomal formulation improves bioavailability dramatically.

  Goal: Suppress inflammatory pathways and prostaglandin-mediated signals associated with senescent cell secretions.



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Targeted Supplements

EGCG – 300 mg/day

Why:

Epigallocatechin gallate from green tea:

- Activates AMPK
- Downregulates mTOR
- Promotes autophagy.
- Inhibits:
  - Oxidative stress
  - Glycation
  - DNA damage

  —all contributors to the SASP profile.

  Goal: Promote senescent cell clearance, metabolic repair, and protection from lipid and protein oxidation.

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Targeted Supplements

- Resveratrol – 200 mg/day
- Why:
- Activates SIRT1
- Mimics caloric restriction
- Enhancing mitochondrial biogenesis
- and cellular stress resilience.
- Suppresses
  - SASP expression
  - Improves Nrf2 activation

  Goal: Extend healthspan by modulating aging-related pathways and decreasing the inflammatory burden of senescent cells.

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Targeted Supplements

Magnesium Glycinate – 300–400 mg/day

Why:

- ATP production
- DNA repair
- Cofactor for glutathione synthesis and detoxification pathways
- Often depleted in chronic stress states.

  Goal: Optimize mitochondrial output and antioxidant function; reduce DNA damage contributing to senescence signaling.

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Targeted Supplements

Berberine – 500 mg BID

Why:

- Metabolic adaptogen
- Activates AMPK
- Improves insulin sensitivity
- Mimics intermittent fasting effects
- Suppresses mTOR activity
- Promoting autophagy
- Reduces age-related metabolic rigidity.

  Goal: Tune metabolism, reduce insulin signaling stress, and create an environment less supportive of senescent cell survival.

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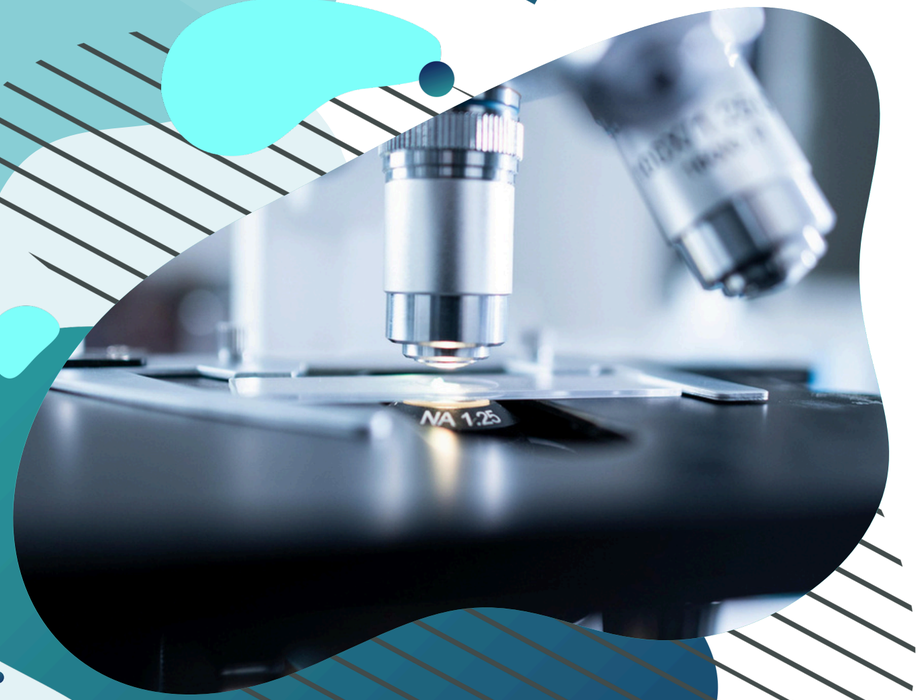
Senolytic Pulse (Once Monthly)



Fisetin – 1500 mg/day × 2 days
Quercetin – 1000 mg/day × 2 days
Why:

- Blavonoid-based senolytics
- Selectively inducing apoptosis
  - in senescent cells
  - sparing healthy cells.
- Fisetin, in particular, has shown broad-spectrum senolytic effects in animal and human models.

  Goal: Actively reduce senescent cell burden and blunt the pro-inflammatory SASP phenotype.

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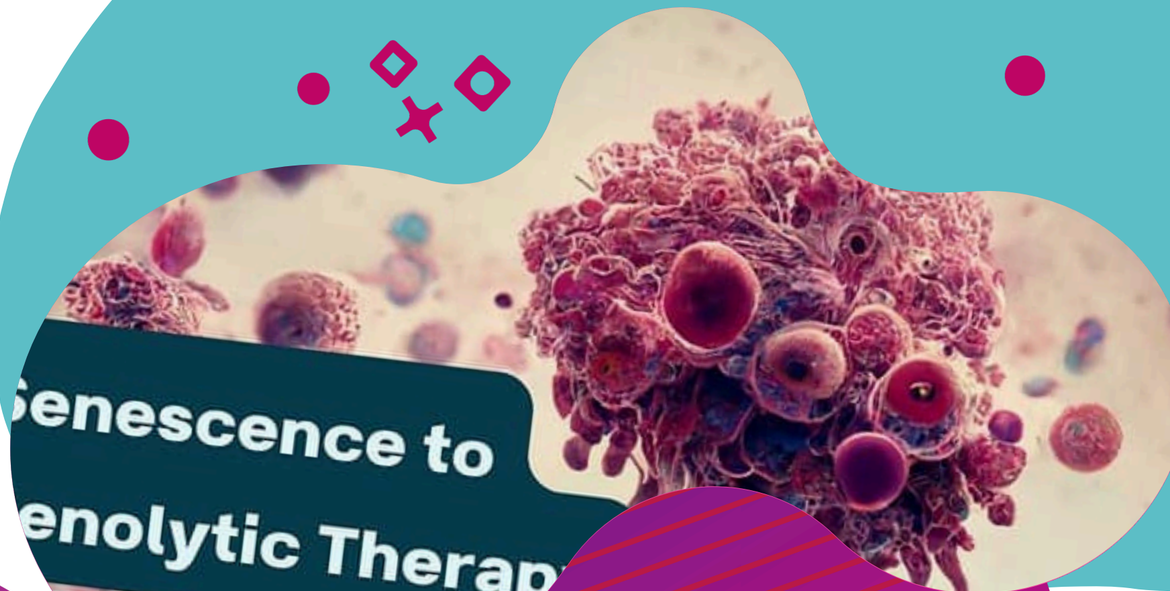
Follow Up

One-Year Follow-Up

- 3 Months ↓ hs-CRP to 1.8, ↓ ox-LDL by 20% Improved HRV, better sleep
- 6 Months ↓ MDA, Nitrotyrosine, improved ferritin. More energy, sharper focus
- 9 Months Normalized 8-OHdG, ↑ adiponectin Leaner body comp, enhanced stamina



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Jackson R.

49 year old

12 Months All labs normalized Patient reports “calm intensity,” Reclassified as SASP-low-risk

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Reversing Biological Age

- You can’t fix what you don’t measure
- Zombie cells are real and targetable
- Precision Age Management = terrain before treatment

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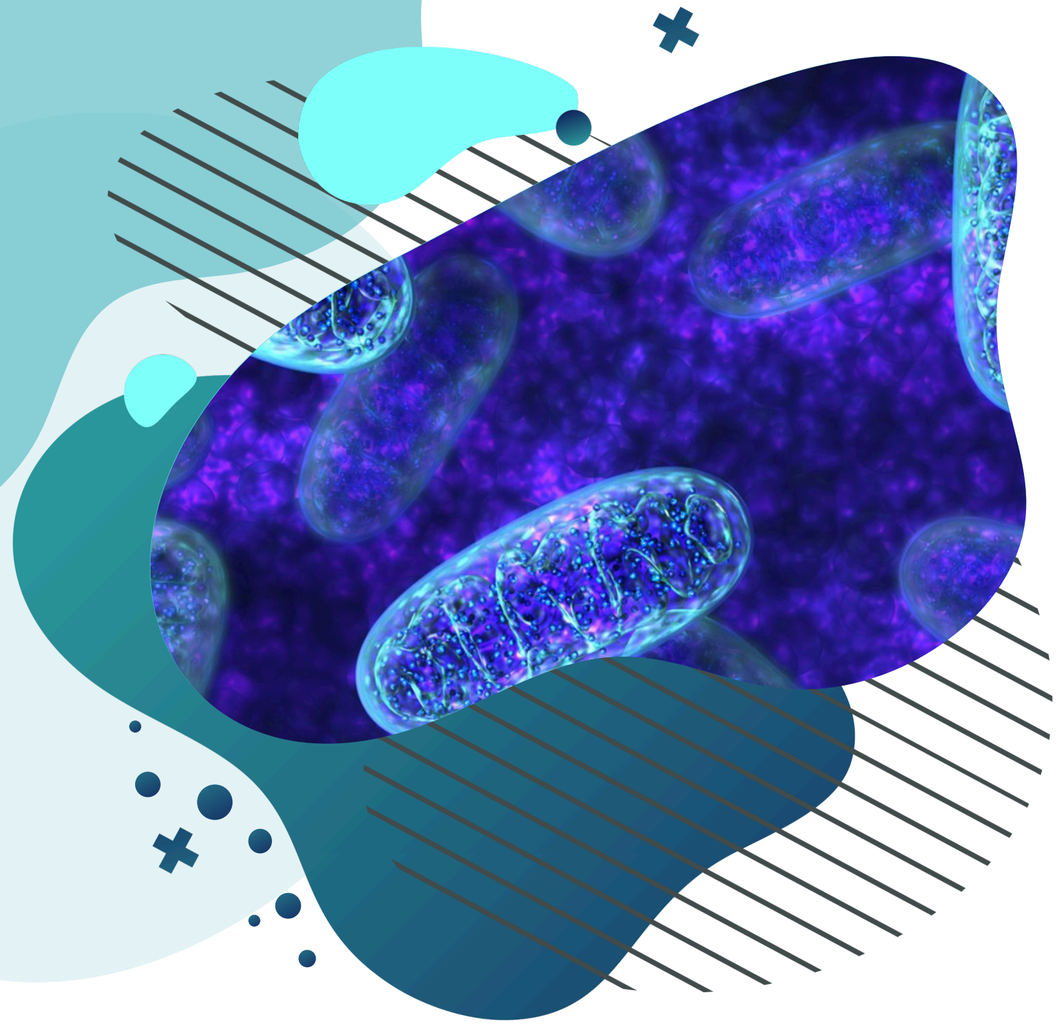

What If Aging Is Just Poor Maintenance?

- Clean the trash (autophagy)
- Fix the wiring (mitochondria)
- Restore the flow (terrain)

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Lets Kill Some Zombies

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Thank You!

- Questions?
- Connect after the session

Longevity Foundations

Redefining
Oxidative Stress
and Cellular Health

Session 3

Dr. Marcos de Andrade, MD

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Historical Pivot in Modern Medicine

Dr. Marcos de Andrade, MD

Meet Your Speaker

Dr. Marcos de Andrade, *MD*

Dr. De has dedicated his life to exploring human potential, earning degrees in Biology, Medicine, and Business, along with a research fellowship at the Cleveland Clinic. He and the Biohax medical team are recognized for advancing medicine through innovative research with impactful results.

Inspired by his own recovery from a serious illness through biohacking, Dr. De founded and self-funded Biohax. Today, he continues to grow the brand globally through speaking engagements and education on epigenetics, performance, longevity, hormone health, heart disease, and more.

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Historical Pivot in Modern Medicine

Modern medicine saves lives, but we started chasing quick fixes and ignoring root causes. How Ehrlich’s and Flexner’s work influenced modern medicine.

Strebhardt K, Ullrich A. Paul Ehrlich's magic bullet concept: 100 years of progress. Nat Rev Cancer. 2008 Jun;8(6):473-80. doi: 10.1038/nrc2394. Epub 2008 May 12. PMID: 18469827.
https://pubmed.ncbi.nlm.nih.gov/18469827/ Duffy TP. The Flexner Report--100 years later. Yale J Biol Med. 2011 Sep;84(3):269-76. PMID: 21966046; PMCID: PMC3178858. https://pmc.ncbi.nlm.nih.gov/articles/PMC3178858/

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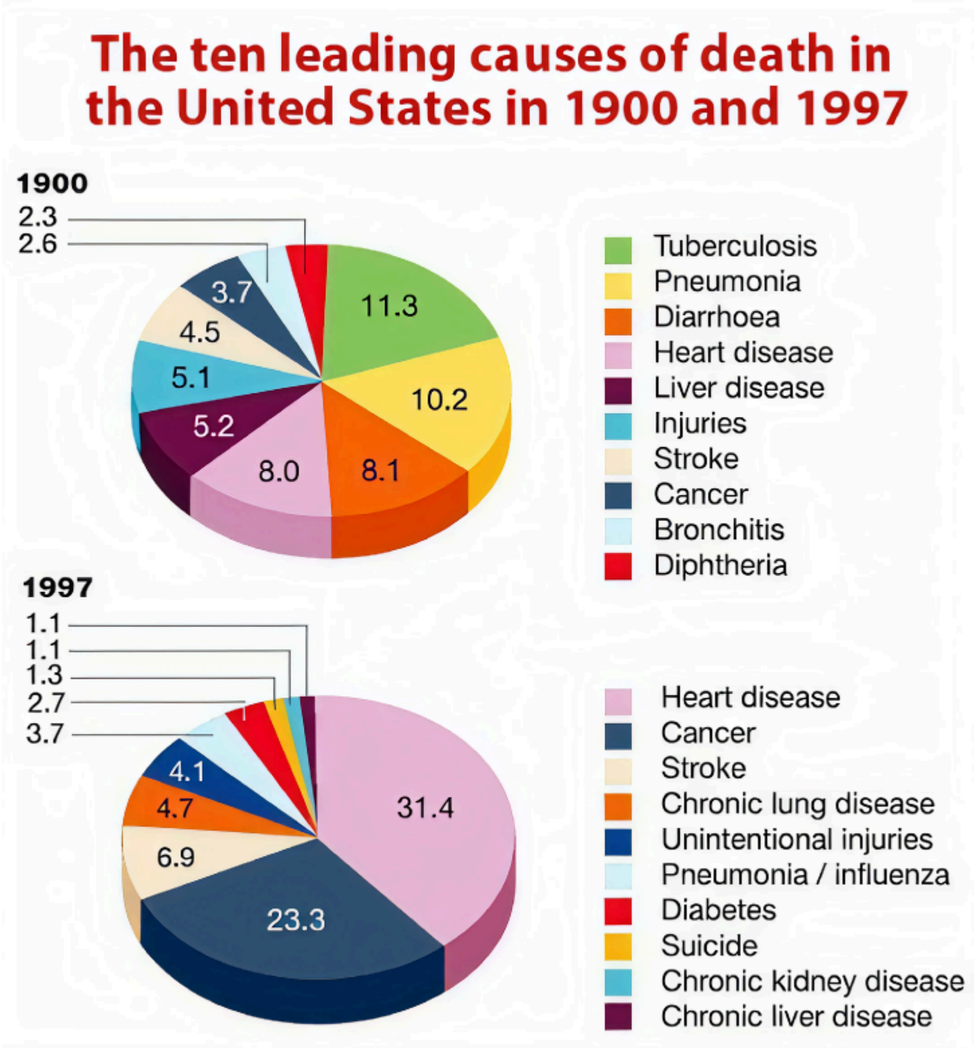
Chronic disease burden is now the problem

Most deaths and disability now come from long-term conditions driven by daily life.



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The Blueprint: Bioidentical Design

The human body runs native ligands, real food, daily movement, steady sleep, morning light, and supportive relationships.

Holtorf K. The bioidentical hormone debate: are bioidentical hormones (estradiol, estriol, and progesterone) safer or more efficacious than commonly used synthetic versions in hormone replacement therapy? Postgrad Med. 2009 Jan;121(1):73-85. doi: 10.3810/pgm.2009.01.1949. PMID: 19179815. https://pubmed.ncbi.nlm.nih.gov/19179815/

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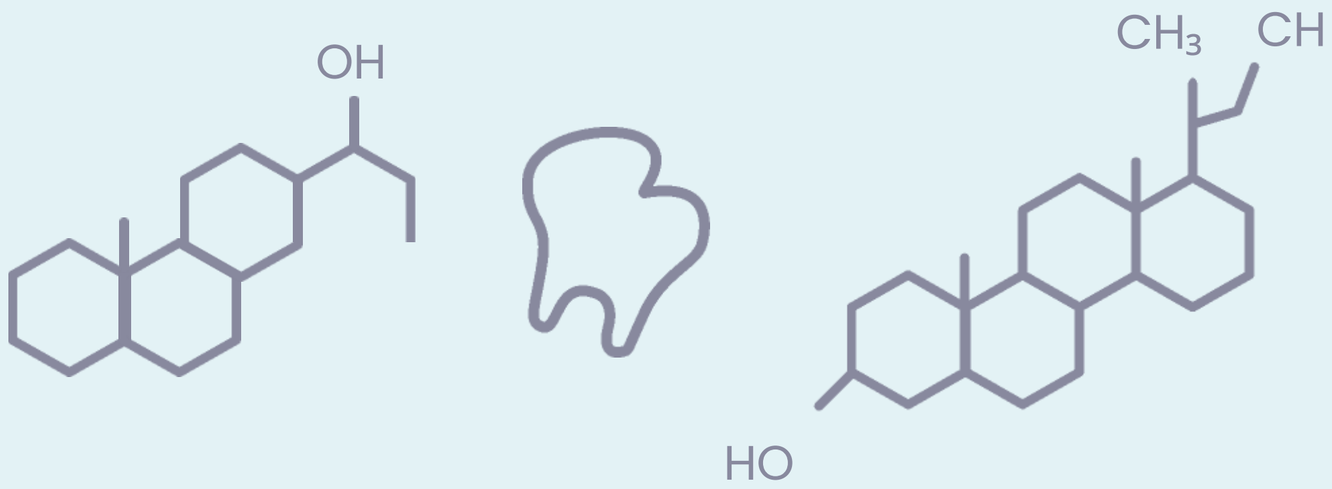

Estradiol

Ethinyl Estradiol

The Betrayal: Synthetic Substitution

Synthetic drugs/ligands aren't identical, use the version and route that fit the body best.

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Bio-Identical Vs. Synthetic

Small changes can alter fit, behavior, and risk, choose the right molecule for the job

Holtorf K. The bioidentical hormone debate: are bioidentical hormones (estradiol, estriol, and progesterone) safer or more efficacious than commonly used synthetic versions in hormone replacement therapy? Postgrad Med. 2009 Jan;121(1):73-85. doi: 10.3810/pgm.2009.01.1949. PMID: 19179815. https://pubmed.ncbi.nlm.nih.gov/19179815/

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Immune reaction to foreign inputs

Brief immune activation protect you;
constant ones wear you down.
First, quiet the constant ones.

Chen L, Deng H, Cui H, Fang J, Zuo Z, Deng J, Li Y, Wang X, Zhao L. Inflammatory responses and inflammation-associated diseases in organs. Oncotarget. 2017 Dec 14;9(6):7204-7218. doi: 10.18632/oncotarget.23208. PMID: 29467962; PMCID: PMC5805548. https://pmc.ncbi.nlm.nih.gov/articles/PMC5805548/

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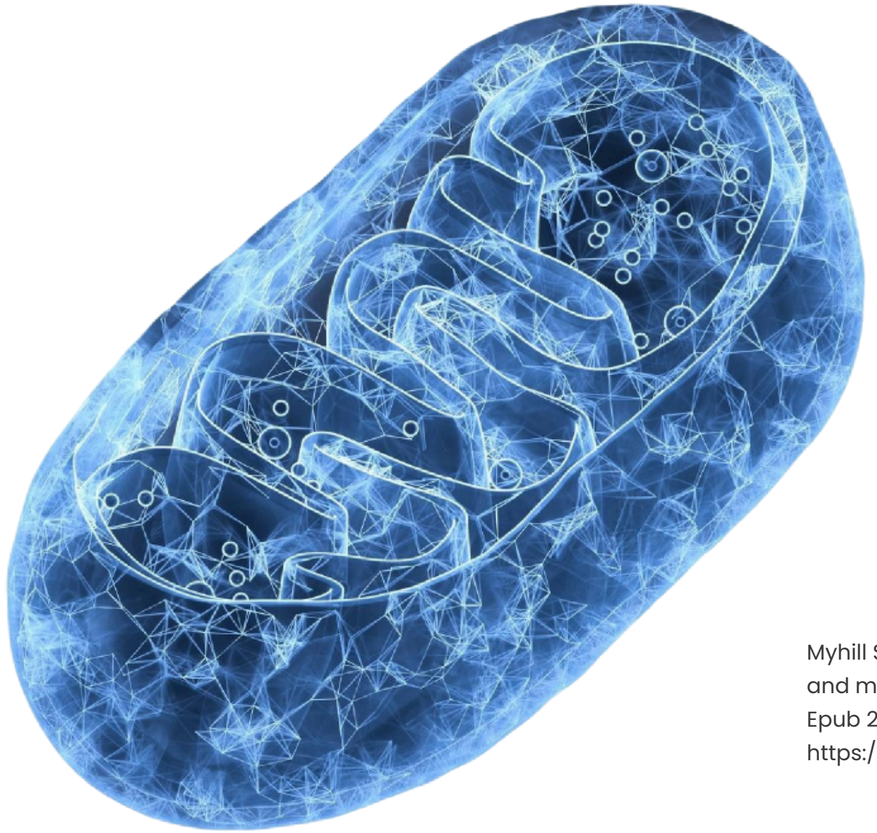
Mitochondrial Impact

Daily habits can slow these energy factories. Restore basics first, then consider add-ons.



Myhill S, Booth NE, McLaren-Howard J. Chronic fatigue syndrome and mitochondrial dysfunction. Int J Clin Exp Med. 2009;2(1):1-16. Epub 2009 Jan 15. PMID: 19436827; PMCID: PMC2680051. https://pmc.ncbi.nlm.nih.gov/articles/PMC2680051/

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Mitochondrial Therapies

Lifestyle first; consider NAD related compounds, targeted peptides, or HBOT case-by-case.

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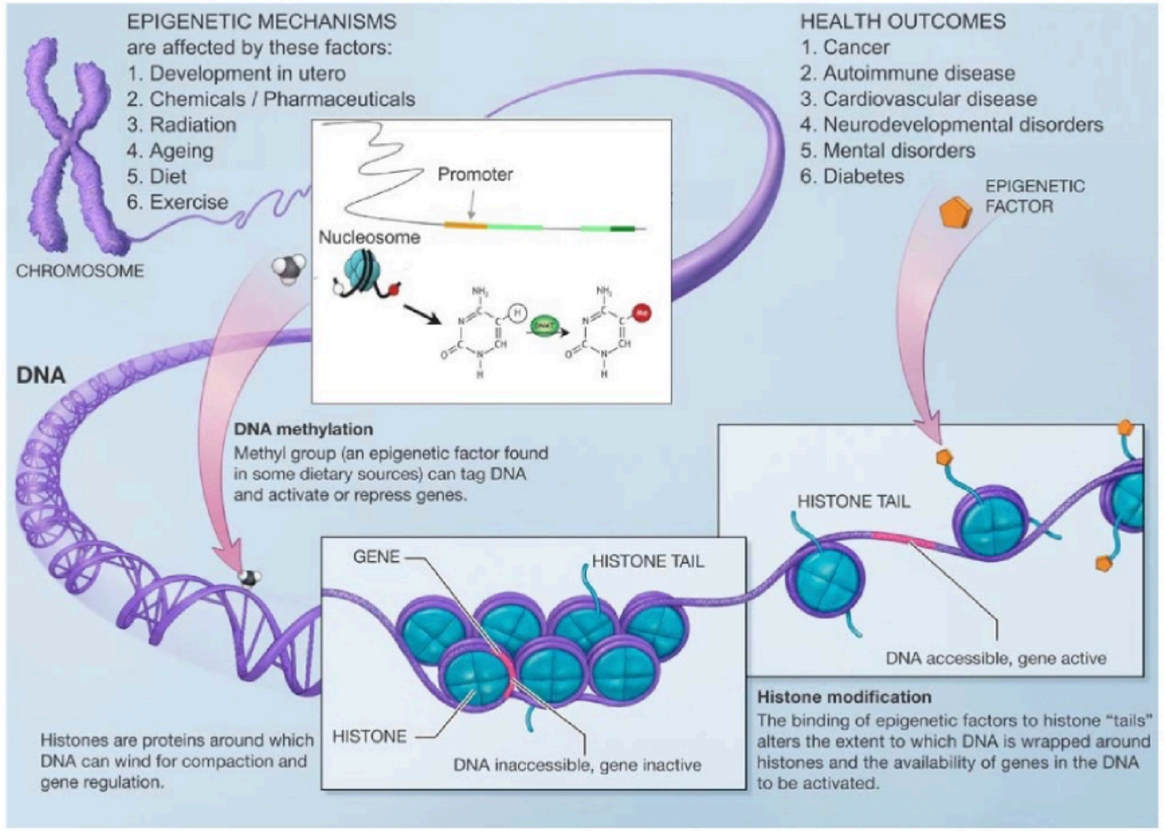
Epigenetics: DNA Methylation and histone acetylation

Small tags on DNA can turn genes on or off. Food, sleep, movement, and stress shift these tags. Open DNA is easier to read; while closed DNA is not, daily habits nudge this open–close switch.



Zhang L, Lu Q, Chang C. Epigenetics in Health and Disease. Adv Exp Med Biol. 2020;1253:3-55. doi: 10.1007/978-981-15-3449-2_1. PMID: 32445090. https://pubmed.ncbi.nlm.nih.gov/32445090/

Bošković A, Rando OJ. Transgenerational Epigenetic Inheritance. Annu Rev Genet. 2018 Nov 23;52:21-41. doi: 10.1146/annurev-genet-120417-031404. Epub 2018 Aug 30. PMID: 30160987. https://pubmed.ncbi.nlm.nih.gov/30160987/

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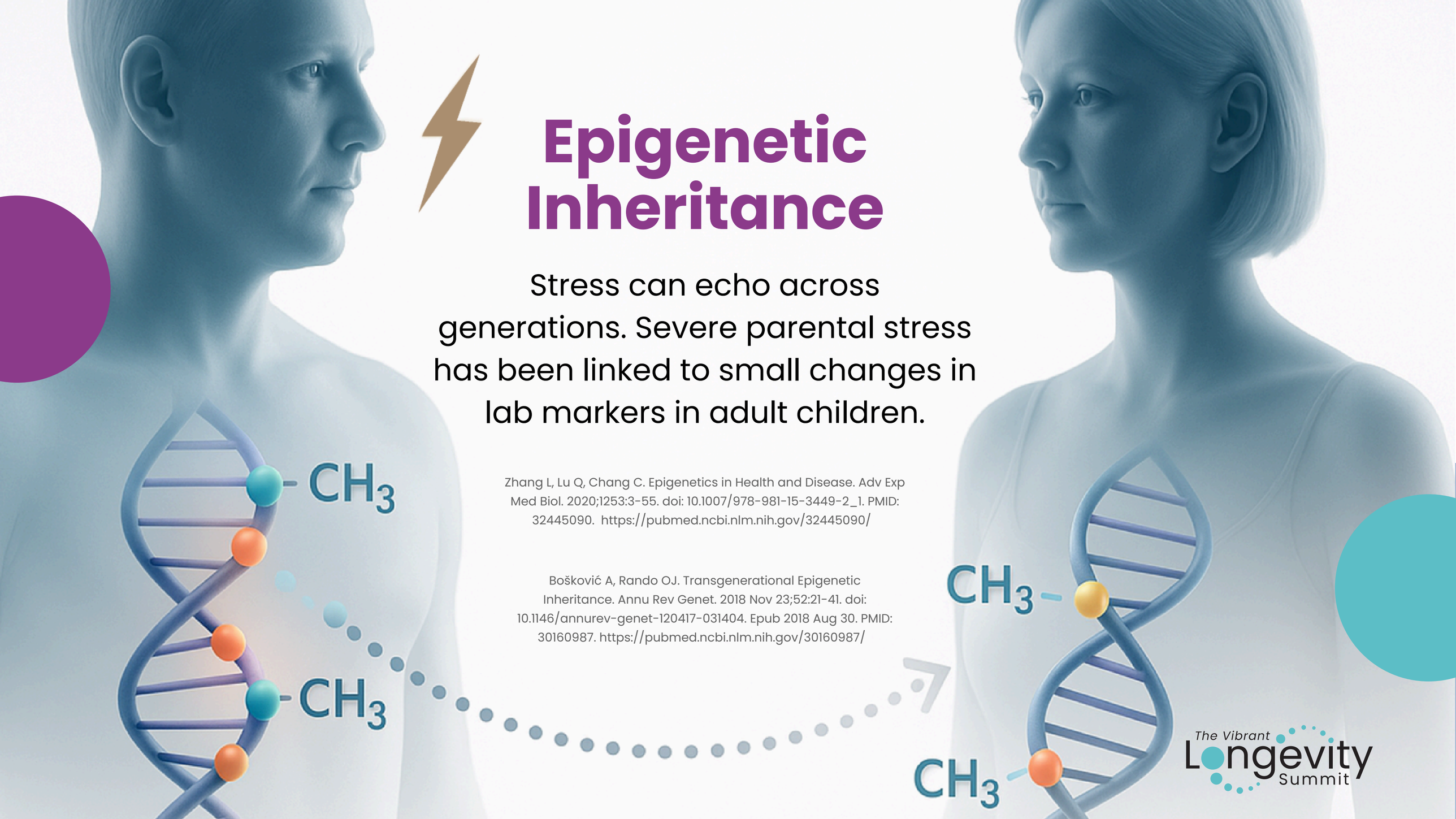
Epigenetic Inheritance

Stress can echo across generations. Severe parental stress has been linked to small changes in lab markers in adult children.

Zhang L, Lu Q, Chang C. Epigenetics in Health and Disease. Adv Exp Med Biol. 2020;1253:3-55. doi: 10.1007/978-981-15-3449-2_1. PMID: 32445090. https://pubmed.ncbi.nlm.nih.gov/32445090/

Bošković A, Rando OJ. Transgenerational Epigenetic Inheritance. Annu Rev Genet. 2018 Nov 23;52:21-41. doi: 10.1146/annurev-genet-120417-031404. Epub 2018 Aug 30. PMID: 30160987. https://pubmed.ncbi.nlm.nih.gov/30160987/



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Biological Age Clocks

Pick the right clock, and watch for trends, not isolated numbers.

Ho KM, Morgan DJ, Johnstone M, Edibam C. Biological age is superior to chronological age in predicting hospital mortality of the critically ill. Intern Emer g Med. 2023 Oct;18(7):2019-2028. doi: 10.1007/s11739-023-03397-3. Epub 2023 Aug 28. PMID: 37635161; PMCID: PMC10543822. https://pmc.ncbi.nlm.nih.gov/articles/PMC10543822/

Levine ME, Lu AT, Quach A, Chen BH, Assimes TL, Bandinelli S, Hou L, Baccarelli AA, Stewart JD, Li Y, Whitsel EA, Wilson JG, Reiner AP, Aviv A, Lohman K, Liu Y, Ferrucci L, Horvath S. An epigenetic biomarker of aging for lifespan and healthspan. Aging (Albany NY). 2018 Apr 18;10(4):573-591. doi: 10.18632/aging.101414. PMID: 29676998; PMCID: PMC5940111. https://pmc.ncbi.nlm.nih.gov/articles/PMC5940111/

Belsky DW, Caspi A, Corcoran DL, Sugden K, Poulton R, Arseneault L, Baccarelli A, Chamarti K, Gao X, Hannon E, Harrington HL, Houts R, Kothari M, Kwon D, Mill J, Schwartz J, Vokonas P, Wang C, Williams BS, Moffitt TE. DunedinPACE, a DNA methylation biomarker of the pace of aging. Elife. 2022 Jan 14;11:e73420. doi: 10.7554/eLife.73420. PMID: 35029144; PMCID: PMC8853656. https://pmc.ncbi.nlm.nih.gov/articles/PMC8853656/

Lu AT, Quach A, Wilson JG, Reiner AP, Aviv A, Raj K, Hou L, Baccarelli AA, Li Y, Stewart JD, Whitsel EA, Assimes TL, Ferrucci L, Horvath S. DNA methylation GrimAge strongly predicts lifespan and healthspan. Aging (Albany NY). 2019 Jan 21;11(2):303-327. doi: 10.18632/aging.101684. PMID: 30669119; PMCID: PMC6366976. https://pmc.ncbi.nlm.nih.gov/articles/PMC6366976/

Horvath S. An epigenetic biomarker of aging for lifespan and healthspan. Aging (Albany NY). 2018 Apr 18;10(4):573-591. doi: 10.18632/aging.101414. PMID: 29676998; PMCID: PMC5940111. https://pmc.ncbi.nlm.nih.gov/articles/PMC5940111/

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Biological age reversal

A short program nudged a lab measure of aging in the right direction, within weeks.

Ho KM, Morgan DJ, Johnstone M, Edibam C. Biological age is superior to chronological age in predicting hospital mortality of the critically ill. Intern Emer g Med. 2023 Oct;18(7):2019-2028. doi: 10.1007/s11739-023-03397-3. Epub 2023 Aug 28. PMID: 37635161; PMCID: PMC10543822.
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Levine ME, Lu AT, Quach A, Chen BH, Assimes TL, Bandinelli S, Hou L, Baccarelli AA,
Stewart JD, Li Y, Whitsel EA, Wilson JG, Reiner AP, Aviv A, Lohman K, Liu Y, Ferrucci L,

Horvath S. An epigenetic biomarker of aging for lifespan and healthspan. Aging (Albany NY). 2018 Apr 18;10(4):573-591. doi: 10.18632/aging.101414. PMID: 29676998; PMCID: PMC5940111.
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Belsky DW, Caspi A, Corcoran DL, Sugden K, Poulton R, Arseneault L, Baccarelli A, Chamarti K, Gao X, Hannon E, Harrington HL, Houts R, Kothari M, Kwon D, Mill J, Schwartz J, Vokonas P, Wang C, Williams BS, Moffitt TE. DunedinPACE, a DNA methylation biomarker of the pace of aging. Elife. 2022 Jan 14;11:e73420. doi: 10.7554/eLife.73420. PMID: 35029144; PMCID: PMC8853656.
https://pmc.ncbi.nlm.nih.gov/articles/PMC8853656/

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https://pmc.ncbi.nlm.nih.gov/articles/PMC6366976/

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Food quality at the source

Some soils and crops lost nutrients, source better, and test when it matters.

Davis DR, Epp MD, Riordan HD. Changes in USDA food composition data for 43 garden crops, 1950 to 1999. J Am Coll Nutr. 2004 Dec;23(6):669-82. doi: 10.1080/07315724.2004.10719409. PMID: 15637215. https://pubmed.ncbi.nlm.nih.gov/15637215/

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Dietary pattern and risk

Ultra-processed patterns raise risk, choose whole foods, fiber, protein, and better fat balance.

Rico-Campà A, Martínez-González MA, Alvarez-Alvarez I, Mendonça RD, de la Fuente-Arrillaga C, Gómez-Donoso C, Bes-Rastrollo M. Association between consumption of ultra-processed foods and all cause mortality: SUN prospective cohort study. BMJ. 2019 May 29;365:l1949. doi: 10.1136/bmj.l1949. PMID: 31142450; PMCID: PMC6538973. https://pubmed.ncbi.nlm.nih.gov/31142450/

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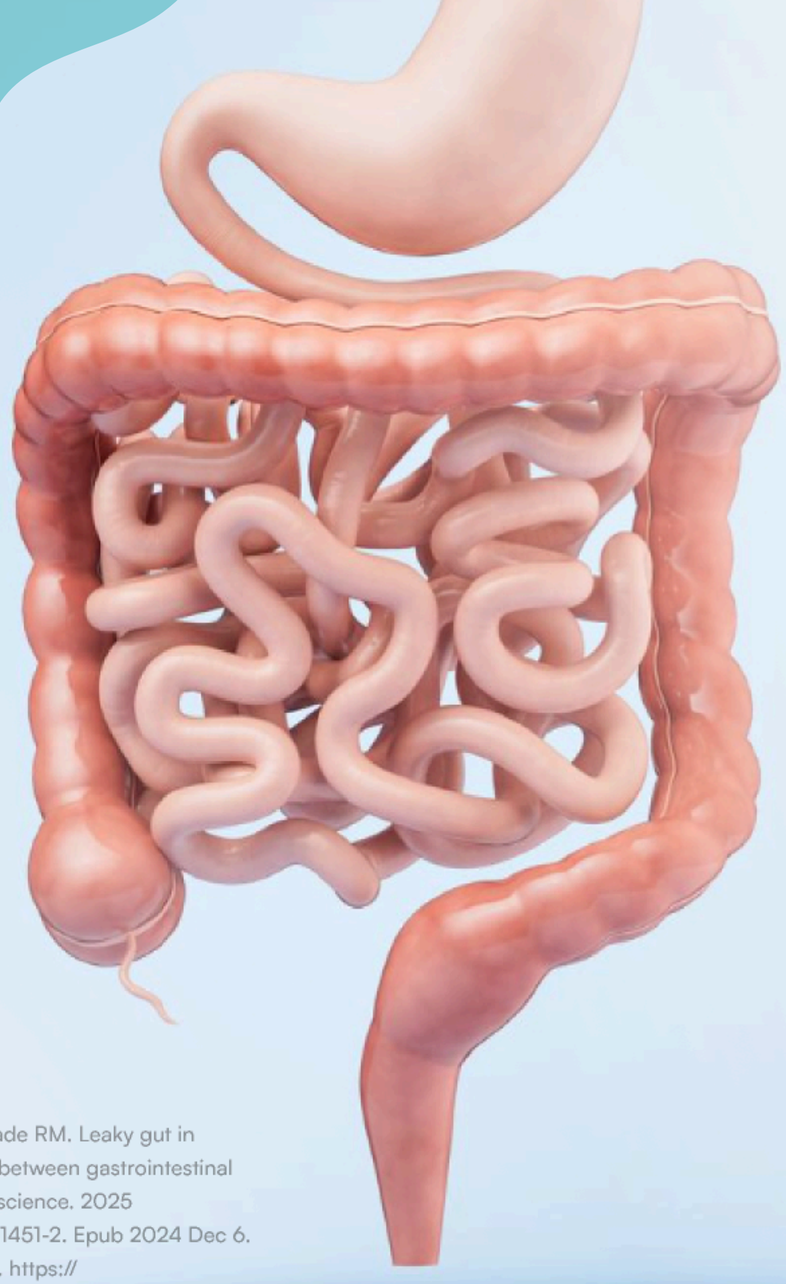

Gut to Immune Link

An irritated gut lets more byproducts slip into blood, calm the gut to calm the body.

Escalante J, Artaiz O, Diwakarla S, McQuade RM. Leaky gut in systemic inflammation: exploring the link between gastrointestinal disorders and age-related diseases. Geroscience. 2025 Feb;47(1):1-22. doi: 10.1007/s11357-024-01451-2. Epub 2024 Dec 6. PMID: 39638978; PMCID: PMC11872833. https://pmc.ncbi.nlm.nih.gov/articles/PMC11872833/

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Hormones, chosen wisely

Match the hormone and delivery to the goal, adjust according to risks.

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Spiritual inputs and physiology

Spiritual practices can calm your system and improve HRV, sleep, and mood.

Li S, Stampfer MJ, Williams DR, VanderWeele TJ. Association of Religious Service Attendance With Mortality Among Women. JAMA Intern Med. 2016 Jun 1;176(6):777-85. doi: 10.1001/jamainternmed.2016.1615. PMID: 27183175; PMCID: PMC5503841. https://pmc.ncbi.nlm.nih.gov/articles/PMC11872833/

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Faith meets science

Daily choices add up, act on them and measure progress.

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Li S, Stampfer MJ, Williams DR, VanderWeele TJ. Association of Religious Service Attendance With Mortality Among Women. JAMA Intern Med. 2016 Jun 1;176(6):777-85. doi: 10.1001/jamainternmed.2016.1615. PMID: 27183175; PMCID: PMC5503841. https://pmc.ncbi.nlm.nih.gov/articles/PMC11872833/

Return to the blueprint

Basics first; test, tailor, and measure, then add targeted therapy.

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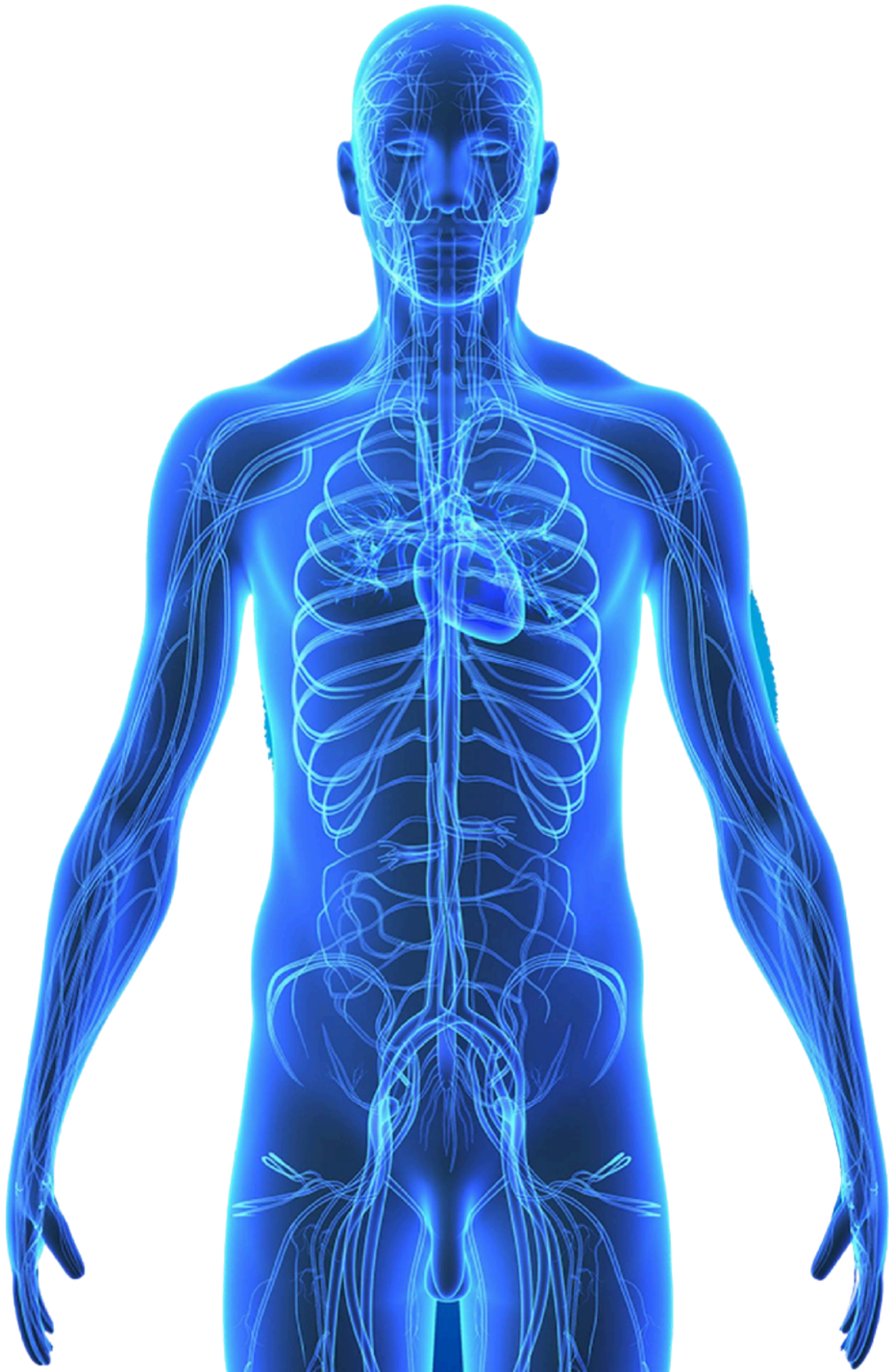
Real-life Example: Biohax

Start with basics, measure, adjust, then add targeted therapy if needed.



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Biohax protocol overview

Assess → Interpret → Act → Measure

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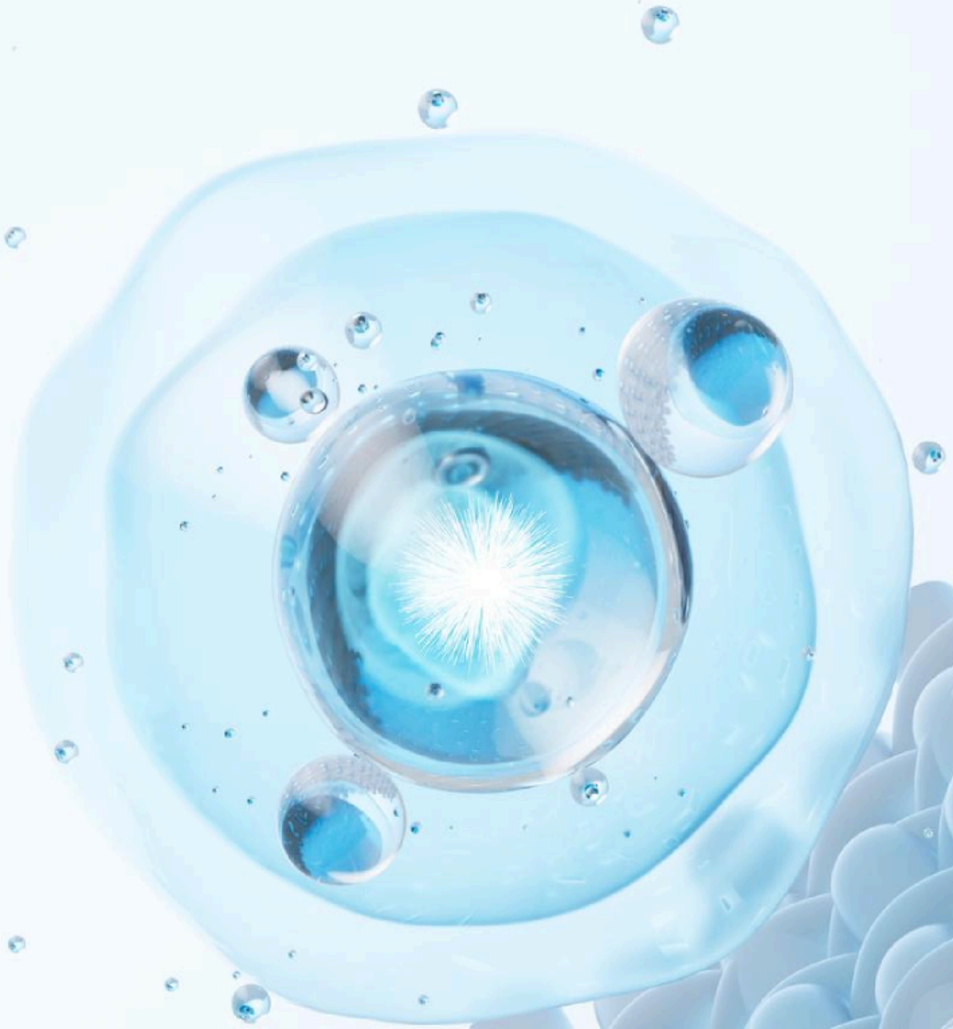
Regeneration vs replacement

If a system can heal, support healing; replace only when recovery won't happen in time.



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What change looks like

- Lower inflammation
- Better insulin sensitivity,
- Higher fitness
- Age markers nearer real age

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Better tools, used wisely

Use wearables for daily signals and age clocks for trends, but also look beyond data.

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Role of AI in care

Use AI to save time and spot patterns, keep people in charge of outcomes.

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Your Invitation

Pick one habit, one test and one date.
Put them on your calendar.

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Closing Vision

Health was not lost. It was forgotten.
We can remember it together.

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Longevity Foundations

Redefining Oxidative Stress and Cellular Health

Session 4

Dr. Jay Goodbinder, ND, DC, DABCI

Dr. Jay Goodbinder
ND, DC, DABCI

Owner and Lead Clinician of the Epigenetics Healing Center

Board Certified Chiropractic Internist (Focus on Endocrinology, Biochemistry, Physiology, and Immunology)

Naturopathic Physician

Best Selling Author (Defending Your Life)

Epigenetics research (1 year)

Owner of Lifesupport Health Products

Creator of “The Passion for Patients Weekend” for Doctors and other clinicians to wow patients with clinical excellence

International Speaker (Tokyo, Bahrain, Dubai, London, Bangkok, Paris, Mexico City, Milan)

Shared stage with Suzanne Somers, Dr. Oz, Dr. Drew

Presented at: New York Academy of Medicine, Harvard Faculty Club, CNN, and Nasdaq

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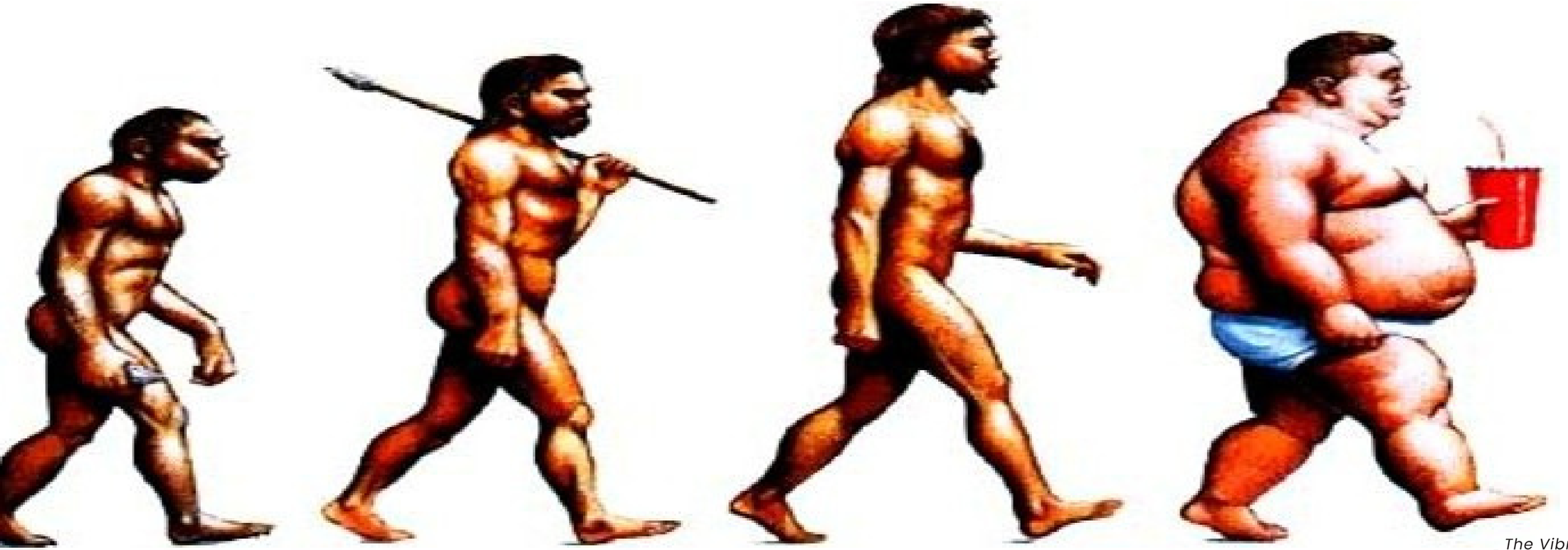
When adjustments are made for childhood mortality, our life expectancy is almost exactly the same as thousands of years ago.

https://pdfs.semanticscholar.org/c1b7/a4fa5c686a70dba254b6ac7d240e61a4a063.pdf



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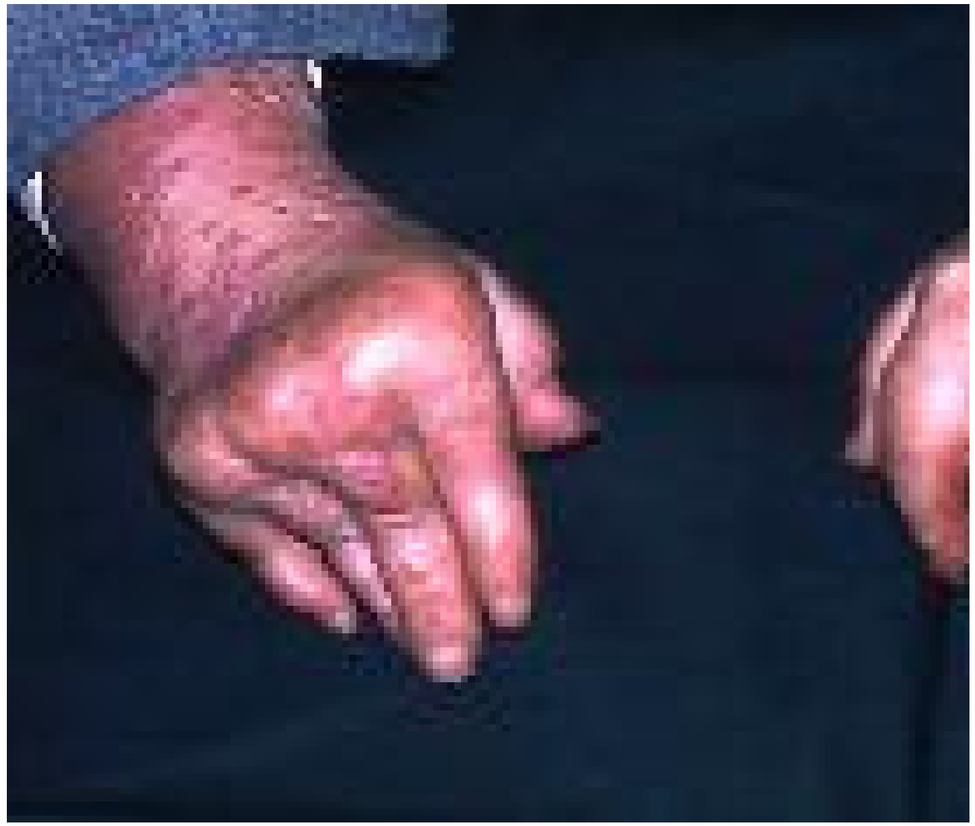
The Difference

Currently about 45% of the US population has at least 1 chronic disease

https://www.sciencedaily.com/releases/2015/06/150608081753.htm



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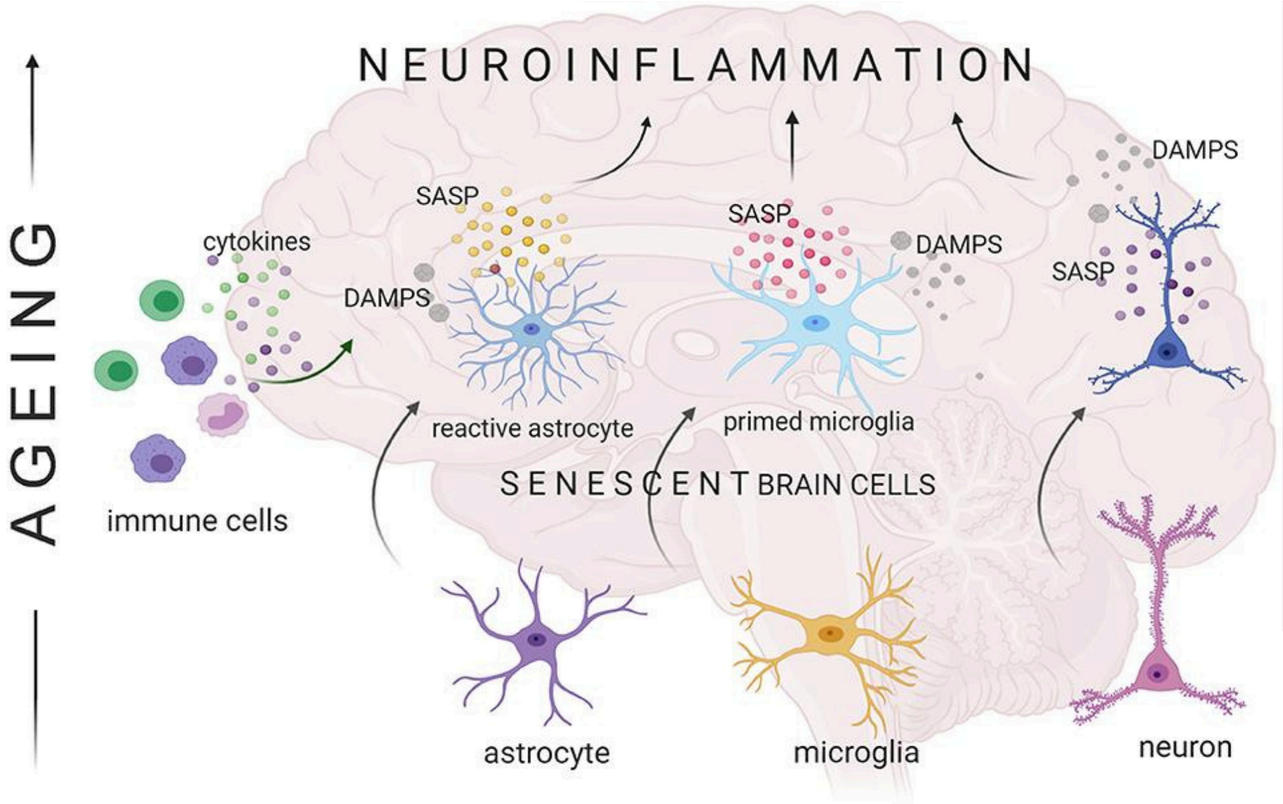
The Aging Process

The accelerated aging process is caused primarily by a deficiency of cellular antioxidant in the presence of increased Reactive Oxygen Species and Reactive Nitrogen Species. (Dr. G)



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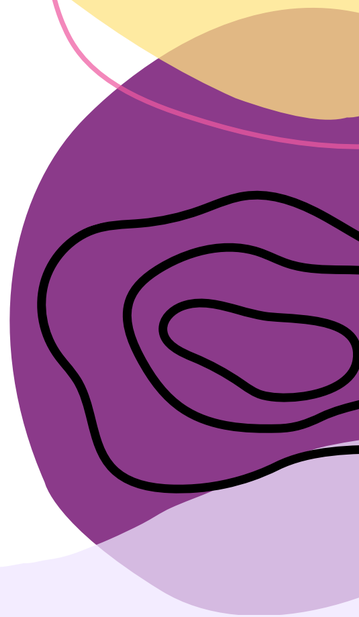
Microbiome and Inflammation

- Microbial Dysbiosis associated with pro-inflammatory cytokine cascade
  - IL-6, TNF-A, IL-17, IL-12, IL-23

- Targeted biologics
  - Enbrel, Humira, Skyrizi(tnf-a,il-12, il-6, and more)



Anti-cytokine cytokine

Pathogen free microbiota associated with increase IL-10

J Clin Invest. 2019 Sep 3; 129(9): 3702–3716.
Published online 2019 Aug 5. doi: 10.1172/JCI93820

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Is all disease just

- Exposures(environmental toxins, mycotoxins, metals, lyme)
- Infections(viral, bacterial, fungal, parasitic)
- Genetic expression(FUT, COMT, MTHFR, GAD, CBS and more)
- Poor lifestyle(diet, exercise, outdoors, stress)

Inflammaton in Gut tissue/BBB

- Lectins
- Antibiotics
- Chronic stress
- Infections
- Sucralose
- Sugar alcohols
- PPI
- Red synthetic food dyes

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Gut Barrier repair/BBB Repair

- Ghrh
- BPC 157/KPV
- lazarotide
- Glutamine
- DGL
- Aloe
- Bacteria...beneficial
- Low lectins
- regularity

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Inflammation is a normal part of the body's defense system against injury or infection. Inflammation within the nervous system or neuroinflammation is a complex phenomenon with both protective and detrimental effects in various neurological diseases. Initially serving as a natural defense mechanism during acute events such as infections, injuries, or trauma, inflammation helps contain the damage, clear debris, and initiate the healing process (Chen et al., 2018). However, in chronic conditions, sustained neuroinflammation can be detrimental, perpetuating a cycle of neuronal damage and degeneration. Chronic neuro-inflammation is characterized by excessive activation of glial cells, including microglia and astrocytes. These cells release pro-inflammatory cytokines, chemokines, and ROS, which can worsen neuronal damage and lead to progressive neural deterioration seen in diseases such as AD, PD, HD, and MS (Gąssowska-Dobrowolska et al., 2023).

Most Common Neurodegenerative Diseases

Parkinson’s

Dementia/Alzheimers

MS

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Alzheimer’s

Amyloid plaques can be present in the brains of individuals without Alzheimer's disease. While amyloid plaques are a hallmark of Alzheimer's, their presence alone doesn't guarantee a diagnosis.

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Parkinson’s

Lewy body pathology is found in the brains of 10 to 30% of older adults without any parkinsonian motor symptoms and is called incidental Lewy body disease (ILBD).

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Multiple Sclerosis

Fibroblasts, cells typically associated with wound healing and scar formation, are showing promise in the field of regenerative medicine, particularly in myelin repair. Research indicates that fibroblasts can be reprogrammed or stimulated to promote the regeneration of myelin, the protective sheath around nerve fibers, which is damaged in diseases like multiple sclerosis.

Clinical Picture

- 40 year old Male
- MS for 10 years
- Couldn’t move his legs or walk for 3 years
- Previously went to mayo clinic with no results
- Previously went to Mexico for stem cells and it slowed progression for 6 months, continued to progress thereafter

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What we did

- Baseline blood work:
  - (CBC w diff, cmp, lipid, full thyroid panel, ferritin, iron status, all immunoglobulins, vitamin D, uric acid, magnesium, testosterone, homocysteine, psa)

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Functional tests

- Neural autoimmune panel
- Organic acid
- Mycotoxins
- Envirotox
- Stool
- Tickborne 1.0

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Unremarkable

- Organic Acid testing
- Tickborne 1.0

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Remarkable

- A1C 5.7
- Testosterone 453
- Low igm
- High Reverse t3
- HS CRP 3.5
- High liver enzymes
- MPO 3825

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Remarkable

- Gut Zoomer
  - Parasite found(isospora belli)
  - Low Shannon and Simpson index
  - Fat digestion and vegetable fiber digestion
  - Pathogenic bacteria found(campylobacter)
  - Candida found

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Remarkable cont.

- Mycotoxins
  - Nivalenol
  - Zearalanone
- Neural zoomer plus
  - Many autoimmune markers indicative of MS
- Envirotoxins
  - 4 nonylphenol
  - Mono-phthalate

How we treated

- Veggies, Fruit and Meat
- Supplementation: binders, natural antibiotics, gut healers, fish oil, multivitamins, spores
- prescription anti-parasitics for 2 months
- IV NR, IV resveratrol, IV curcuminoids
- GHRH(ibutamoren)
- BPC157, TB500, KPV, GHK-CU

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Demyelination Antigens	Current (IgG + IgA)	Current IgM	Previous (IgG + IgA)	Previous IgM
Anti-Myelin proteolipid protein	10.3	7.6	21.4 (01-13-2025)	4.0 (01-13-2025)

The myelin proteolipid protein (PLP) is an integral protein in the myelin sheath, which is the insulating layer around nerves. PLP accounts for about 50% of the protein content of adult central nervous system myelin (CNS) and is essential for CNS myelin compaction. Anti-myelin PLP is believed to be associated with multiple sclerosis (MS) as there is an observed elevated T cell reactivity to myelin PLP in patients with MS.

Neuromuscular disorders	Current (IgG + IgA)	Current IgM	Previous (IgG + IgA)	Previous IgM
Anti-Acetylcholine receptors	10.2	5.9	11.0 (01-13-2025)	2.7 (01-13-2025)

Acetylcholine receptors (AChR) are present at nerve terminals, especially at the neuromuscular junction. They get activated by the neurotransmitter acetylcholine which helps in nerve conduction and brings about muscle action. Anti-acetylcholine antibodies interfere with muscle function and cause muscle weakness. Studies reveal that serum anti-AChR usually has an inverse relationship to muscle strength. Anti-AChR antibody is associated with myasthenia gravis (MG) which is characterized by weakness in muscles, fatigue, double vision, difficulties with speech and chewing. Anti-AChR antibody can also lead to restricted ocular myasthenia which affects the muscles that move the eyes and eyelids, resulting in blurry vision and drooping eyelids.

Brain Autoimmunity	Current (IgG + IgA)	Current IgM	Previous (IgG + IgA)	Previous IgM
Anti-Cerebellum	12.7	4.8	18.8 (01-13-2025)	5.4 (01-13-2025)

The cerebellum plays an important role in motor control and coordination. It does not initiate movement but aids in its coordination, timing, and precision. Cerebellum is one of the main targets in the central nervous system for autoimmunity. Anti-cerebellum antibodies could affect the functions of the cerebellum, thus giving rise to symptoms such as decreasing coordination, unsteady walking, impaired balance with frequent falls, heart problems, loss of fine motor skills, muscle tremors, slurred speech, and vision problems. Increased progression of these symptoms could lead to conditions like cerebellar ataxia and paraneoplastic cerebellar degeneration.

Anti-Purkinje cell	11.7	7.2	12.4 (01-13-2025)	6.3 (01-13-2025)

Purkinje cells are neurons of the cerebellar cortex. They play pivotal roles in coordination, control, and learning of movements. Most Purkinje neurons release a neurotransmitter called GABA (gamma-aminobutyric acid), which exerts inhibitory actions on neurons and thereby reduces the transmission of nerve impulses. Antibodies to Purkinje cells are associated with paraneoplastic cerebellar degeneration which is characterized by unsteady gait, double vision, and difficulty with fine hand movements. These symptoms can progress to give rise to cerebellar ataxia.

	(IgG + IgA)	IgM	(IgG + IgA)	IgM
Anti-AMPA receptor	12.4	9.0	14.3 (01-13-2025)	6.6 (01-13-2025)

AMPA receptor (AMPAR) is involved in excitatory neural impulse transmission, where it responds to the neurotransmitter glutamate. It is widely distributed in the central nervous system and brings about fast excitatory synaptic transmission. AMPAR plays a role in learning and memory. Anti-AMPAR antibodies are associated with symptoms like fever, seizures, headache, movement disorders, sensitivity to light and/or sound, neck stiffness, and loss of consciousness. Progression of these symptoms can lead to conditions like encephalitis, seizures, memory impairment, or psychosis.

Anti-Glycine receptor	10.3	4.2	11.5 (01-13-2025)	4.0 (01-13-2025)

Glycine receptors are inhibitory receptors present in the brainstem and the spinal cord. Its action leads to inhibition in neurotransmission. Antibodies against glycine receptors are seen to be associated with symptoms like muscle spasms, stiffness, and exaggerated startle responses. These symptoms could progress to conditions like hyperekplexia, rigidity, spasms, and myoclonus. They may also be accompanied by optic neuritis and cognitive decline. Anti-glycine receptor is also seen in patients with epilepsy.

Anti-Contactin-Associated Protein-like 2 Antibodies	11.5	5.5	12.4 (01-13-2025)	7.5 (01-13-2025)

Contactin-associated protein occurs at the paranodal junction, which is the junction between neurons and their corresponding glial (assistive, non-neuronal) cells. It has various functions but is mainly involved with nerve cell conduction. Anti-contactin-associated protein-like 2 (CASPR2) antibodies are associated with symptoms like anxiety, depression, irritability, mental confusion, and hallucinations, which could lead to limbic encephalitis in severe cases. CASPR2 antibodies are also seen to give rise to symptoms like muscle stiffness, abnormal muscle contractions, cramps, delayed muscle relaxation and increased sweating. Severe progression of this condition could lead to acquired neuromyotonia.

Anti-Dopamine receptor 1	11.2	4.4	12.6 (01-13-2025)	3.8 (01-13-2025)

Dopamine 1 receptor (D1R) is the most abundant dopamine receptor. It gets activated by the neurotransmitter dopamine. Dopamine receptors play an essential role in daily life functions, of which D1R is responsible for memory, attention, impulse control, regulation of renal function, and locomotion. Neuropsychiatric and movement disorders are associated with autoantibodies against D1R. D1R is also associated with the pathogenesis of Parkinson's disease (PD).

Anti-Dopamine receptor 2	11.6	4.9	15.5 (01-13-2025)	3.7 (01-13-2025)

Real Results

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Anything Is Possible

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