A FUNCTIONAL MEDICINE APPROACH TO HEALTH AND CHRONIC ILLNESS

RICK MENET MD, MPH

7 years of steady decline due MS


7 Year conventional therapy


1 Year FM therapy

Functional Medicine Take-Home Points

1. *Functional Medicine looks for the root causes based on how the body works - Systems Biology*

2. *Personalized Medicine (n of 1) – What works for one individual may not work for another- Epigenetics and unique environmental exposures*

3. *Personal Responsibility – Empowerment through Transformational change in Diet & Lifestyle,*

FUNCTIONAL MEDICINE MATRIX

Physiology and Function: Organizing the Patient's Clinical Imbalances

Assimilation
(e.g., Digestion, Absorption, Microbiota/GI, Respiration)

Defense & Repair
(e.g., Immune, Inflammation, Infection/Microbiota)

Structural Integrity
(e.g., from Subcellular Membranes to Musculoskeletal Structure)

Energy
(e.g., Energy Regulation, Mitochondrial Function)

Communication
(e.g., Endocrine, Neurotransmitters, Immune messengers)

Biotransformation & Elimination
(e.g., Toxicity, Detoxification)

Transport
(e.g., Cardiovascular, Lymphatic System)

THE INSTITUTE FOR FUNCTIONAL MEDICINE®

FUNCTIONAL MEDICINE MATRIX

Retelling the Patient's Story

Antecedents
(Predisposing Factors—Genetic/Environmental)

Triggering Events
(Activators)

Mediators/Perpetuators
(Contributors)

Physiology and Function: Organizing the Patient's Clinical Imbalances

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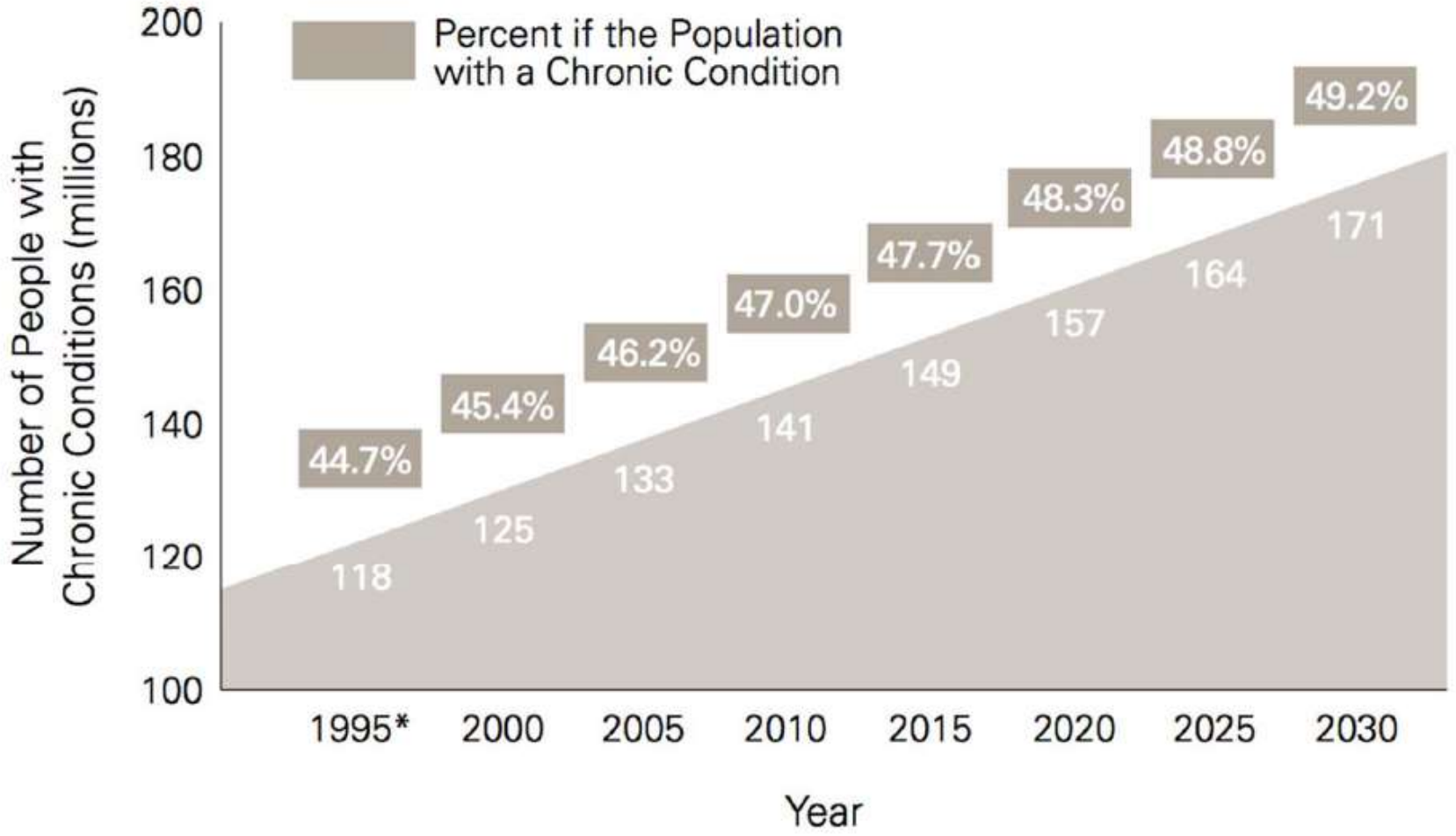
Transport
(e.g., Cardiovascular, Lymphatic System)

Modifiable Personal Lifestyle Factors

Sleep & Relaxation	Exercise & Movement	Nutrition	Stress	Relationships

Name: _____ Date: _____ CC: _____

Chart 1: The Number of People with Chronic Conditions is Rapidly Increasing



Source: Wu, Shin-Yi, and Green, Anthony. *Projection of Chronic Illness Prevalence and Cost Inflation.* RAND Corporation, October 2000.

Crisis of Chronic Diseases

- 75% of health care costs are due to chronic diseases
- 60% of deaths are due to chronic diseases
  - Heart disease
  - Cancer
  - Diabetes
- Leading causes of death worldwide are chronic diseases

WHO 2016

ThedaCare™

Number of Americans with Alzheimer's expected to soar in coming decades

The number of people living with Alzheimer's disease and related dementias will double by 2060, according to a new study from the Centers for Disease Control and Prevention.

Alzheimer's disease is the sixth leading cause of death in the United States and the fifth leading cause among those aged 65 and older. It's an irreversible, progressive brain disorder that slowly robs people of memory and, eventually, a person's ability to perform even the simplest tasks. There is no cure.

In 2014, there were 5 million people in the U.S. with Alzheimer's disease and related dementias – about 1.6 percent of the U.S. population. That number is projected to grow to 13.9 million, nearly 3.3 percent of the population, in 2060.

DO YOU REALLY NEED THAT PILL?

HOW TO AVOID SIDE EFFECTS, INTERACTIONS, AND OTHER DANGERS OF OVERMEDICATION

JENNIFER JACOBS, MD, MPH

OVERMEDCATION

- Prescription Drug costs in US in 2016 was 380 billion dollars and projected to total 590 billion by 2020.
- Five or more prescription drugs in 21% of the population (40% of those over 65.
- Anti-depressant drugs prescribed to 12%
- Antibiotics drugs to 17%
- Advertising spent 14 million dollars per day in 2015
- Prescriptions to treat side effects

Adverse Drug Reactions

- 2017 – FDA reports that ~ 450 people die daily from drug related side effects
  - EQUIVALENT TO A BOEING 747 GOING DOWN EVERY DAY!

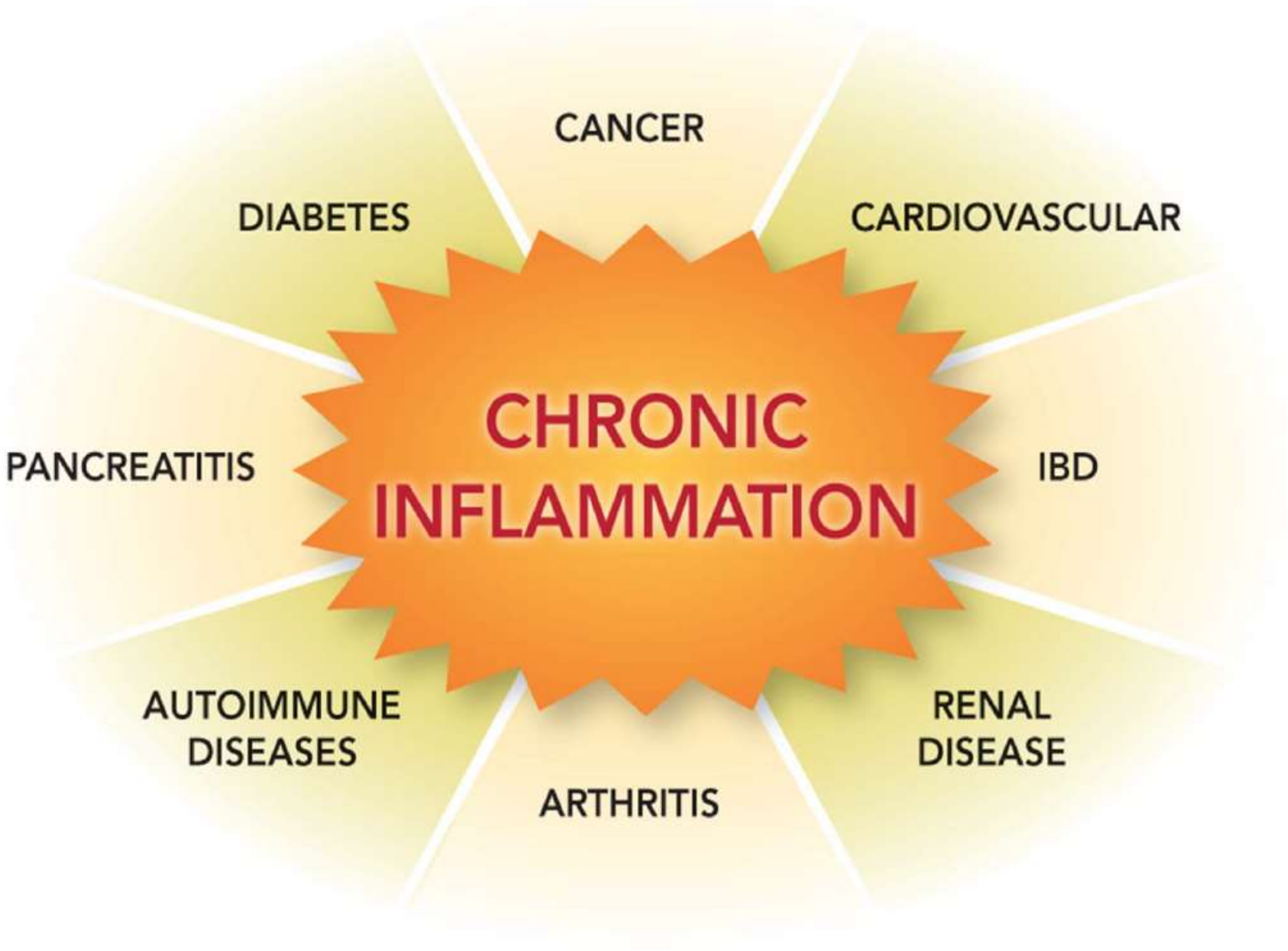
- 2017 – FDA reported 900,000 serious ADRs, 2500 per day including, hospitalizations, life threatening, and serious reactions

- 2014 – Pharmaceutical Industry reported 1.2 million ADR’s

IF YOU WANT TO TRANSFORM THE WAY YOU PRACTICE, YOU NEED A PLAN

FUNCTIONAL MEDICINE

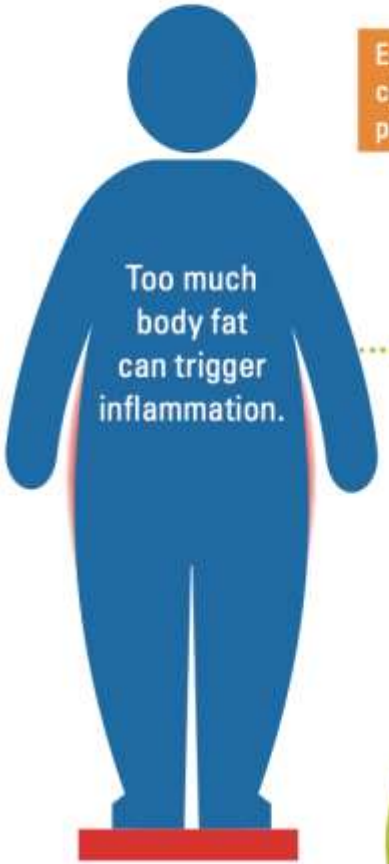
addresses the underlying causes of disease, using a systems-oriented approach and engaging both patient and practitioner in a therapeutic partnership.



CAUSES OF CHRONIC INFLAMMATION:

- STRESS
- FOOD SENSITIVITIES
- ALLERGENS
- TOXINS
- MICROBES
- NUTRITION
- TRAUMA

How excess body fat can cause cancer



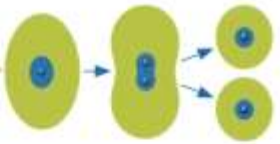
Too much body fat can trigger inflammation.

Excess body fat increases the risk for certain types of cancer, including colorectal, esophageal, kidney, pancreatic, post-menopausal breast and endometrial cancers.

Inflammation causes the pancreas to produce more insulin.

Extra fat cells also make estrogen.

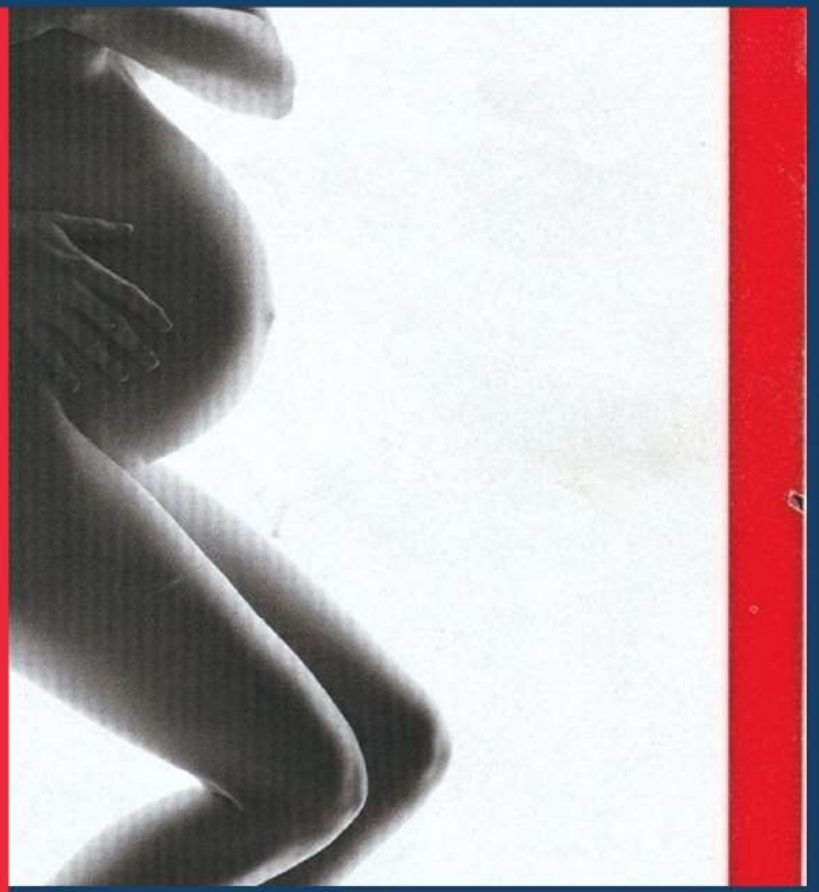
Too much insulin and estrogen cause cells to divide more than usual.



In some cases, these new cells can form cancerous tumors.

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History®

The Epigenetics Revolution



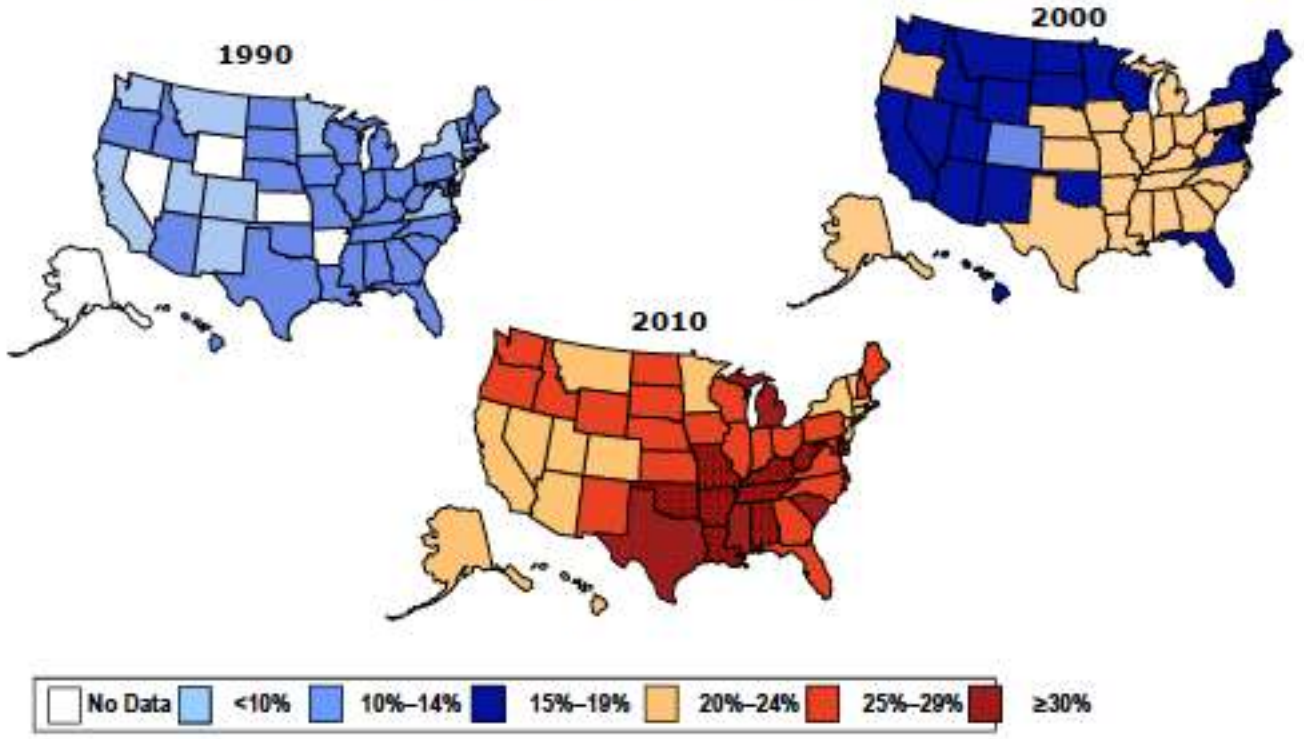
IFM

A Functional Medicine Approach to Type II Diabetes

ROLE OF INSULIN RESISTANCE

Obesity Trends* Among U.S. Adults
BRFSS, 1990, 2000, 2010

(*BMI ≥30, or about 30 lbs. overweight for 5' 4" person)



http://www.cdc.gov/obesity/data/trends.html

Excess body weight: A major health issue in America

Modern life in America has led many people to eat more unhealthy foods, eat bigger food portions, and be less active. As a result, the number of Americans who are overweight or obese (very overweight) has been rising. About 1 in 3 American adults is now obese, and another 1 in 3 is overweight.

Being overweight or obese can have far-reaching health consequences. According to the US Centers for Disease Control and Prevention (CDC), excess body weight increases a person's risk for:

- Heart disease
- Type 2 diabetes
- High blood pressure
- High cholesterol levels
- Stroke
- Liver and gallbladder disease
- Sleep apnea and respiratory problems
- Arthritis
- Abnormal menstrual periods and infertility in women
- Certain cancers


DIABESITY

With ‘bliss points’ and ‘mouth feel,’ food industry plays role in hedonic eating

FOOD INDUSTRY IS MANIPULATING PROCESSED FOOD TO BE IRRESISTIBLE

NASHVILLE, Tenn. — The growing obesity epidemic in the U.S. is helped along, in part, by a savvy food industry that uses a combination of science and expert marketing to influence vulnerable consumers, according to a speaker here.

***SALT SUGAR FAT: HOW THE FOOD GIANTS HOOKED US* BY MICHAEL MOSS**

ObesityWeek 2018.


The Empowering Neurologist - David Perlmutt...
Watch later
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REVERSING TYPE II DIABETES
DR. SARAH HALLBERG
davidperlmutter MD
empowering neurologist
0:00 / 38:26
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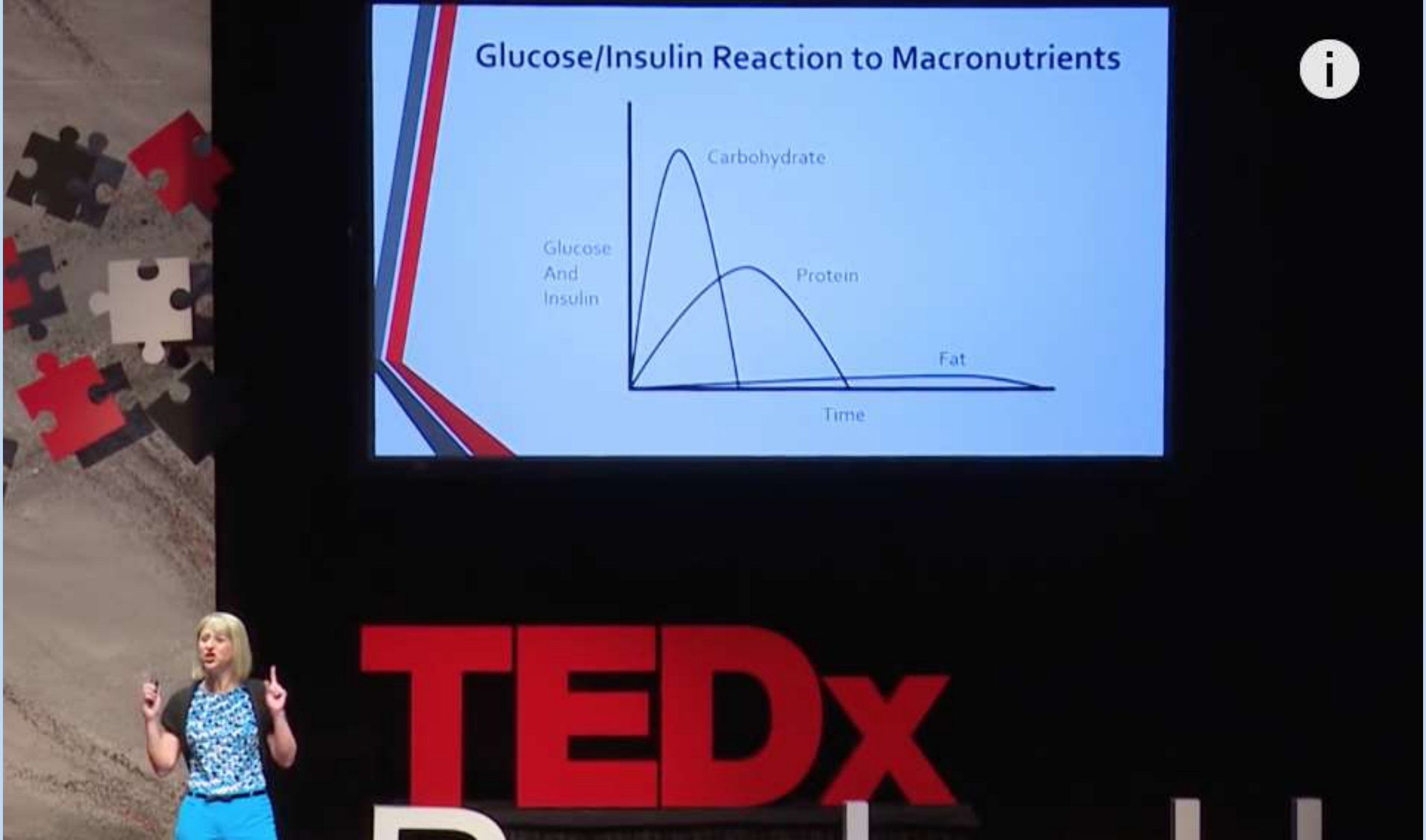

Glucose/Insulin Reaction to Macronutrients
Carbohydrate
Protein
Fat
Glucose
And
Insulin
Time
TEDx

Virta's Clinical Trial

Our peer-reviewed, published research demonstrates that type 2 diabetes reversal can happen fast—and our results last.

Decrease Medications

94%

of insulin users reduced or eliminated usage after 1 year

Reverse Diabetes

60%

of patients reversed their type 2 diabetes after 1 year*

Lower A1c

▼ 1.3%

Average HbA1c reduction after 1 year

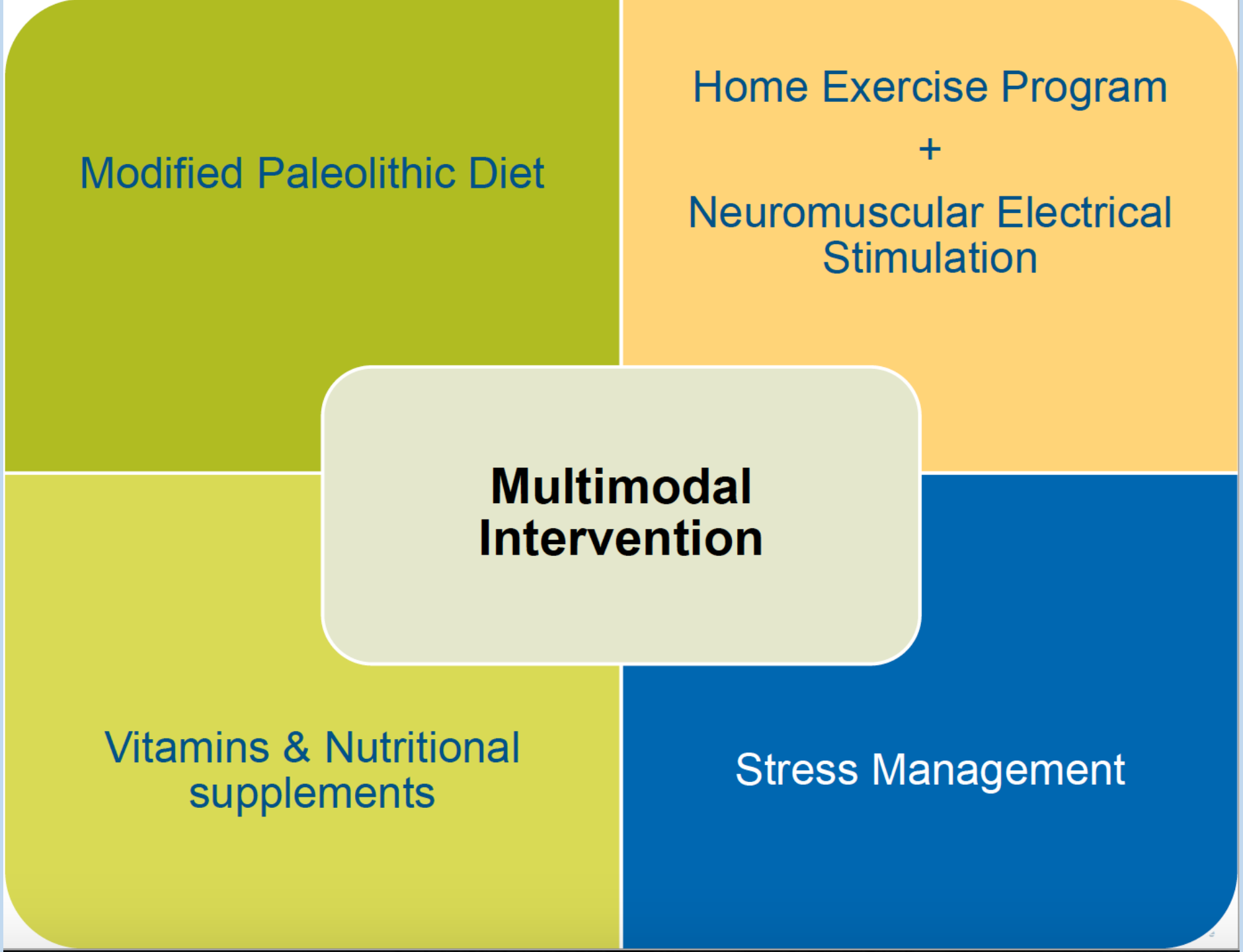
Being overweight or obese is clearly linked with an increased risk of many types of cancer, including cancers of the:

- Breast (in women past menopause)
- Colon and rectum
- Endometrium (lining of the uterus)
- Esophagus
- Kidney
- Pancreas

Being overweight or obese might also raise the risk of other cancers, such as:

- Gallbladder
- Liver
- Non-Hodgkin lymphoma
- Multiple myeloma
- Cervix
- Ovary
- Aggressive forms of prostate cancer

The Wahls Protocol


Modified Paleolithic Diet
Home Exercise Program
+
Neuromuscular Electrical Stimulation
Multimodal Intervention
Vitamins & Nutritional supplements
Stress Management

Daily - Eat 9 Cups Vegetables/Fruit

3 Greens

3 Colored

3 Sulfur

Grass-fed Meats, Organ Meats, and Wild Fish

BRISTOL STOOL CHART

Type 1	Separate hard lumps	Very constipated

Type 2	Lumpy and sausage like	Slightly constipated

Type 3	A sausage shape with cracks in the surface	Normal

Type 4	Like a smooth, soft sausage or snake	Normal

Type 5	Soft blobs with clear-cut edges	Lacking fibre

Type 6	Mushy consistency with ragged edges	Inflammation

Type 7	Liquid consistency with no solid pieces	Inflammation

Sleep & Relaxation Takeaways

ASSESSMENT

- How restful
- Hours slept
- Sleep studies as needed

TREATMENT

- Appropriate light
  - UV exposure in a.m., No UV exposure p.m.
- Melatonin
- Taurine
- Magnesium
- Epsom salts
- Essential oils
- Massage
- OSA treatments as needed

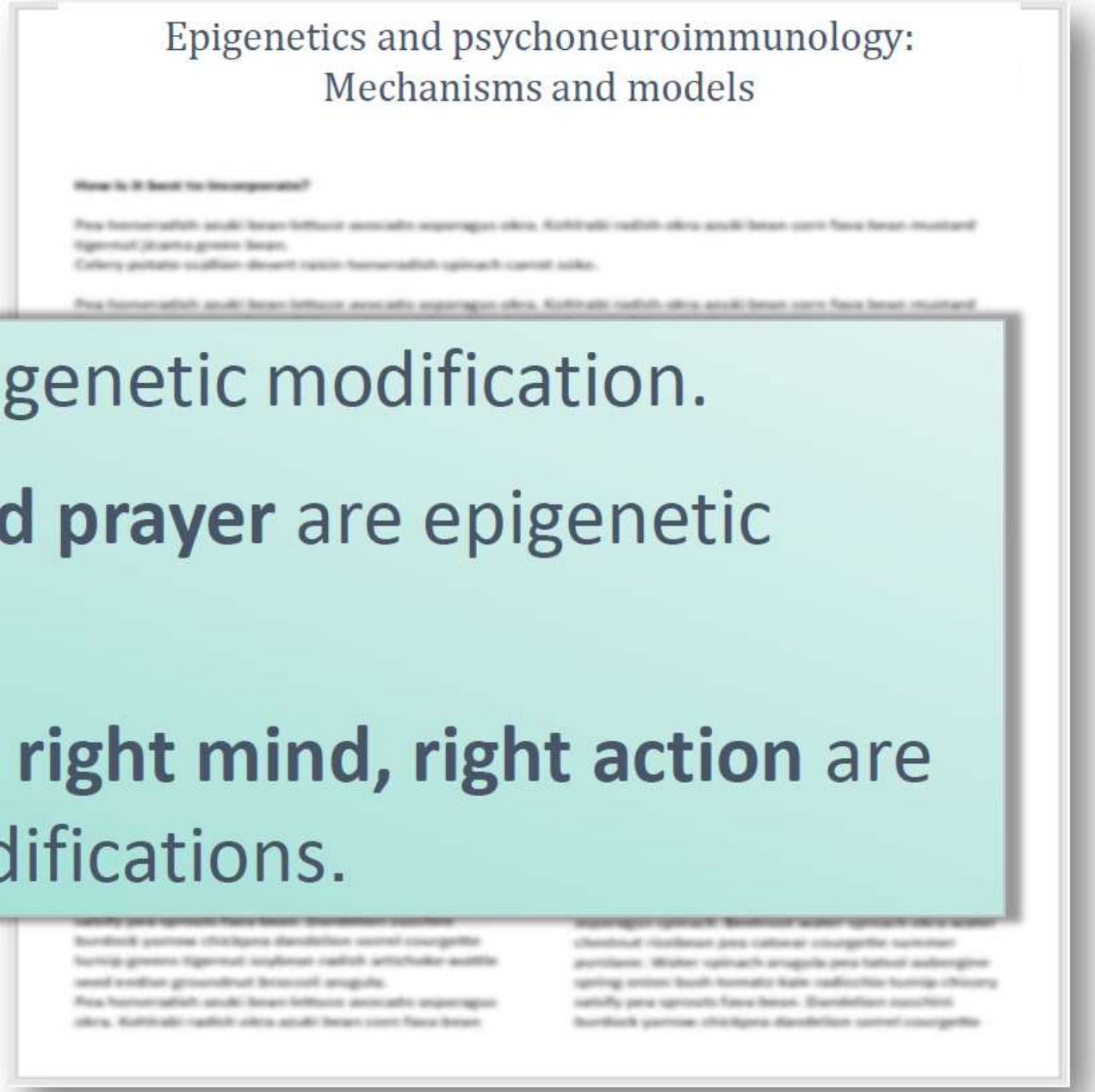
Exercise & Movement Takeaways

ASSESSMENT

- DNL questionnaire
- Tolerance for activity

TREATMENT

- Physical/ occupational therapy
- Yoga, Tai Chi
- Strength and balance training
- Do not over-train
- IFM Exercise Prescription



- **Stress** is an epigenetic modification.
- **Meditation and prayer** are epigenetic modifications.
- **Right thought, right mind, right action** are epigenetic modifications.

Mathews HL, Janusek LW. Epigenetics and Psychoneuroimmunology: Mechanisms and Models. Brain, behavior, and immunity. 2011;25(1):25-39. doi:10.1016/j.bbi.2010.08.009.

Stress Takeaways

ASSESSMENT

- Ask about stress-reducing practices

TREATMENT

- Meditation
- Mindfulness
- Gardening
- Fishing, hunting
- Journaling
- HeartMath

- For patients AND Caregivers

Relationships Takeaways

ASSESSMENT

- Any supportive peer or individual

TREATMENT

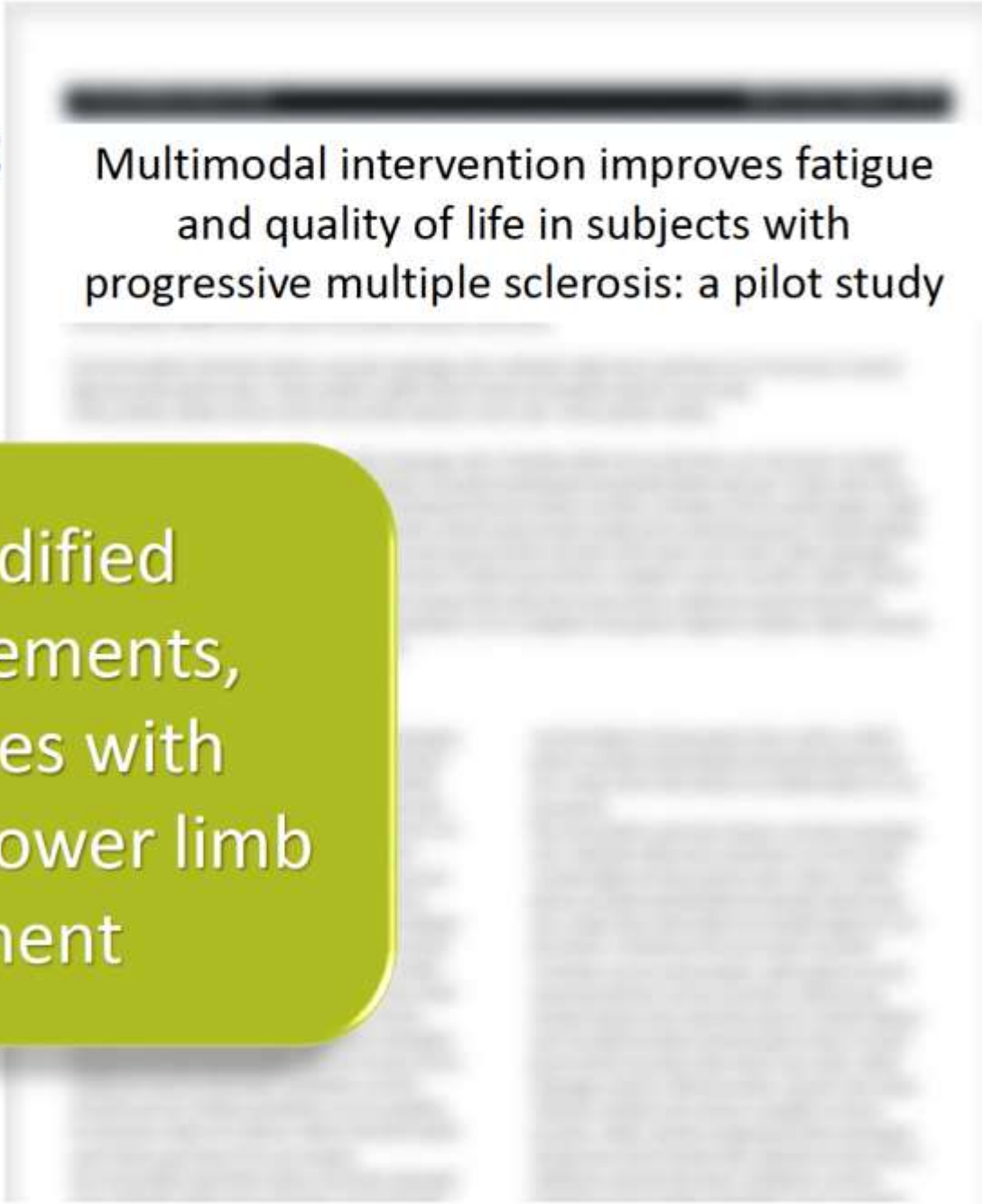
- Encourage connecting virtually or in person
- Remember to take care of the caregivers!

Study Diet

Food	Instruction	Servings
Green leafy vegetables	Recommended*	3 cups cooked/6 cups raw=3srvg
Sulfur-rich vegetables	Recommended*	3 cups raw or cooked= 3srvg
Intensely colored fruits or vegetables	Recommended*	3 cups raw or cooked =3 srvg
Omega-3 oils	Encouraged	2 tablespoons
Animal protein	Encouraged	4 ounces or more
Gluten-containing grain	Excluded	
Dairy	Excluded	
Eggs	Excluded	

IFM

Multimodal intervention improves fatigue and quality of life


Multimodal intervention improves fatigue and quality of life in subjects with progressive multiple sclerosis: a pilot study

A multimodal intervention: modified Paleolithic diet, nutritional supplements, stretching, strengthening exercises with electrical stimulation of trunk and lower limb muscles, and stress management

Wahls T, Bisht B, Darling W et al. Multimodal intervention improves fatigue and quality of life in subjects with progressive multiple sclerosis: a pilot study. *Degenerative Neurological and Neuromuscular Disease*. 2015:19. doi:10.2147/dnnd.s76523.

Critical Clinical Pearl

- Interrupting treatments that have been working predictably causes rebound relapses.
- This is true with drugs and modifiable lifestyle factors (MLF).
- If a patient wants to transition from drug interventions to MLF interventions, **you must employ MLF for at least 12-36 months with good effect before even attempting to gradually reduce the drug therapy.**

EDITORIAL

Rebound Relapses After Ceasing Another Disease-Modifying Treatment in Patients With Multiple Sclerosis

Are There Lessons to Be Learned?

Rhonda Voskuhl, MD

"Do no harm" is the timeless tenet of the Hippocratic Oath. It applies not only to how patients tolerate prescribed treatments but also to how they tolerate treatments after discontinuation. The sum of both events must be considered in the overall risk-benefit ratio before starting any given treatment.

The article by Hatcher et al[1] published in this issue of *JAMA Neurology* describes a rebound in multiple sclerosis (MS) relapses after ceasing fingolimod treatment. Rebound occurred in 5 of 46 patients (10.9%) at their center, with additional reports identified by the authors' literature search. Rebounds in relapses were clinically severe and accompanied by magnetic resonance imaging lesion activity that surpassed the level of activity before starting treatment. The first step during postmarketing experience is to report adverse events to increase awareness and provide a rationale for a registry to be created to establish frequency. This is addressed by Hatcher et al.[1] The next step is to understand why rebound is occurring in order to prevent it from happening to other patients in the future. Finally, a discussion of the altered risk-benefit ratio of fingolimod within the context of currently evolving schools of thought regarding disease-modifying treatments in relapsing-remitting MS (RRMS) is warranted.

Related article page 790

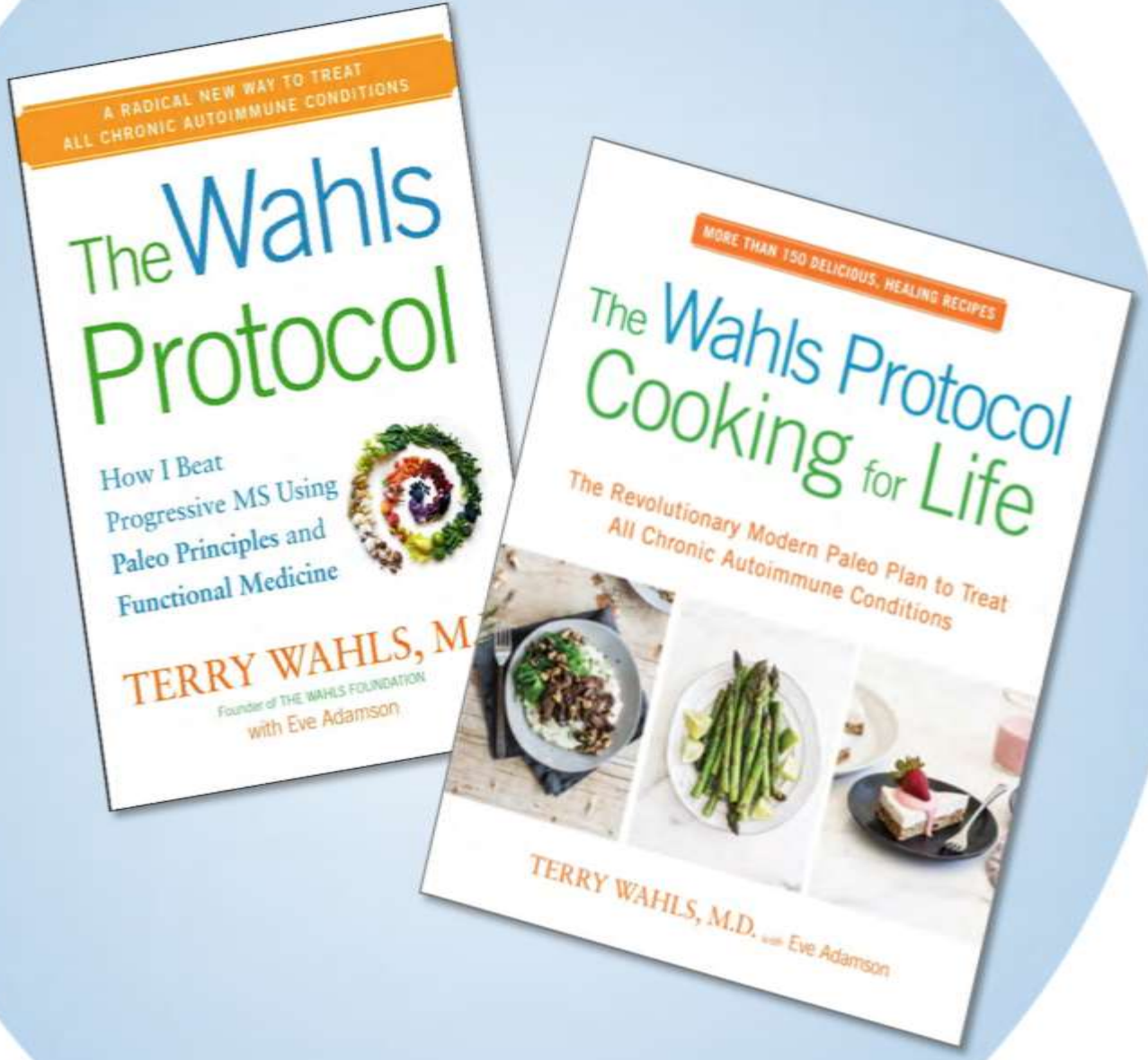
The present article[1] documenting rebound relapse activity after stopping fingolimod treatment is particularly important because a similar rebound of disease activity after stopping natalizumab treatment has been well documented.[2-4]

Why would fingolimod and natalizumab be associated with a rebound in disease activity after ceasing treatment compared with other MS disease-modifying therapies that have not been associated with a rebound? It may have to do with targeting immune cell trafficking. Fingolimod acts on sphingosine-1-phosphate receptors, which results in sequestration of lymphocytes in secondary lymphoid tissues and leads to less infiltration of these cells into the brain during MS. In turn, this reduces inflammatory lesions in white matter and reduces MS relapses. Natalizumab acts on α-4 integrin receptors on lymphocytes to block their passage across the blood-brain barrier and reduce their infiltration into the brain. Perhaps treatments that are designed to block immune cells from entering the brain, either at the level of the blood-brain barrier with natalizumab or at the level of lymphoid tissues with fingolimod, can be associated with rebound relapses after ceasing treatment. In contrast, rebound disease activity over and above pretreatment levels is not an issue for first-line disease-modifying treatments for MS (eg, glatiramer acetate and interferon-β), despite extensive use for decades. These relatively safe, long-standing disease-modifying injectable treatments do not directly target immune cell trafficking but instead act indirectly through a variety of immunomodulatory mechanisms to reduce the proinflammatory profile of immune cells in peripheral blood, with a downstream effect of less inflammation in the brain.

In many biological systems, chronic receptor blocking and/or receptor stimulation can eventually affect not only the expression of target receptors but also the expression of transcription factors involved in receptor stimulation. Accordingly, chronic targeting of the receptor interactions related to trafficking immune cells from lymphoid organs could lead to compensatory changes in other molecules redundant in such trafficking. It is tempting to speculate that chronic sphingosine-1-phosphate receptor blocking with fingolimod could have unexpected effects on receptor physiology in secondary lymphoid tissues. Therefore, sudden cessation of fingolimod treatment could result in even more efflux of lymphocytes from lymph nodes than during pretreatment as a consequence of these upregulated compensatory pathways. This efflux would involve immune cells that are not otherwise immunomodulated, paving the way for rebounds of disease activity even higher than pretreatment. Future studies are needed to discern the mechanism of rebound relapses with cessation of fingolimod treatment, as Hatcher et al[1] acknowledge. However, these future studies will require much more than quantification of immune cell types in the blood during rebound; they will likely entail assessment of receptor physiology within secondary lymphoid tissues. Such mechanistic studies are important because there may be implications for treatments beyond fingolimod, both currently and in the future.

Given the new finding of the potential for rebound relapses after ceasing fingolimod treatment, a discussion of the altered risk-benefit ratio is salient and should be considered in the context of larger treatment-related issues in MS. Indeed, this is timely in light of evolving opinions regarding the selection of the best disease-modifying treatment approach for early RRMS. There are 2 schools of thought regarding how aggressive treatments should be in patients with early RRMS. In the past, most physicians started patients with the safest MS treatments. If they "failed" such treatments, as evidenced by relapse frequency at or near pretreatment levels, then escalation

R Voskuhl. Rebound Relapses After Ceasing Another Disease-Modifying Treatment in Patients With Multiple Sclerosis: Are There Lessons to Be Learned? JAMA Neurol. 2016 May 2. doi: 10.1001/jamaneurol.2016.0934. [Epub ahead of print]



www.terrywahls.com

RRMS & Fatigue Study funded by NMSS

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ED RAPP – Stay Strong Vs ALS

- Conventional Medical Care at John Hopkins
- Functional Medicine with supplements, detox, and IV lipids
- Wahl's Protocol
- Foundation Training
- Chiropractic and Acupuncture
- Prayer and Affirmation

The Collaborative Care Team

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1. *Functional Medicine looks for the root causes based on how the body works - Systems Biology*

2. *Personalized Medicine (n of 1) – What works for one individual may not work for another- Epigenetics and unique environmental exposures*

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NEW YORK TIMES BESTSELLER

"A MONUMENTAL WORK."
—DAVID PERLMUTTER, MD
author of the #1 New York Times bestsellers *Grain Brain* and *Brain Maker*

The End *of* Alzheimer's

The First Program to Prevent and Reverse Cognitive Decline

DALE E. BREDESEN, MD
Professor and Founding President, Buck Institute; Professor, UCLA

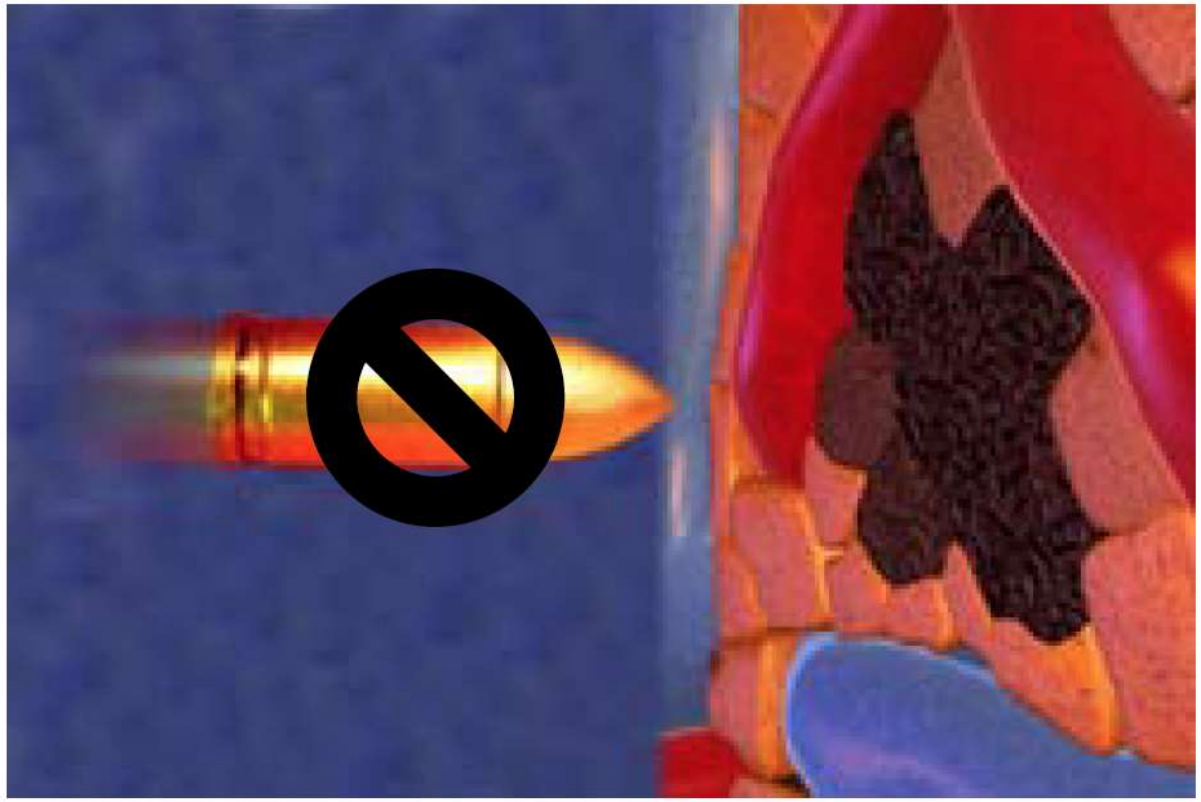
Reversing Cognitive Decline

- Functional Medicine Approach to Alzheimer's disease – multimodal intervention -Case series
- Patients with Alzheimer's disease (AD), amnestic mild cognitive impairment (aMCI), or subjective cognitive impairment (SCI)
- **9 of 10** patients who have utilized the multi-modal program **displayed improvements in cognition within 3-6 months**
- 10 of 10 improved [quantitative neuropsychological testing composite memory score at the 32nd percentile. After four months → 61st percentile].

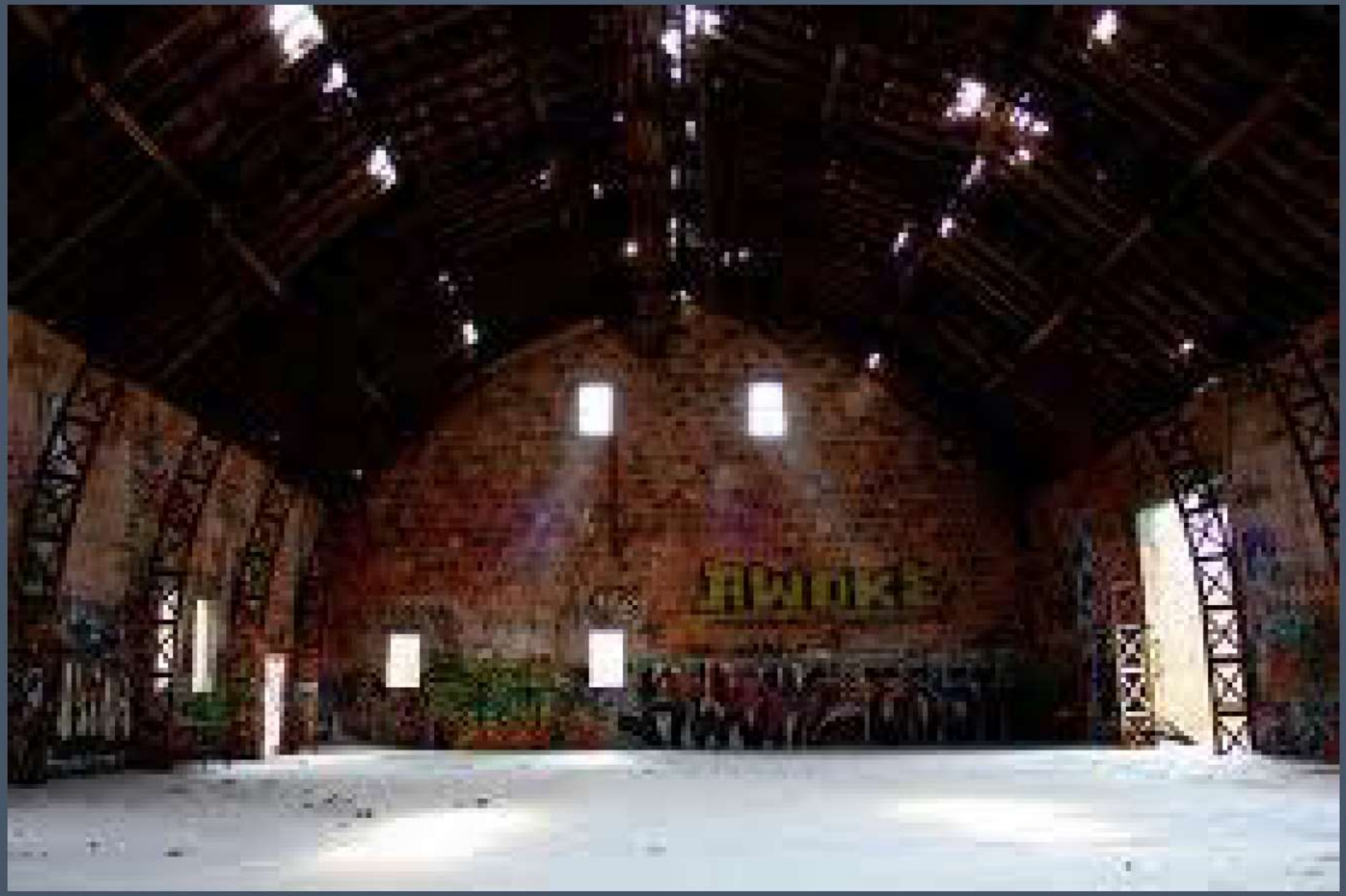
1. Bredesen DE. Reversal of cognitive decline: A novel therapeutic program. Aging (Albany NY). 2014;6(9):707-717.
2. Bredesen DE, Amos EC, Canick J, et al. Reversal of cognitive decline in Alzheimer's disease. Aging (Albany NY). 2016;8(6):1250-1258. doi:10.18632/aging.100981.

20th Century Therapeutics

SILVER BULLETS

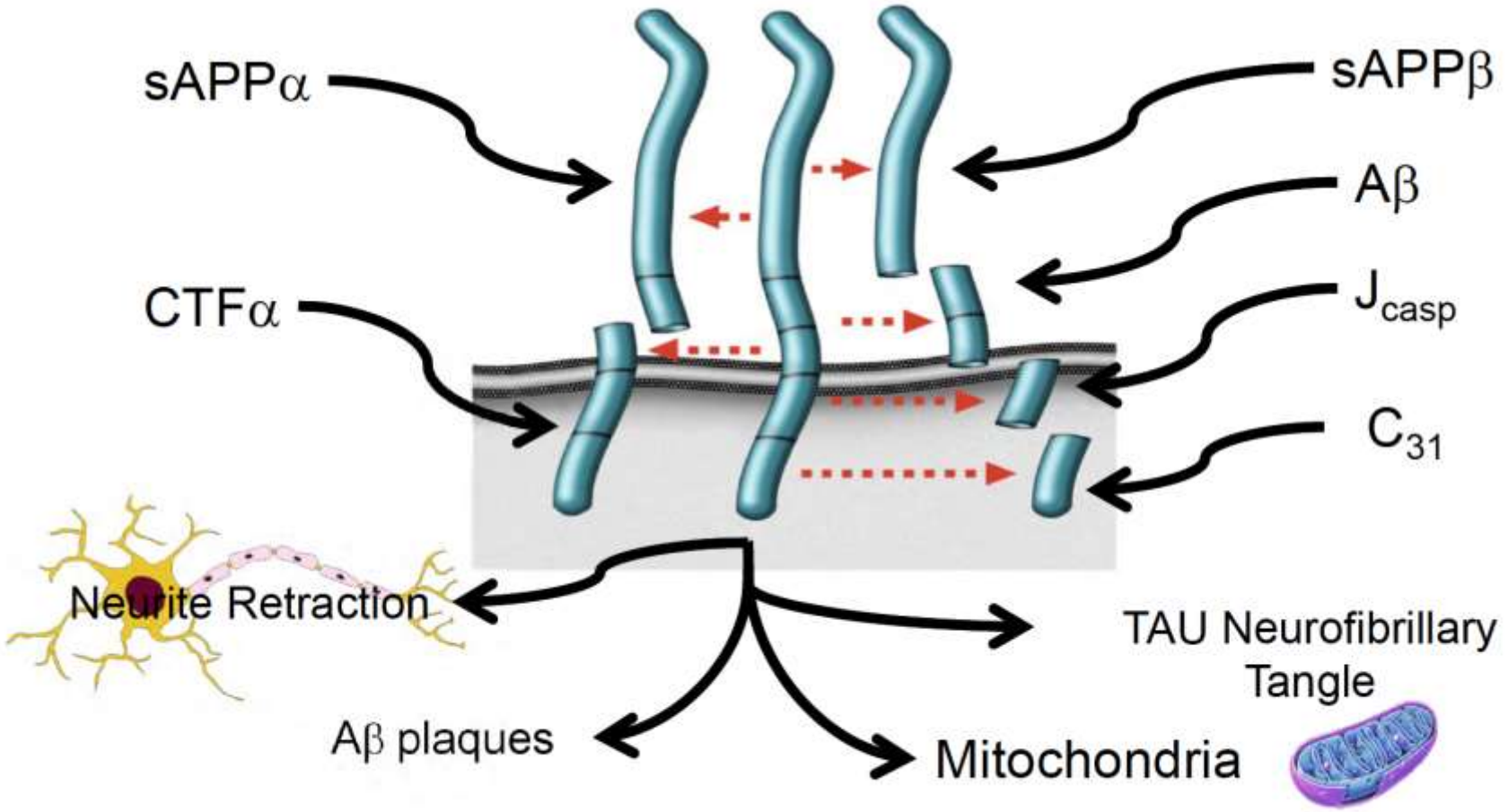


A roof with 36 holes…



Trophic, Anti-AD

Anti-trophic, Pro-AD



The "Perfect" Alzheimer's Drug Would:

Reduce APP **b**-cleavage, reduce **g**-cleavage, increase **a**-cleavage, reduce caspase-6 cleavage, reduce caspase-3 cleavage, prevent oligomerization, increase neprilysin, increase IDE, increase microglial clearance of A**b**, increase autophagy, increase BDNF, increase NGF, increase netrin-1, increase ADNP, reduce homocysteine, increase PP2A activity, reduce phospho-tau, increase phagocytosis index, increase insulin sensitivity, improve axoplasmic tran and biogenesis, reduce oxidative damage and op ergic neurotransmission, increase synaptoblastic s g, improve LTP, optimize estradiol, progesterone, E2:P ratio, free T3, free T4, TSH, pregnenolone, testosterone, cortisol, DHEA, and insulin, reduce inflammation, increase resolvins, enhance detoxification, improve vascularization, increase cAMP, increase glutathione, provide synaptic components, optimize all metals, increase GABA, increase vitamin D signaling, increase SirT1, reduce NFkB, increase telomere length, reduce glial scarring, enhance repair...

Threshold Effect

“Berfooda Triangle”

Foods to avoid

Simple Carbohydrates

Saturated Fats

Lack of Fiber

~Dale Bredesen MD

"Whole foods,
Mostly plants,
Not too much!"

~Michael Pollen

Table 1. Therapeutic System 1.0

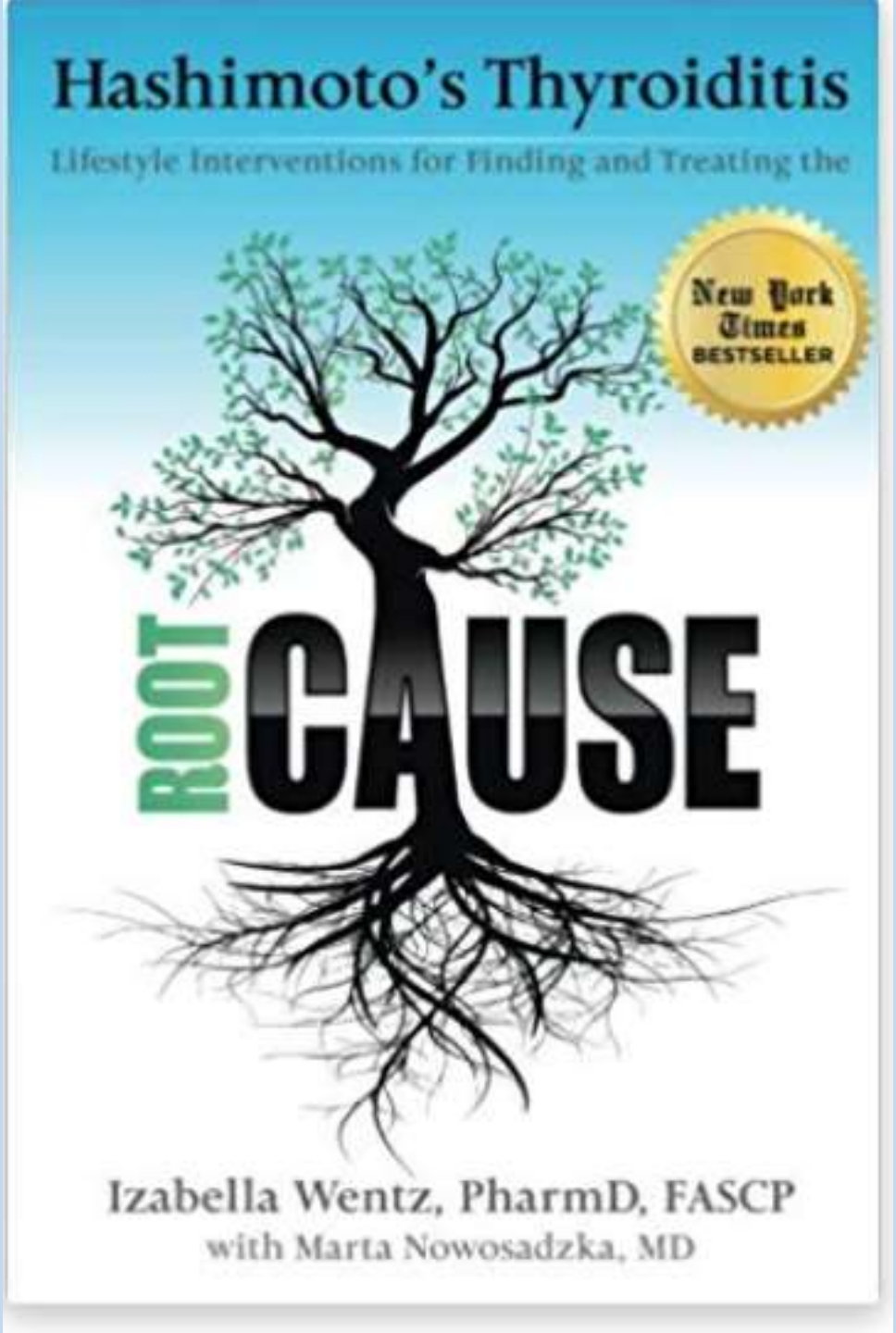
Goal	Approach	Rationale and References
Optimize diet: minimize simple CHO, minimize inflammation.	Patients given choice of several low glycemic, low inflammatory, low grain diets.	Minimize inflammation, minimize insulin resistance.
Enhance autophagy, ketogenesis	Fast 12 hr each night, including 3 hr prior to bedtime.	Reduce insulin levels, reduce Aβ.
Reduce stress	Personalized—yoga or meditation or music, etc.	Reduction of cortisol, CRF, stress axis.
Optimize sleep	8 hr sleep per night; melatonin 0.5mg po qhs; Trp 500mg po 3x/wk if awakening. Exclude sleep apnea.	[36]
Exercise	30-60′ per day, 4-6 days/wk	[37, 38]
Brain stimulation	Posit or related	[39]
Homocysteine <7	Me-B12, MTHF, P5P; TMG if necessary	[40]
Serum B12 >500	Me-B12	[41]
CRP <1.0; A/G >1.5	Anti-inflammatory diet; curcumin; DHA/EPA; optimize hygiene	Critical role of inflammation in AD
Fasting insulin <7; HgbA1c <5.5	Diet as above	Type II diabetes-AD relationship
Hormone balance	Optimize fT3, fT4, E2, T, progesterone, pregnenolone, cortisol	[5, 42]
GI health	Repair if needed; prebiotics and probiotics	Avoid inflammation, autoimmunity
Reduction of A-beta	Curcumin, Ashwagandha	43-45
Cognitive enhancement	Bacopa monniera, MgT	[46, 47]
25OH-D3 = 50-100ng/ml	Vitamins D3, K2	[48]
Increase NGF	H. erinaceus or ALCAR	[49, 50]
Provide synaptic structural components	Citicoline, DHA	[51].
Optimize antioxidants	Mixed tocopherols and tocotrienols, Se, blueberries, NAC, ascorbate, α-lipoic acid	[52]
Optimize Zn:fCu ratio	Depends on values obtained	[53]
Ensure nocturnal oxygenation	Exclude or treat sleep apnea	[54]
Optimize mitochondrial function	CoQ or ubiquinol, α-lipoic acid, PQQ, NAC, ALCAR, Se, Zn, resveratrol, ascorbate, thiamine	[55]
Increase focus	Pantothenic acid	Acetylcholine synthesis requirement
Increase SirT1 function	Resveratrol	[32]
Exclude heavy metal toxicity	Evaluate Hg, Pb, Cd; chelate if indicated	CNS effects of heavy metals
MCT effects	Coconut oil or Axona	[56]

CHO, carbohydrates; Hg, mercury; Pb, lead; Cd, cadmium; MCT, medium chain triglycerides; PQQ, polyquinoline quinone; NAC, N-acetyl cysteine; CoQ, coenzyme Q; ALCAR, acetyl-L-carnitine; DHA, docosahexaenoic acid; MgT, magnesium threonate; fT3, free triiodothyronine; fT4, free thyroxine; E2, estradiol; T, testosterone; Me-B12, methylcobalamin; MTHF, methyltetrahydrofolate; P5P, pyridoxal-5-phosphate; TMG, trimethylglycine; Trp, tryptophan

E Bredesen. Reversal of cognitive decline:
novel therapeutic program.
aging (Albany NY). 2014 Sep; 6(9): 707–717.

HYPOTHYROIDISM

- THYROID REPLACEMENT THERAPY
- VS
- TREATMENT OF AUTOIMMUNE DISEASE
- HASHIMOTO'S THYROIDITIS


Hashimoto's Thyroiditis
Lifestyle Interventions for Finding and Treating the
New York Times BESTSELLER
ROOT CAUSE
Izabella Wentz, PharmD, FASCP
with Marta Nowosadzka, MD

MANAGEMENT OF HYPOTHRYOIDISM

- TRADITIONAL STANDARD OF CARE - THRYROID HORMONE REPLACEMENT GUIDED BY TSH

- FUNCTIONAL MEDICINE APPROACH – DIETARY/LIFESTYLE CHANGES TO REMOVE TRIGGERS AND MEDIATORS. SUPPLEMENTATION/NUTRIENTS, PHYTONUTRIENTS, THYROID HORMONE REPLACEMENT GUIDED BY COMPLETE THRYROID PANEL AND PATIENT'S CLINICAL STATUS

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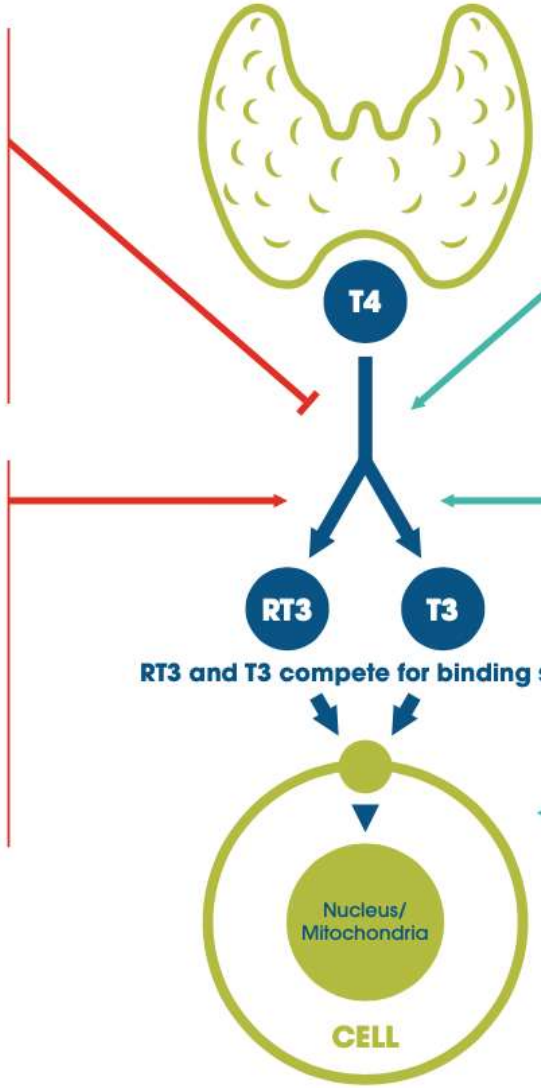
Factors that Affect Thyroid Function



Factors that inhibit proper production of thyroid hormones

- Stress
- Infection, trauma, radiation, medications
- Fluoride (antagonist to iodine)
- Toxins: pesticides, mercury, cadmium, lead
- Autoimmune disease: Celiac

Factors that contribute to proper production of thyroid hormones

- Nutrients: iron, iodine, tyrosine, zinc, selenium vitamin E, B2, B3, B6, C, D

Factors that increase conversion of T4 to RT3

- Stress
- Trauma
- Low-calorie diet
- Inflammation (cytokines, etc.)
- Toxins
- Infections
- Liver/kidney dysfunction
- Certain medications

Factors that increase conversion of T4 to T3

- Selenium
- Zinc

Factors that improve cellular sensitivity to thyroid hormones

- Vitamin A
- Exercise
- Zinc

Key Nutrients to Consider in Thyroid Regulation

- **Selenium**
- Zinc
- Iron
- Iodine
- Vitamin A
- Vitamin D

Treatment Strategies

- **Foundational:**
  - Dietary/Lifestyle changes to remove triggers and/or address mediators
  - Supplementation-nutrients, phytonutrients, medications

- **Hormone Replacement**

EPISODE #126

HASHIMOTO'S AIP MEDICAL STUDY

WITH DR. ROB ABBOTT & ANGIE ALT

DR. ROB ABBOTT

ANGIE ALT

PHOENIX HELIX PODCAST