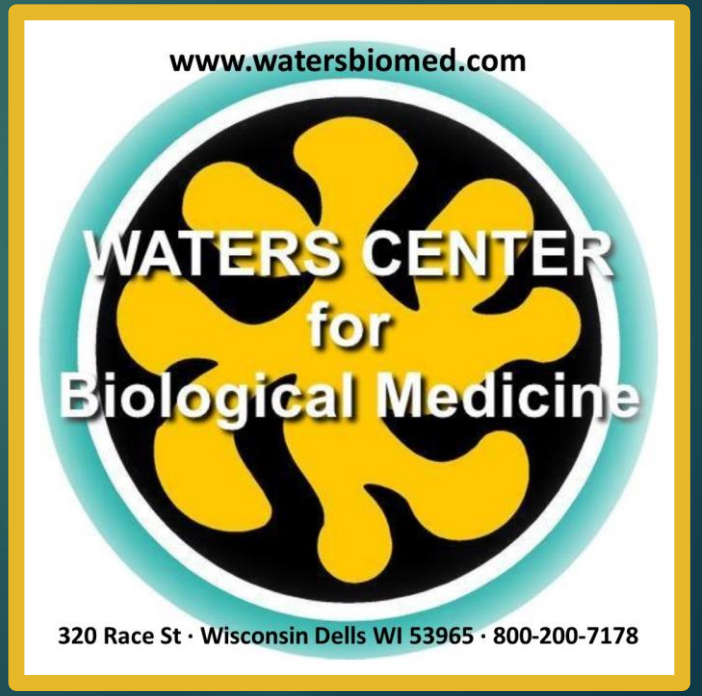
Prediabetic Epidemic

IS THIS WHY I'M TIRED AND OVERWEIGHT?

Robert S. Waters, M.D.



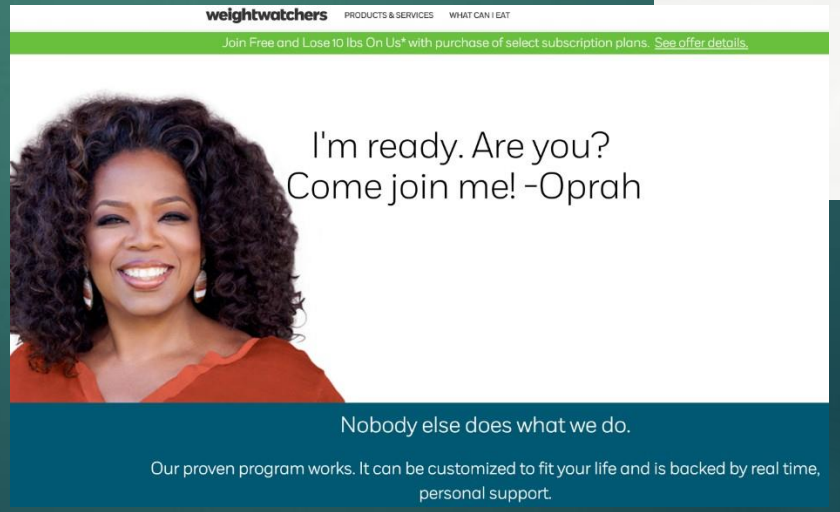
Most of us want more energy and to be our ideal weight…but health is so much more than that…

Prediabetes leads to Diabetes and ultimately…

- STROKE
- DEMENTIA
- DIALYSIS
- HEART SURGERY
- BLINDNESS
- AMPUTATION
- NURSING HOME

What should we eat?

- Low Fat?
- Low Carb?
- Jenny Craig?
- Weight Watchers?
- Mediterranean?
- Vegetarian?
- Fruititarian?
- Paleo?



Who should we trust?

- American Diabetes Association?
- American Medical Association?
- Food & Drug Association?
- American Heart Association?
- Certified Diabetic Educators?
- TV Doctors?
- Dieticians & Nutritionists?
- Our ancestors?

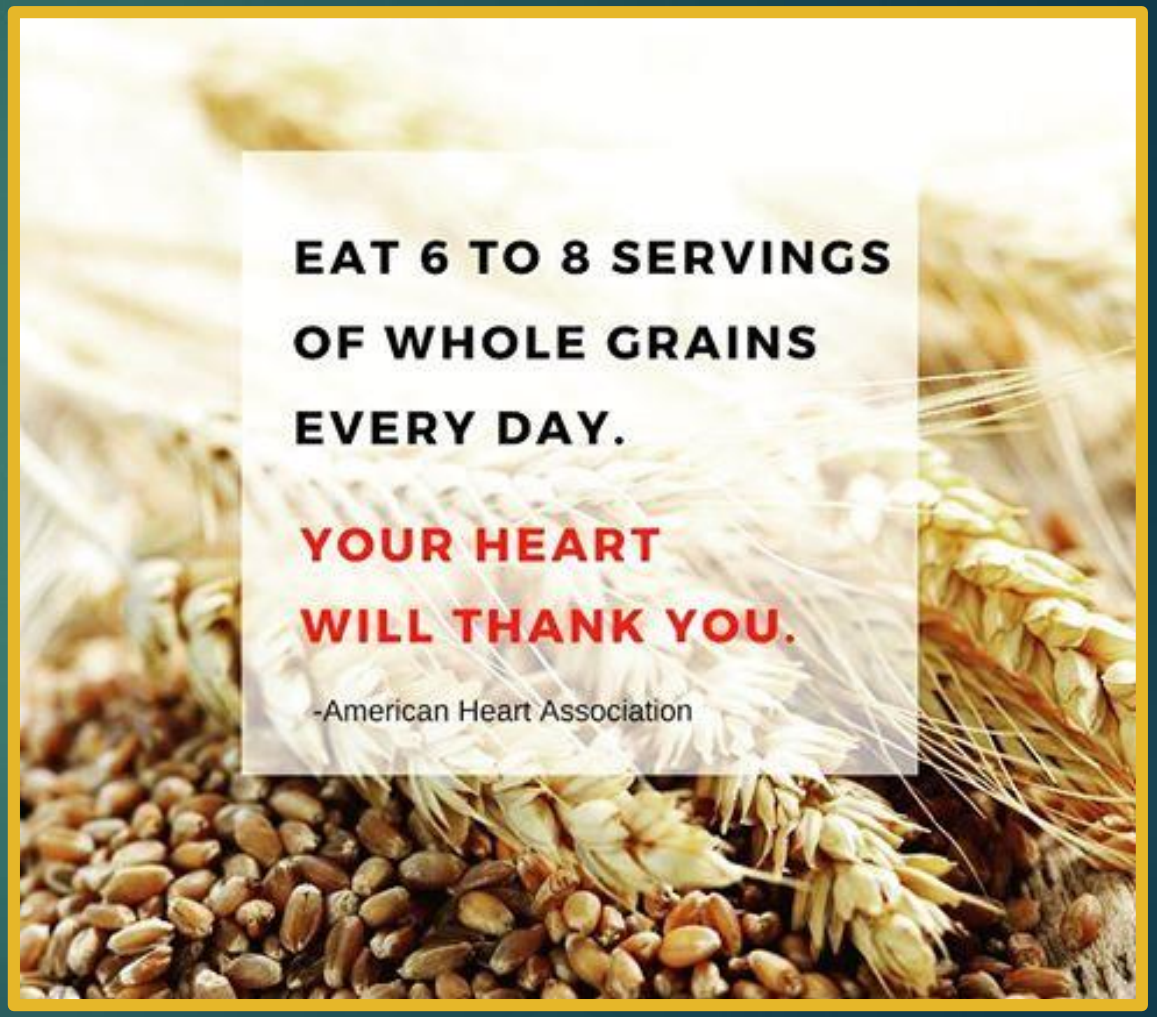
If you heard it from:

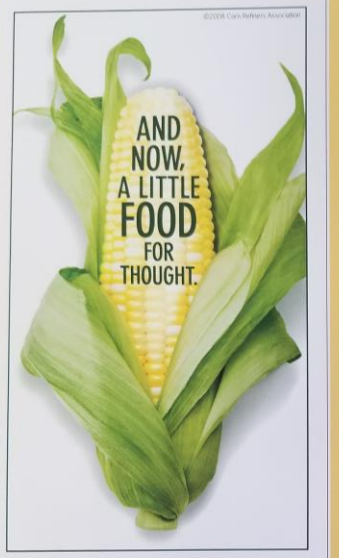
- A Federal Agency
- A Pharmaceutical / Chemical Company
- The American Medical Association or an affiliate
- The Mass Media

You should question it

and probably run away!


Diet & Health
TIME
THE WEEKLY NEWSMAGAZINE
PHYSIOLOGIST
ANCEL KEYS


EAT 6 TO 8 SERVINGS
OF WHOLE GRAINS
EVERY DAY.
YOUR HEART
WILL THANK YOU.
-American Heart Association



A little sweetness in life is good. And what sweetens lots of our favorite foods and beverages are sugars made from corn, including high fructose corn syrup. It's nutritionally the same as table sugar. Has the same number of calories, too. But like many foods, sweeteners should be enjoyed in moderation. Please visit www.SweetSurprise.com and learn the facts. You're in for a Sweet Surprise!

CORN REFINERS ASSOCIATION

Get the facts at www.SweetSurprise.com

"Because the composition of HFCS and sucrose are so similar, particularly on absorption by the body, it appears unlikely that HFCS contributes more to obesity or other conditions than sucrose."

American Medical Association
Report 3 of the Council on Science and Public Health (A-08)
June 2008

"Thus, it is the position of The American Dietetic Association that consumers can safely enjoy a range of nutritive and nonnutritive sweeteners when consumed in a diet that is guided by current federal nutrition recommendations ... as well as individual health goals."

American Dietetic Association
Use of Nutritive and Nonnutritive Sweeteners,
Journal of the American Dietetic Association
February 2004

In 1983, the U.S. Food and Drug Administration (FDA) formally listed high fructose corn syrup as safe for use in food and reaffirmed that decision in 1996. In its 1996 ruling, the FDA noted that "the saccharide composition (glucose to fructose ratio) of HFCS is approximately the same as that of honey, invert sugar and the disaccharide sucrose [table sugar]."

Food and Drug Administration
Federal Register
August 23, 1996

CORN REFINERS ASSOCIATION

November 11, 2008

Dear American Medical Association Member:

Because you are a trusted source of information about nutrition and health matters, we would like to provide you with the latest science on high fructose corn syrup. As you are aware, there has been a significant amount of media attention directed at this versatile sweetener made from corn. Unfortunately, many stories have reported information that lacks scientific merit.

As a result, the Corn Refiners Association has launched a multimedia campaign to communicate the facts about high fructose corn syrup and provide balanced information to allow consumers to make informed decisions based on science.

High Fructose Corn Syrup Summary Points:

- High fructose corn syrup has the same calories and sweetness as table sugar and honey. Nutritionally, they are all the same. All three sweeteners contain nearly the same one-to-one ratio of two sugars—fructose and glucose—either as monosaccharides or disaccharides.
- Many confuse pure "fructose" with "high fructose corn syrup," a sweetener that never contains fructose alone, but always in combination with an essentially equivalent amount of a second sugar (glucose). Recent studies that have examined pure fructose—often at abnormally high levels—have been inappropriately applied to high fructose corn syrup and have caused significant consumer confusion.
- The American Medical Association recently concluded that "high fructose syrup does not appear to contribute to obesity more than other caloric sweeteners."
- The U.S. Food and Drug Administration (FDA) recently stated, referring to a process commonly used by the high fructose corn syrup industry, that it "would not object to the use of the term 'natural' on a product containing HFCS produced by [that] manufacturing process...."
- In 1983, FDA formally listed high fructose corn syrup as safe for use in food and reaffirmed that decision in 1996.

We hope you find the enclosed information helpful. We also invite you to visit our website **www.HFCSfacts.com** and click on the **Health Professionals** button to receive timely updates.

Please feel free to contact us if we may be of assistance by providing additional information about the products made from corn.

Thank you for your consideration.

Sincerely,

Audrae Erickson
President
Corn Refiners Association

Sincerely,

James M. Rippe, M.D.
Cardiologist
Professor of Biomedical Sciences
University of Central Florida

Sincerely,

Kristine Clark, Ph.D., R.D., F.A.C.S.M.
Director of Sports Nutrition
Pennsylvania State University

1701 Pennsylvania Avenue, N.W. • Suite 950 • Washington, D.C. 20006-5806 • (202) 331-1634 • Fax (202) 331-2054 • www.SweetSurprise.com

CORN REFINERS ASSOCIATION

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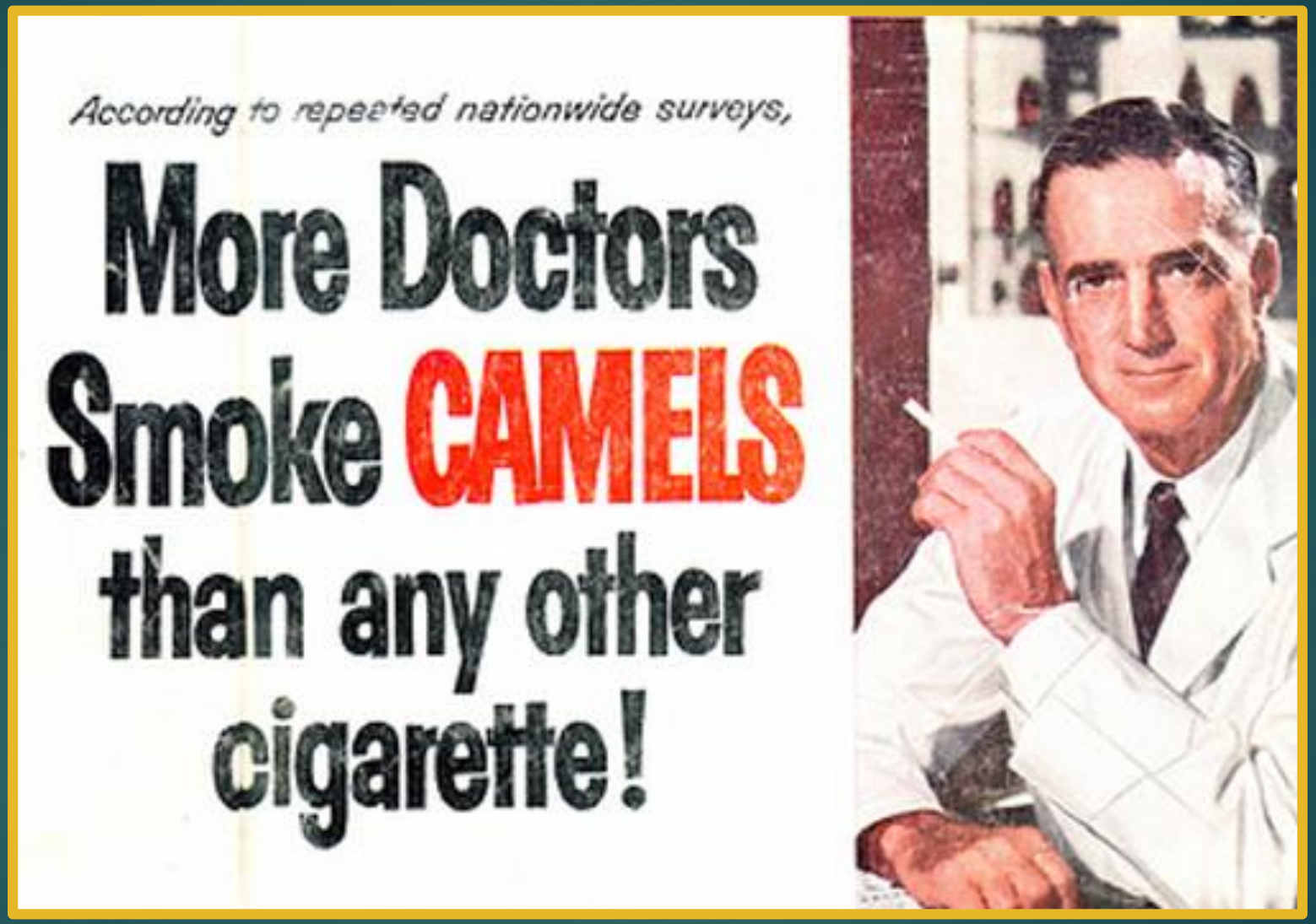

According to repeated nationwide surveys,
More Doctors
Smoke CAMELS
than any other
cigarette!

Sugar Advertising

Woman's Day. 1971

Life Magazine. 1969

The U.S. government is poised to withdraw longstanding warnings about cholesterol

The Washington Post

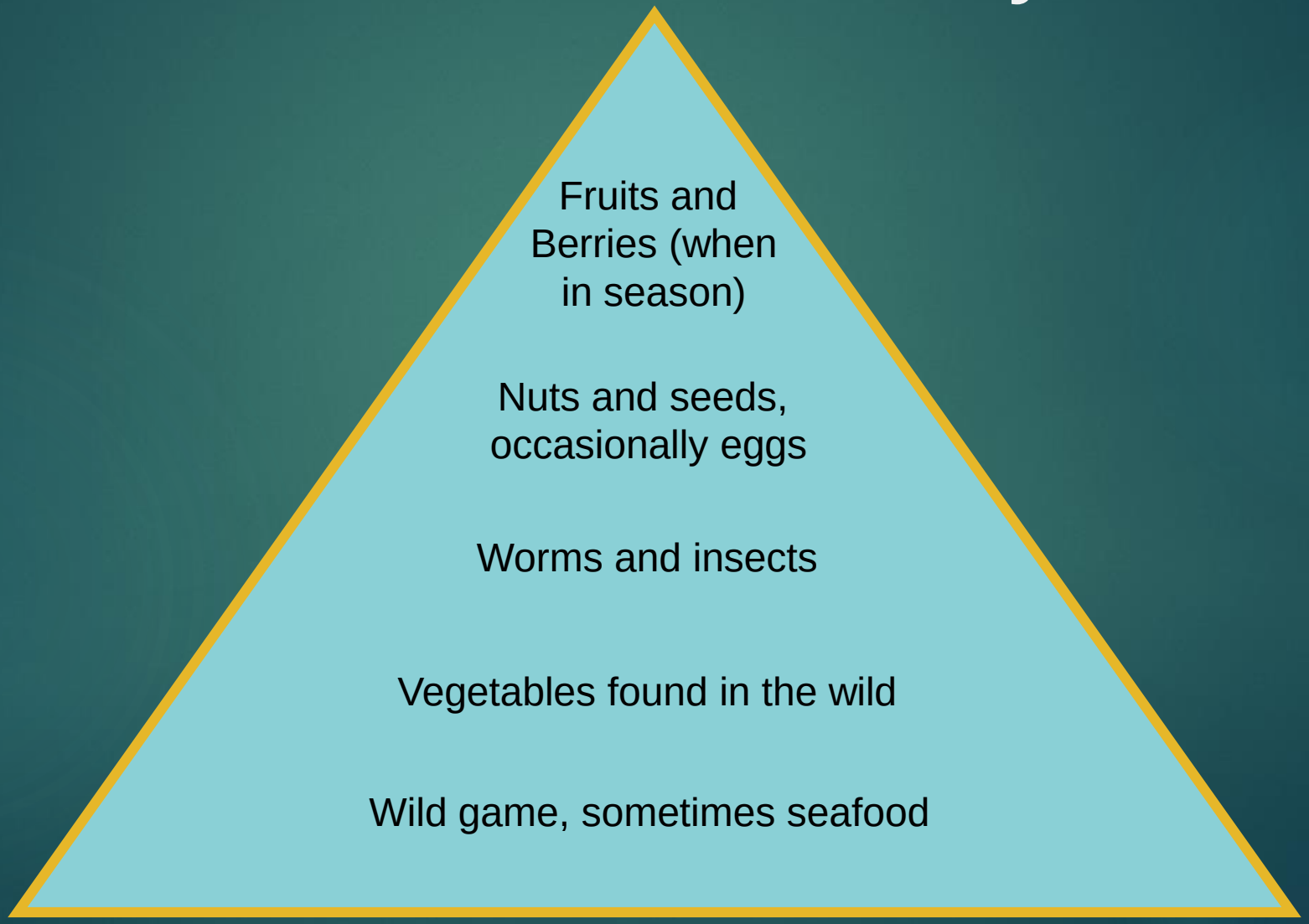
By Peter Whoriskey
February 10, 2015

Remember this guy…he was wrong…

Where does the real evidence come from?

- Stomach contents preserved in bogs bodies.
- Archaeological Data
- Historical Documents – Tacitus
- Analysis of Modern Hunter-Gatherer Cultures
- Existing Traditional Cultures

The Paleolithic Food Pyramid



“Current carbohydrates often take the form of sugars and sweeteners… Products of this sort, together with items made from highly refined grain flours, constitute empty calories… devoid of accompanying essential amino and fatty acids, vitamins, minerals, and possibly phytochemicals.”

~ S.B. Eaton, M. Shostak, et al., 1988

“The fiber in pre-agricultural diets came almost exclusively from fruits, roots, legumes, nuts, and other naturally occurring non-cereal plant sources, so it was less associated with phytic acid than is fiber from cereal grans (phytic acid interferes with absorption).”

~ S.B. Eaton

“Their nomadic foraging lifestyle required vigorous physical exertion, and skeletal remains indicate that they were typically more muscular than we are today. Life during the agricultural period was also strenuous, but industrialization has progressively reduced obligatory physical exertion.”

~ S.B. Eaton & M. Konner, 1983

The differences Eaton et al. found between paleolithic and our modern diets:

- They ate NO refined carbohydrates.
- They ate a high fiber diet.
- Their foraging lifestyle required vigorous exertion.

We forage mostly in our refrigerators and grocery store isles

Paleo nutrients

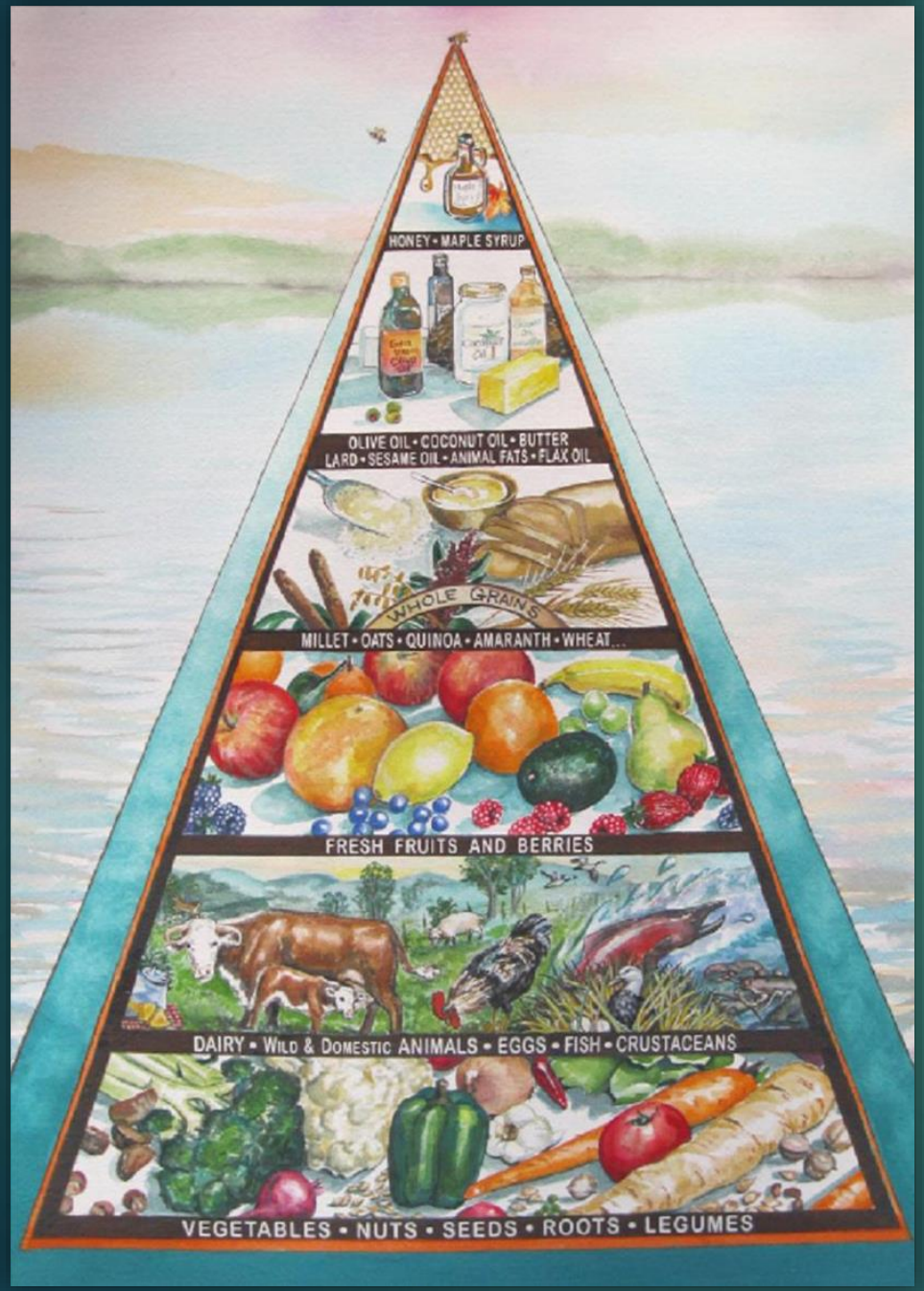
Nutrients	Paleolithic Intake	RDA	Current U.S. Intake
Vitamin C	604 mg	60mg	77-109mg
Vitamin E	33 mg	8-10 mg	7-10 mg
Calcium	1,956 mg	800-1,200 mg	750 mg
Magnesium	700 mg	350 mg	250 mg
Potassium	10.5 g.	3.5 g.	2.5 g
Zinc	43 mg	12-15 mg	5-14 mg
Fiber	50-104 g.	25-35 g.	10 g.

** Amount recommended by the American Dietetic Association

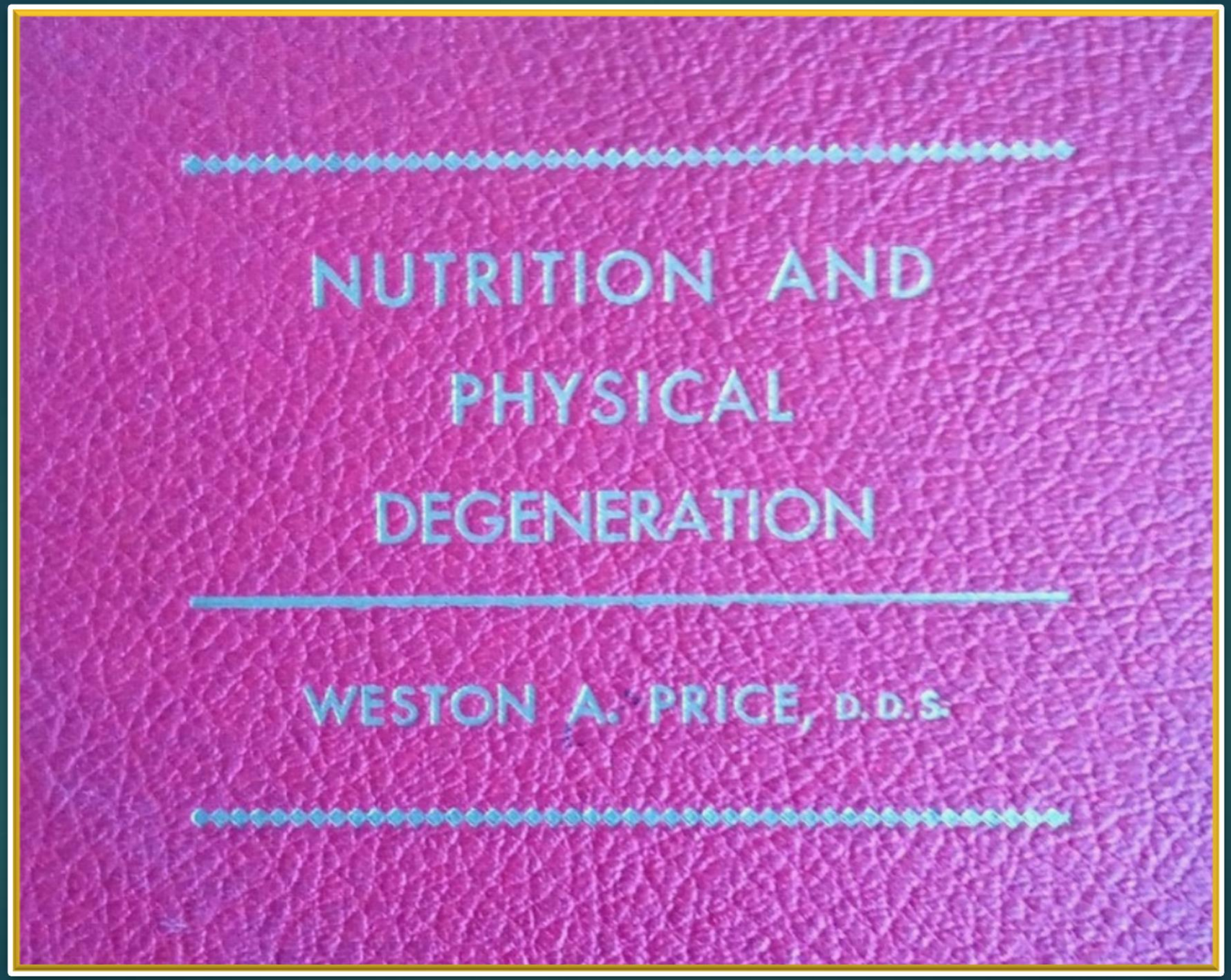
Composition of commonly eaten foods

(per 4oz as a percent of fatty acids)

	Grainfed Beef	Grassfed Beef	Wild Venison	Wild Fish
Protein (grams)	16	17	35	19-26
Omega-3's (% of Total Fatty Acid)	0.4%	2.8%	4.8%	2-4%



- The foundation of our Ancestral food pyramid is based on abundant use of colorful pigmented vegetables, roots, tubers, nuts and seeds and high in a variety of fibers.
- Animal based foods were sacred and great care was taken to utilize the entire animal.
- Fats were not considered toxic. Eulachon oil from the Candlefish was the most important product traded into the interior; as a result, the trails over which the trade was conducted came to be known as grease trails.
- Fruits and sweets were consumed seasonally and sparingly and from natural sources such as maple and honey.


NUTRITION AND
PHYSICAL
DEGENERATION
WESTON A. PRICE, D.D.S.

Weston A. Price

A Comparison of Primitive and Modern Diets and Their Effects

BY

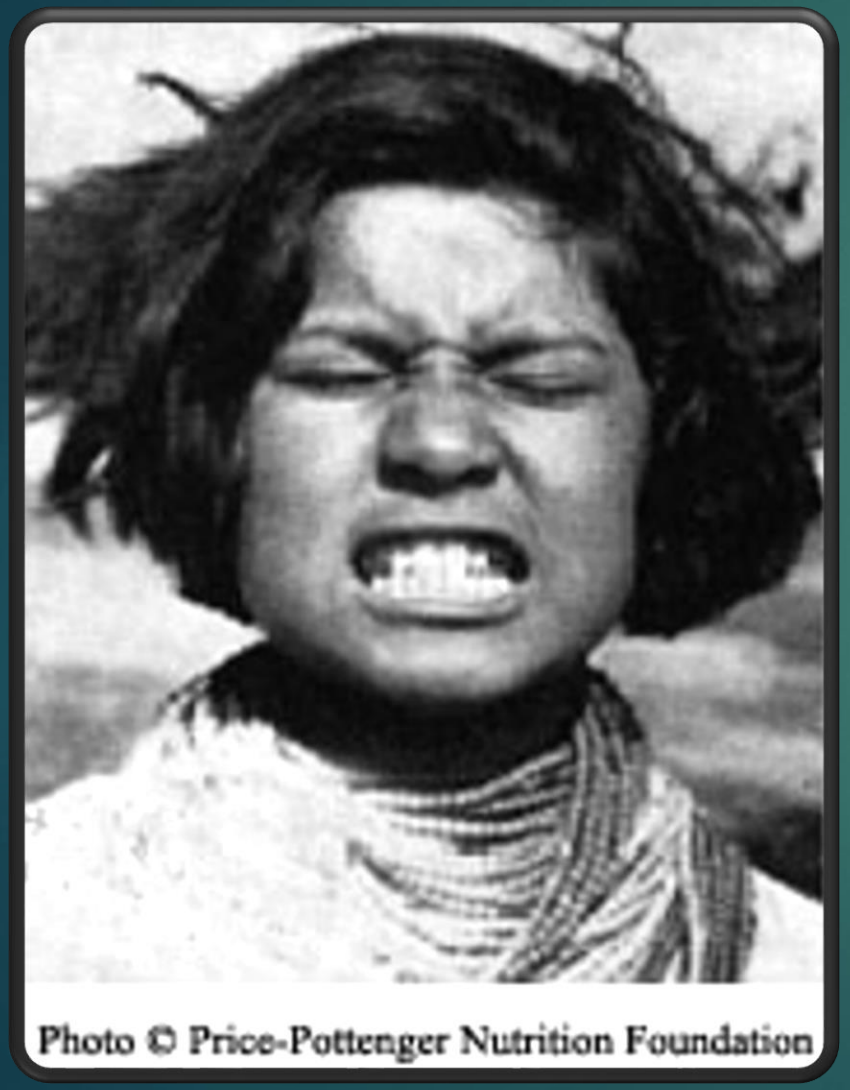
WESTON A. PRICE, M.S., D.D.S., F.A.C.D.

Member Research Commission, American Dental Association
Member American Association of Physical Anthropologists
Honorary Member of the International Mark Twain Society
Honorary Fellowship in the International College of Dentists
Honorary Member of the American Academy of Applied Nutrition
Author "Dental Infections, Oral and Systemic"
"Dental Infections and the Degenerative Diseases"

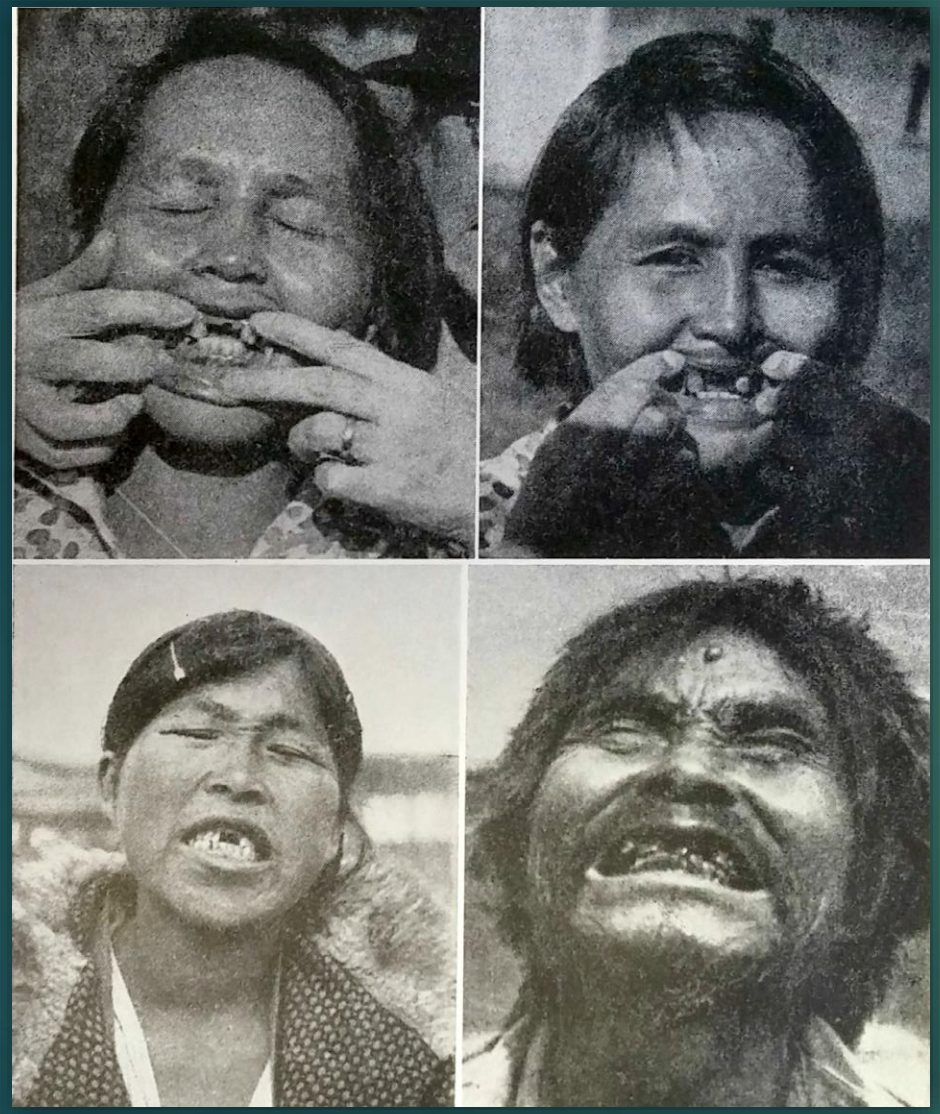
Why are these People so Healthy?

Native people eating traditional foods had physical excellence, splendid facial and dental arch forms, and no cavities.

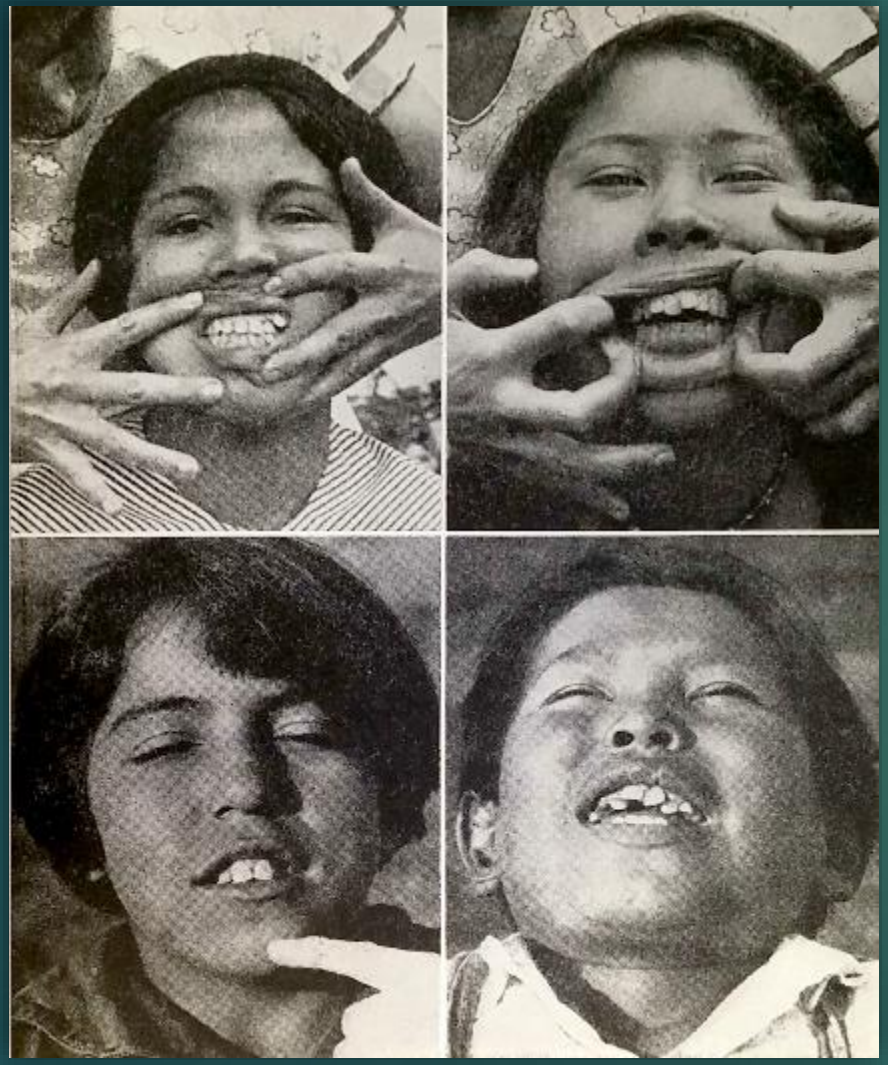
Ancestral Diet



Modern "Processed Food" Diet

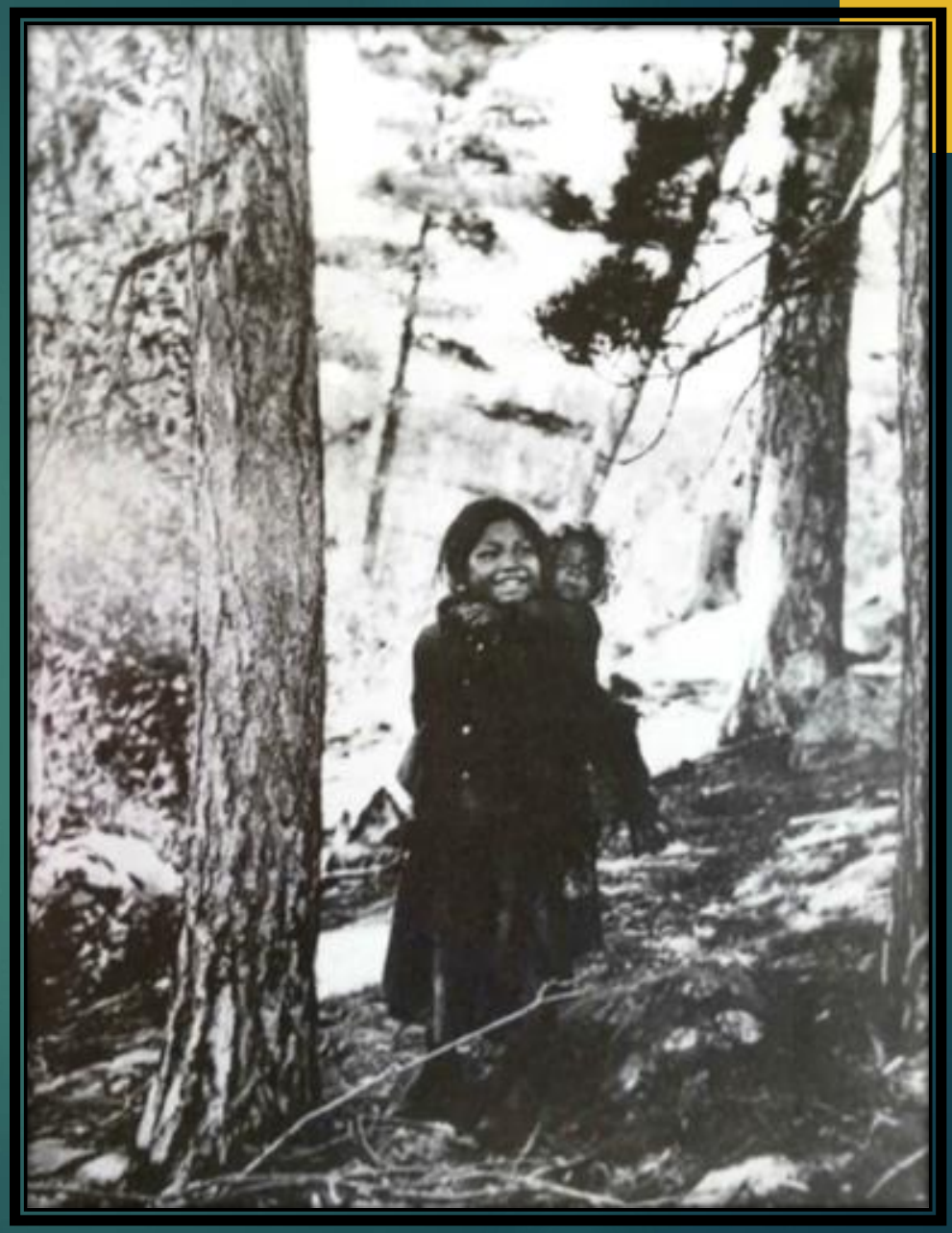


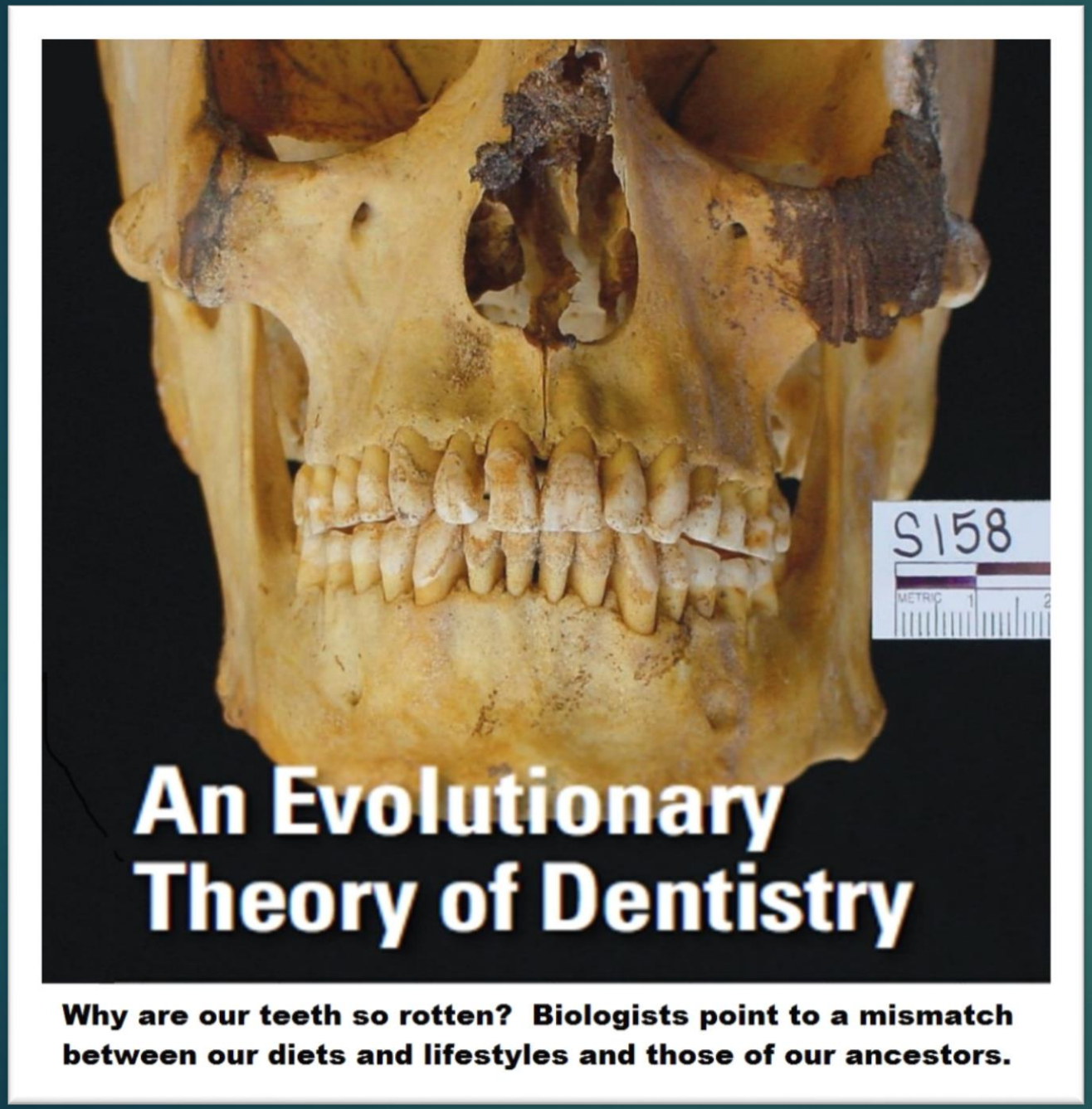
Whenever they had access to the modern foods of commerce the dental conditions were extremely bad. These four individuals are typical.



“The blight of the white man’s commerce is seen everywhere in the distorted countenance of even the first generation after the adoption by the parents of the foods of modern commerce”.


NFECTIONS AND TOBACCO'S.
ICE - CREAM.





Healthy bite. This ancient Egyptian had healthier teeth and jaws than most living humans.

www.sciencemag.org SCIENCE VOL 336 25 MAY 2012

Not-so-sweet tooth. This German jaw from the 16th to 18th century shows the perils of a poor diet in tooth loss, cavities, and gum disease

Perils of a sweet tooth

In Europe, less than 10% of individuals had cavities until Alexander the Great brought sugar to Greece in the 4th century B.C.E., according to earlier studies, says pediatric dentist Kevin Boyd of Children's Memorial Hospital in Chicago, Illinois. Cavities increased first in Greece, then Rome; their incidence also rose throughout Europe in the Middle Ages. But the biggest spike was from 1800 to 1850, when Britain took control of the West Indies and imported far more sugar than previously. Sugar helped fuel the Industrial Revolution, which was a transition from an agriculture-based economy to a machine-based economy. In 1874, the British reduced the tax on sugar, and it became available to all social classes. "In London, mostly 1800 onwards, they have absolutely dreadful teeth," Hillson says.

www.sciencemag.org SCIENCE VOL 336 25 MAY 2012

What Animals did the Celtic people eat during the Iron Age?

- Wild Animals: deer, wild boar, fox, beaver and bear.
- Fish: trout, mackerel and salmon.
- Domesticated animals: chicken, goat, sheep and pigs.
- Eggs from hens and wild bird eggs.

What Plants did the Celtic people eat during the Iron Age?

- Vegetables: leeks, onions, turnips, parsnips and carrots.
- Wild nuts: hazelnuts and walnuts.
- Berries: gooseberries, blackberries, blueberries.
- Grains for bread or porridge
- Herbs: fennel, common sorrel, wild garlic, parsley
- Leaves: nettles and spinach

How the Mid-Victorians Worked, Ate and Died †

Paul Clayton [1,*] and Judith Rowbotham [2]

[1] School of Life Sciences, Oxford Brookes University, UK

[2] Department of History & Law, Nottingham Trent University, UK; E-Mail: jrowbotham@gmail.com

* Author to whom correspondence should be addressed; E-Mail: paulrclayton@gmail.com

† This paper is an extended re-working of three papers published in the *Journal of the Royal Society of Medicine* [1-3].

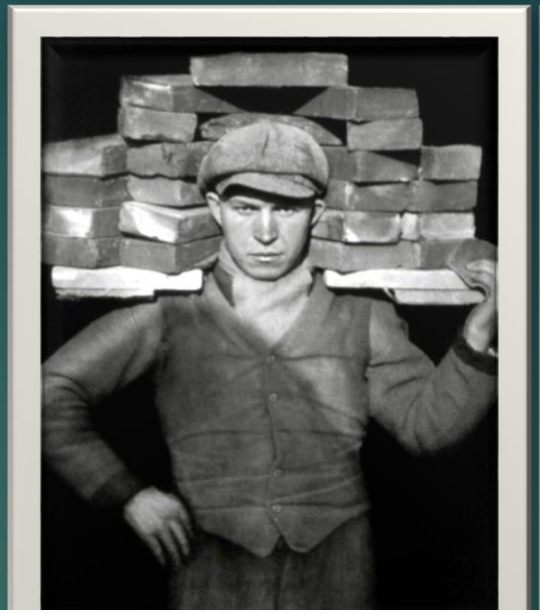
Received: 9 February 2009 / Accepted: 28 February 2009 / Published: 20 March 2009

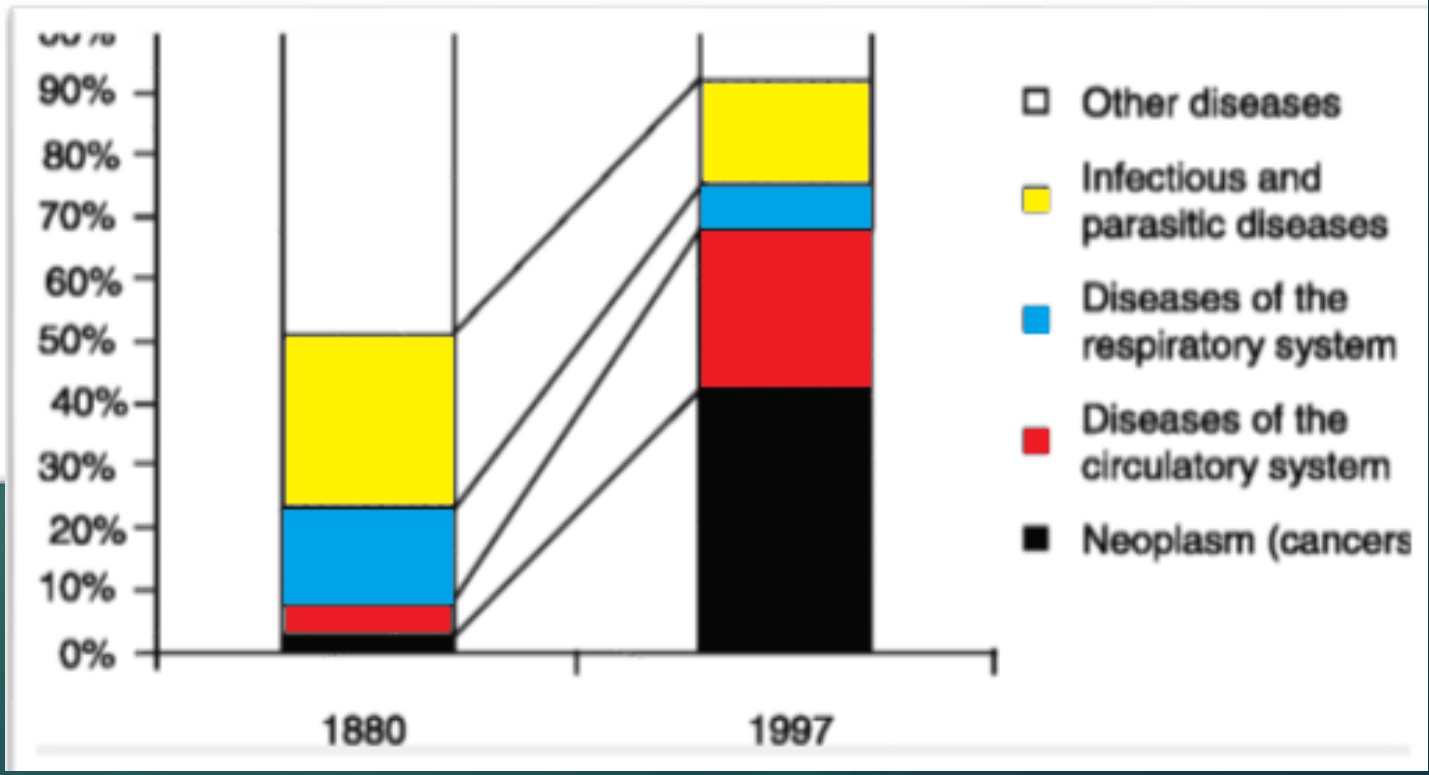
Abstract: Analysis of the mid-Victorian period in the U.K. reveals that life expectancy at age 5 was as good or better than exists today, and the incidence of degenerative disease was 10% of ours. Their levels of physical activity and hence calorific intakes were approximately twice ours. They had relatively little access to alcohol and tobacco; and due to their correspondingly high intake of fruits, whole grains, oily fish and vegetables, they consumed levels of micro- and phytonutrients at approximately ten times the levels considered normal today. This paper relates the nutritional status of the mid-Victorians to their freedom from degenerative disease; and extrapolates recommendations for the cost-effective improvement of public health today.

Keywords: Public health; dietary shift; degenerative disease; Victorian.

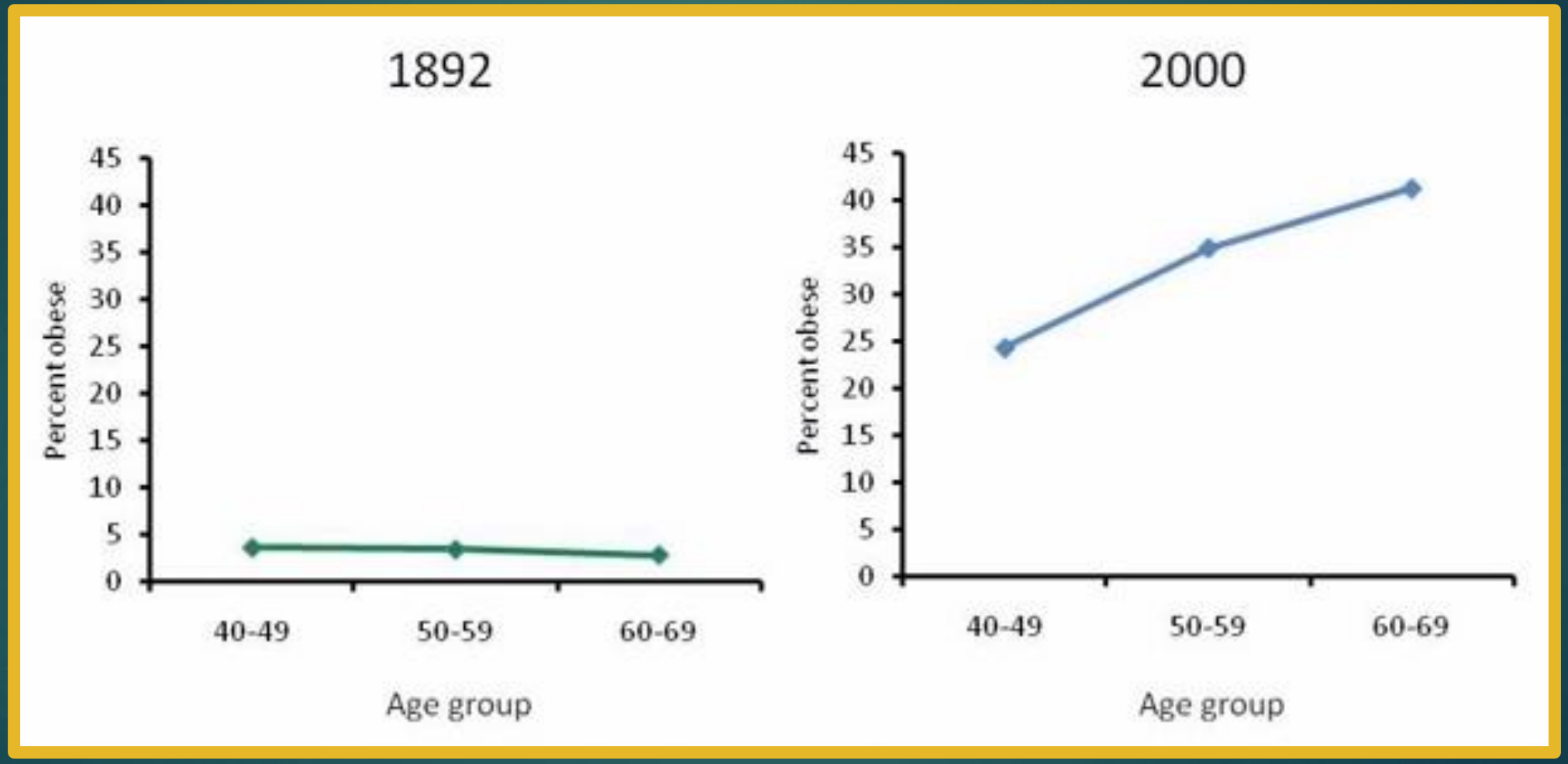
Figure 1. 'Moulders' at the Murston brickfields. The 'moulders' shaped clay into bricks, each man making close on 1,000 every hour for an 8½ hour day and a 58 hour week. One brickie is on record as having made 986,091 bricks between April and September.

In short, the mid-Victorians ate twice as much as we do, but due to their high levels of physica ctivity remained slim; overweight and obesity were relatively rare, and (unless associated with ill ealth) were generally identified as phenomenona associated with the numerically smaller middle an pper-middle class. But it is not just the amount of food the mid-Victorians consumed that is s nfamiliar; the composition of their diet was also very different from our own.





Percent of population with obesity



Dramatic Increase in Heart Disease in the last century

- Sir William Osler, MD, the founder of the field of Internal Medicine, first proposed the cause for heart disease to be infection in 1908. He described angina pectoris as a rare disease.
- Paul Dudley White, MD, President Eisenhower's doctor, graduated Medical School in 1914 and saw only two cases of coronary artery disease in training. He stated that "until the first 2-3 decades of the 20th century, coronary heart disease was rare and not missed by ignorance."
- James B Herrick, MD, first describes heart disease resulting from hardening of the arteries in 1912.

BJSM Online First, published on April 25, 2017 as 10.1136/bjsports-2016-097285

Saturated fat does not clog the arteries: coronary heart disease is a chronic inflammatory condition, the risk of which can be effectively reduced from healthy lifestyle interventions

Aseem Malhotra,[1] Rita F Redberg,[2,3] Pascal Meier[4,5]

Coronary artery disease pathogenesis and treatment urgently requires a paradigm shift. Despite popular belief among doctors and the public, the conceptual model of dietary saturated fat clogging a pipe is just plain wrong. A landmark systematic review and meta-analysis of observational studies showed no association between saturated fat consumption and (1) all-cause mortality, (2) coronary heart disease (CHD), (3) CHD mortality, (4) ischaemic stroke or (5) type 2 diabetes in healthy adults.[1] Similarly in the secondary prevention of CHD there is no benefit from reduced fat, including saturated fat, on myocardial infarction, cardiovascular or all-cause mortality.[2] It is instructive to note that in an angiographic study of postmenopausal women with CHD, greater intake of saturated fat was associated with less progression of atherosclerosis whereas carbohydrate and polyunsaturated fat intake were associated with greater progression.[3]

PREVENTING THE DEVELOPMENT OF ATHEROSCLEROSIS IS IMPORTANT BUT IT IS ATHEROTHROMBOSIS THAT IS THE REAL KILLER

The inflammatory processes that contribute to cholesterol deposition within the artery wall and subsequent plaque formation (atherosclerosis), more closely resembles a 'pimple' (figure 1). Most cardiac events occur at sites with <70% coronary artery obstruction and these do not generate ischaemia on stress testing.[4] When plaques rupture (analogous to a pimple bursting), coronary thrombosis and myocardial infarction can occur within minutes. The limitation of the current plumbing approach ('unclogging a pipe') to the management of coronary disease is revealed by a series of randomised controlled trials (RCTs) which prove that stenting significantly obstructive stable lesions fail to prevent myocardial infarction or to reduce mortality.[5]

DIETARY RCTS WITH OUTCOME BENEFIT IN PRIMARY AND SECONDARY PREVENTION

In comparison with advice to follow a 'low fat' diet (37% fat), an energy-unrestricted Mediterranean diet (41% fat) supplemented with at least four tablespoons of extra virgin olive oil or a handful of nuts (PREDIMED) achieved a significant 30% (number needed to treat (NNT)=61) reduction in cardiovascular events in over 7500 high-risk patients. Furthermore, the Lyon Heart study showed that adopting a Mediterranean diet in secondary prevention improved hard outcomes for both recurrent myocardial infarction (NNT=18) and all-cause mortality (NNT=30), despite there being no significant difference in plasma low-density lipoprotein (LDL) cholesterol between the two groups. It is the alpha linoleic acid, polyphenols and omega-3 fatty acids present in nuts, extra virgin olive oil, vegetables and oily fish that rapidly attenuate inflammation and coronary thrombosis.[6] Both control diets in these studies were relatively healthy, which make it highly likely that even larger benefits would be observed if the Mediterranean diets discussed above were compared with a typical western diet.

LDL CHOLESTEROL RISK HAS BEEN EXAGGERATED

Decades of emphasis on the primacy of lowering plasma cholesterol, as if this was an end in itself and driving a market of 'proven to lower cholesterol' and 'low-fat' foods and medications, has been misguided. Selective reporting may partly explain this misconception. Reanalysis of unpublished data from the Sydney Diet Heart Study and the Minnesota coronary experiment cereal replacing saturated fat with linoleic acid containing vegetable oils increased mortality risk despite significant reductions in LDL and total cholesterol (TC).[7]

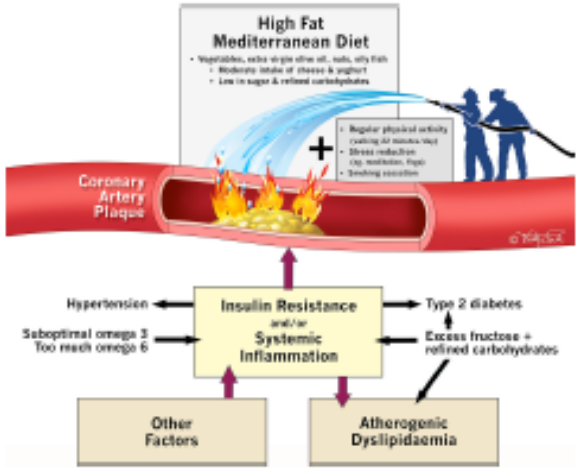


Figure 1 Lifestyle interventions for the prevention and treatment of coronary disease.

[1]Lister Hospital, Academy of Medical Royal Colleges, Stevenage, UK
[2]Philip R Lee Institute for Health Policy Studies, San Francisco, California, USA
[3]Department of Medicine, UCSF School of Medicine, San Francisco, California, USA
[4]Department of Cardiology, University Hospital Geneva, Geneva, Switzerland
[5]Department of Cardiology, University College London, London, UK

Correspondence to Dr Aseem Malhotra, Lister Hospital, Academy of Medical Royal Colleges, Stevenage, UK; aseem_malhotra@hotmail.com

Another risk factor for CHD is environmental stress. Childhood trauma can lead to an average decrease in life expectancy of 20 years. Chronic stress increases glucocorticoid receptor resistance, which results in failure to down regulate the inflammatory response. Combining a complete lifestyle approach of a healthful diet, regular movement and stress reduction will improve quality of life, reduce cardiovascular and all-cause mortality.[10] It is time to shift the public health message in the prevention and treatment of coronary artery disease away from measuring serum lipids and reducing dietary saturated fat. Coronary artery disease is a chronic inflammatory disease and it can be reduced effectively by walking 22 min a day and eating real food. There is no business model or market to help spread this simple yet powerful intervention.

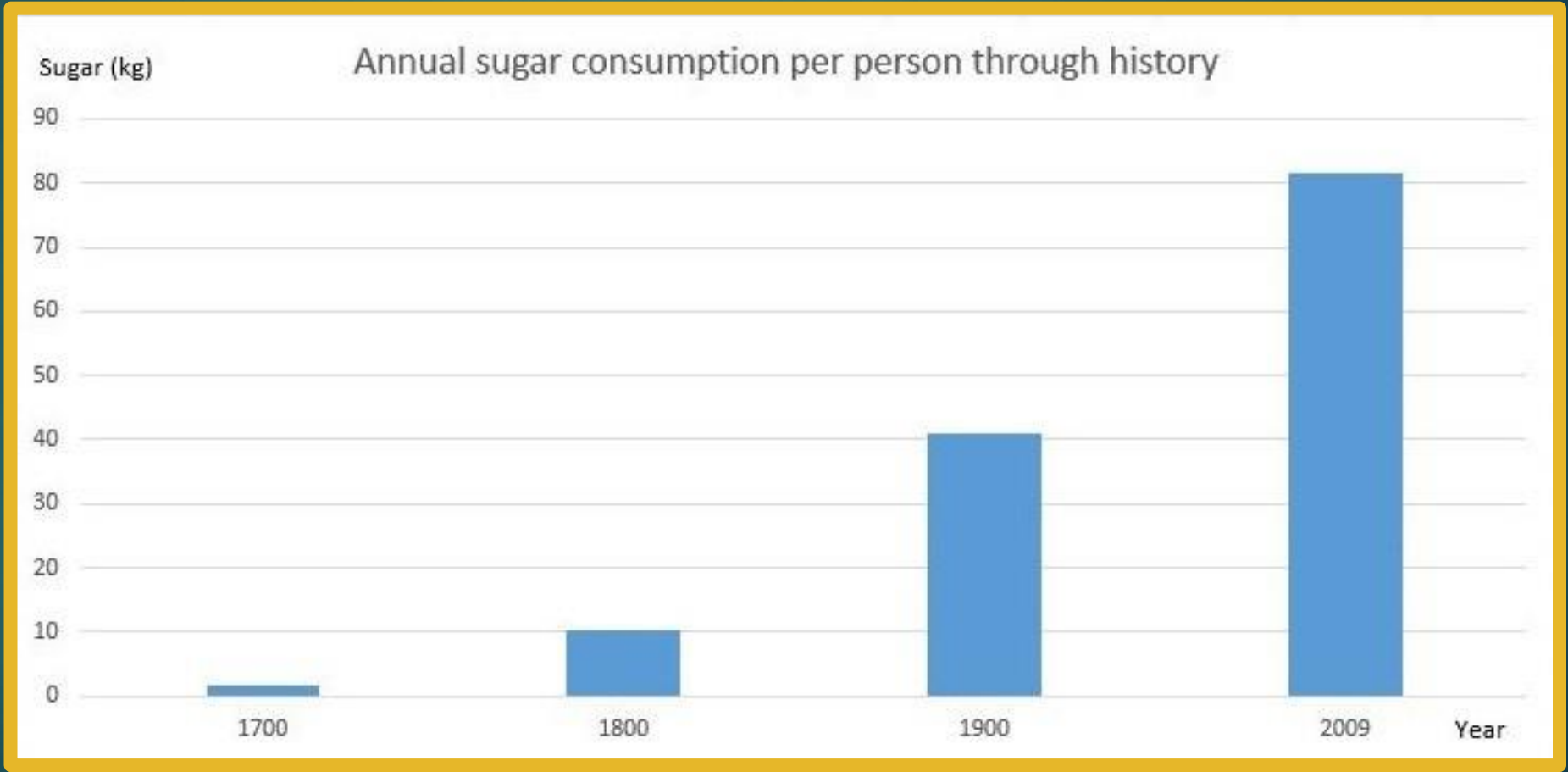
Advice for Diabetics before 1960's

"Foods to be avoided: bread and anything else made with flour... cereals... potatoes and any other white vegetables... foods containing much sugar...

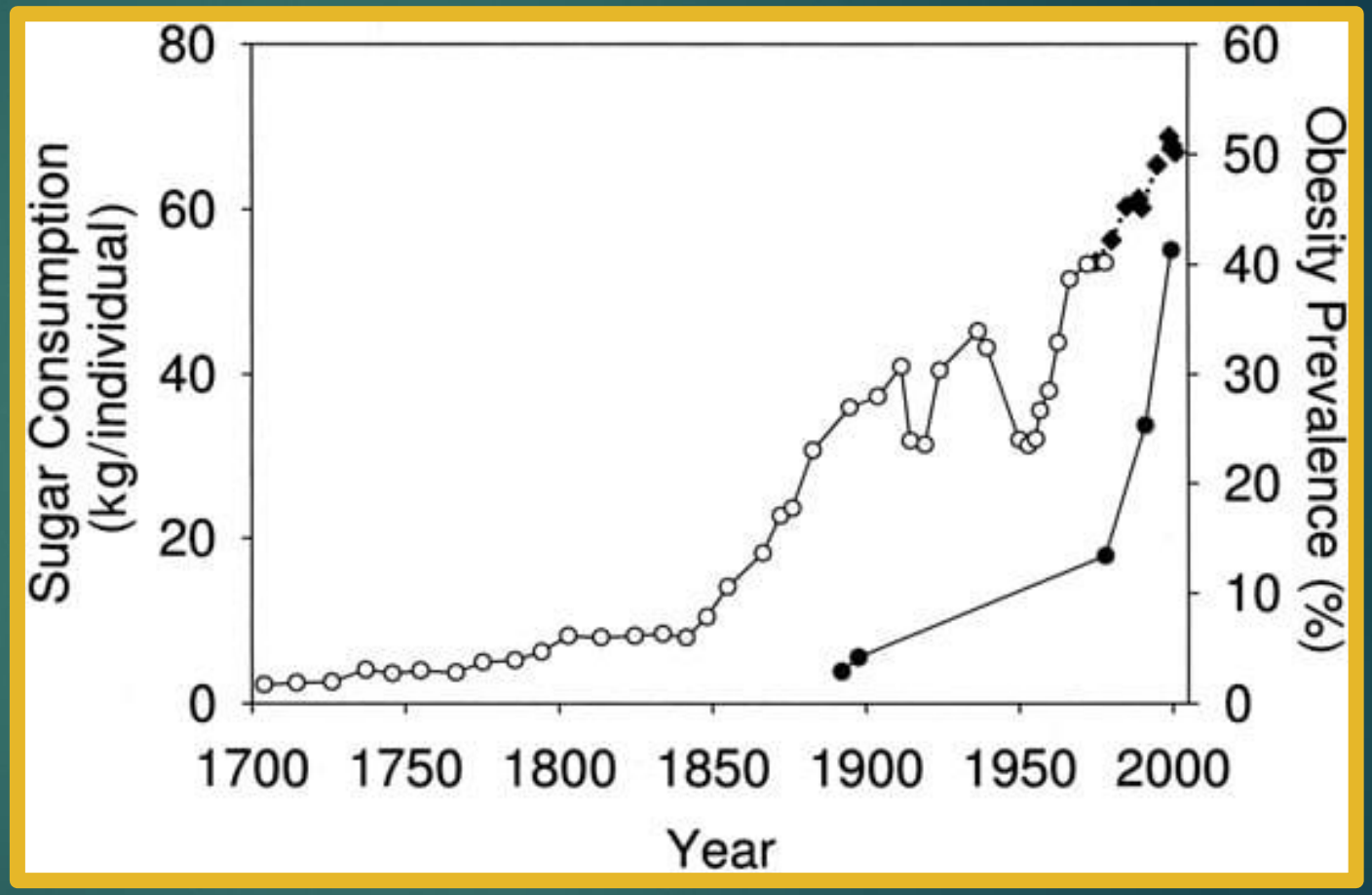
You can eat as much as you like of the following food: meat, fish, birds... green vegetables... eggs... cheese, fruit... except bananas and grapes."

Textbook: The Practice of Endocrinology by Raymond Greene (1951)

Annual sugar consumption per person through history



Total Sugar Intake Has Skyrocketed in The Past 160 Years



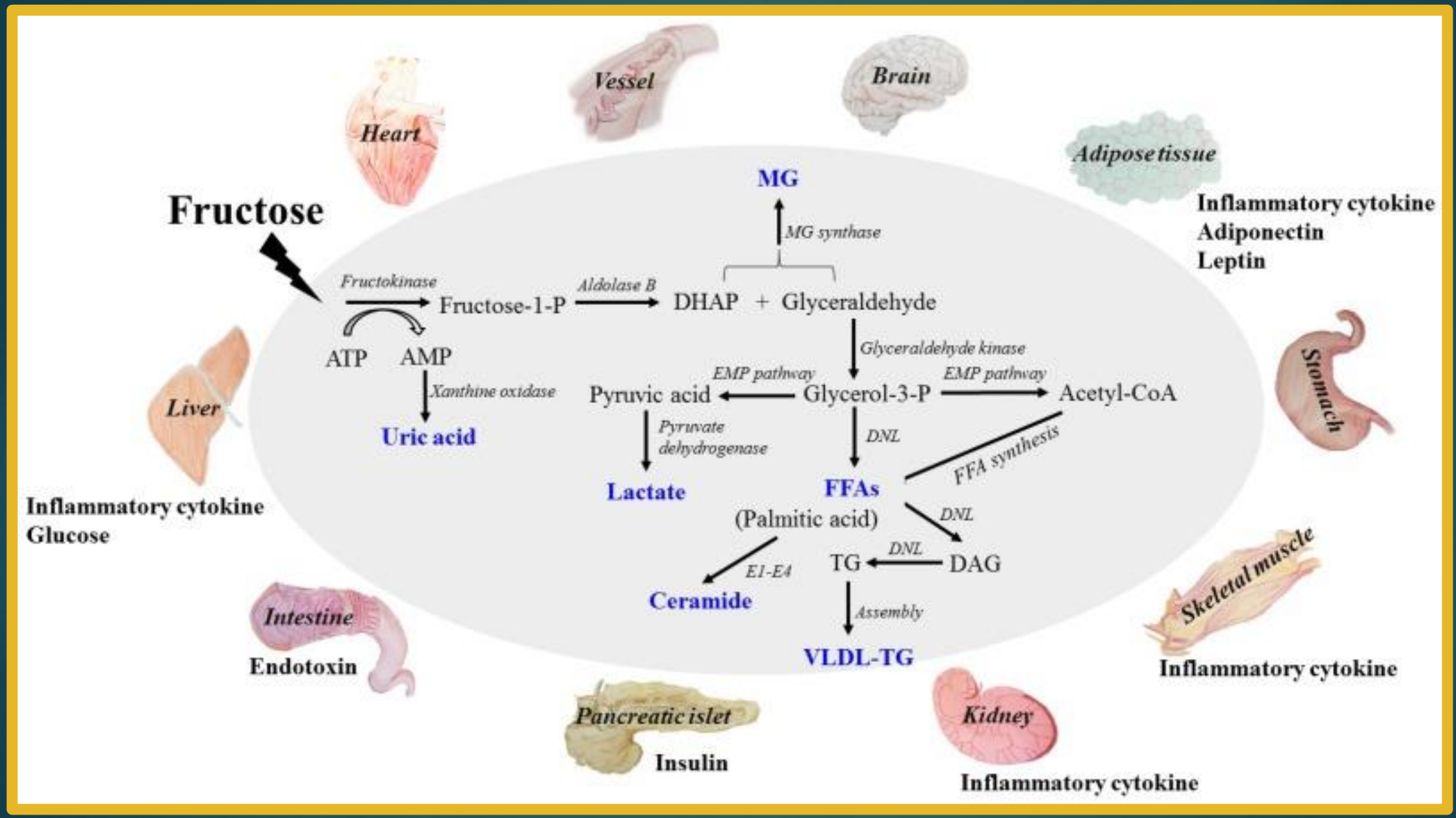
Johnson RJ, et al. Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease. The American Journal of Clinical Nutrition, 2007.

This is called addiction!

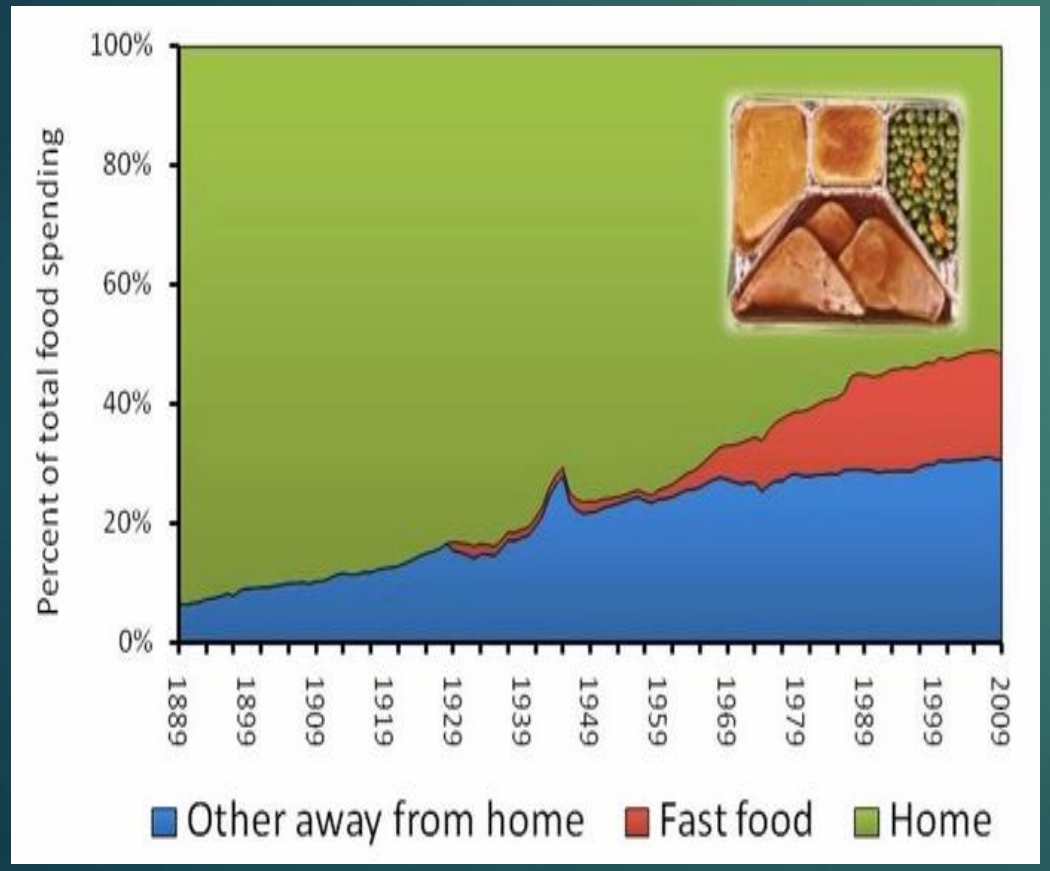
"Compared with glucose ingestion, fructose ingestion attenuates increases in circulating levels of the satiety hormone glucagon-like polypeptide 1 (GLP-1) and does not attenuate levels of ghrelin, an appetite-stimulating hormone. Thus, fructose possibly increases food-seeking behavior and increases food intake."

JAMA, January 2, 2013 – Vol 309, No 1

Therefore…fructose does not satisfy our hunger and increases our desire for more.


Heart
Vessel
Brain
Adipose tissue
Inflammatory cytokine
Adiponectin
Leptin
Fructose
MG
MG synthase
Fructokinase
Fructose-1-P
Aldolase B
DHAP + Glyceraldehyde
ATP
AMP
Xanthine oxidase
Uric acid
Glyceraldehyde kinase
EMP pathway
Pyruvic acid
Glycerol-3-P
EMP pathway
Acetyl-CoA
Liver
Stomach
Pyruvate dehydrogenase
Lactate
DNL
FFA synthesis
FFAs
(Palmitic acid)
DNL
Inflammatory cytokine
Glucose
DNL
E1-E4
TG
DAG
Ceramide
Skeletal muscle
Intestine
Assembly
VLDL-TG
Endotoxin
Inflammatory cytokine
Pancreatic islet
Kidney
Insulin
Inflammatory cytokine

People don’t eat at home anymore…



Coincident with large corporations taking control over a large portion of our food supply

Their budgets allow great influence:

- Huge advertising and public relations budgets.
- Funding of Scientific research

The American Heart Association’s early funding came primarily from Proctor & Gamble which conveniently promoted Crisco in return.

“FOOD-LIKE”

The change to food-like substances from our ancestral diet caused a rapid degeneration of health

(as it did to traditional people when they adopted modern foods and lifestyles)

Food Additives, Altered Food and Water

- Trans oils (hydrogenated vegetable oil)
- Propylene glycol
- Polysorbate 80
- Dyes
  - Blue 2, Yellow 5, Red 40, *etc.*
- “Flavor enhancers” (MSG)
- Avodicarbonide
- Maltodextrin
- Autolyzed Yeast Extract
- Disodium inosinate
- Disodium guanylate
- BHA

Modern Changes in Diet Leads to Disease

- High refined carb diet – especially sugar
- Lack of Exercise
- Deficiencies
  - Magnesium, Chromium, Zinc, Vanadium, Manganese, Omega-3 fatty acids
- Iron Overload
- Lack of plant pigments and other specific molecules in food
- Low Fiber in Diet
- Toxic Metal Accumulation
- Endocrine disruptors and other pollutants

"Diabetes is not a Disease, it is an imbalance"

The Honest Coca-Cola Commercial

Let’s take a moment to recap this history…

- We don’t know whom to trust
- The information keeps changing

However…the FACTS are…

- Our modern diet is very different than our ancestors
- An increase in processed food and sugar consumption has lead to an increase in obesity and heart disease stemming from…

INSULIN RESISTANCE

- Which leads to prediabetes, which leads to diabetes which leads to:

Strokes, Heart Disease, Dialysis, Blindness, Amputations, Dementia and the

NURSING HOME

Are we DOOMED?

No…We can help, but first…

Let’s talk about how these changes have caused a phenomenon called

SYNDROME X and INSULIN RESISTANCE

Gerald Reaven, MD

Endocrinologist and Stanford University Research Scientist

- In 1960, he became interested in DM after Yalour and Berson developed radioimmunoassay to measure insulin. They found that many people with “maturity onset” DM actually had elevated insulin.
- Reaven knew that in 1958, Drs. Albrink and Man demonstrated that triglycerides were just as important as cholesterol in the risk of heart disease.
- In the mid-1960’s, he developed the insulin clamp to change which demonstrated that some people needed a higher infusion of insulin to maintain normal blood sugar compared to others.
- Ahrens and Farquhar showed that high triglycerides were caused NOT by dietary fat but rather too many carbohydrates.
- In 1967, he had showed the insulin stimulates the liver to produce triglycerides and these go up dramatically in people whose muscle and fat tissue becomes resistant to insulin. The pancreas makes more and more insulin to push glucose into the cells. A vicious circle was created of progressively increasing insulin, glucose and triglycerides. The liver then increases its production of VLDL and LDL cholesterol to package the triglycerides to carry them into the blood.

Gerald Reaven, MD

Endocrinologist and Stanford University Research Scientist

- In 1983, Reaven showed that when he gave normal people a 60% carbohydrate diet, triglycerides went up, HDL down and insulin were significantly elevated after the meal.
- Through the 1980's, Reaven's group continued to strengthen the link between elevated sugar, over-production of insulin, elevated triglycerides and LDL, low HDL, hypertension, heart disease, diabetes and carbohydrate intake.
- Even when hypertensive patients have their blood pressure normalized with medication, they continued to manifest insulin resistance.
- He did all of this work over 25 years and was attacked all along the way by "leaders" in the fields of endocrinology and metabolism.
- In 1988, he received the Banting prize for his work and in his acceptance speech, he named the group of related abnormalities "Syndrome X".

Syndrome X = Metabolic Syndrome = Insulin Resistance

- Waist Circumference (BMI)
  - >40 inches in Men
  - >35 inches in Women
- Triglycerides
  - >150
- High Density Cholesterol
  - <40 in Men
  - <50 in Women
- Blood Pressure
  - >140/90
- Insulin Resistance
  - Fasting Glucose >100

Definitions (WHO)

As We See It

Blood Sugar Levels Surge to Record Highs

BY WILLIAM FALOON

The ***Centers for Disease Control and Prevention*** say that more than **1** in **3** American adults have **blood sugar** levels that are too **high**.[1]

The condition they are referring to is **prediabetes**. It occurs when blood sugar markers are elevated, but have not yet reached the **diabetic** threshold.

Last year, UCLA researchers reported that **46%** of California adults are either **prediabetic** or have undiagnosed **type II diabetes**.[2] The severity of this health crisis cannot be overstated.

Diabetic pathologies develop during the **prediabetic** phase.[3] So by the time **type II diabetes** manifests, patients already confront complications that include **kidney impairment**,[4,5] **vision loss**,[6-8] **neuropathy**,[9] **atherosclerosis**,[10-12] and **cancer**.[13-15]

Despite these risks, populations around the world increasingly gorge on deadly foods/drinks that spike

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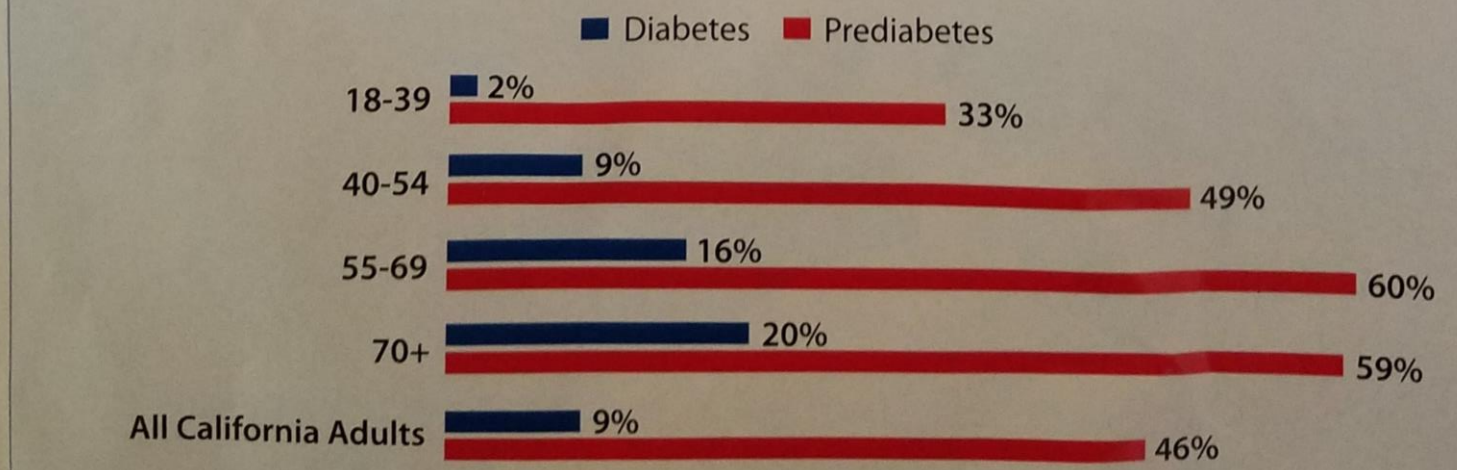
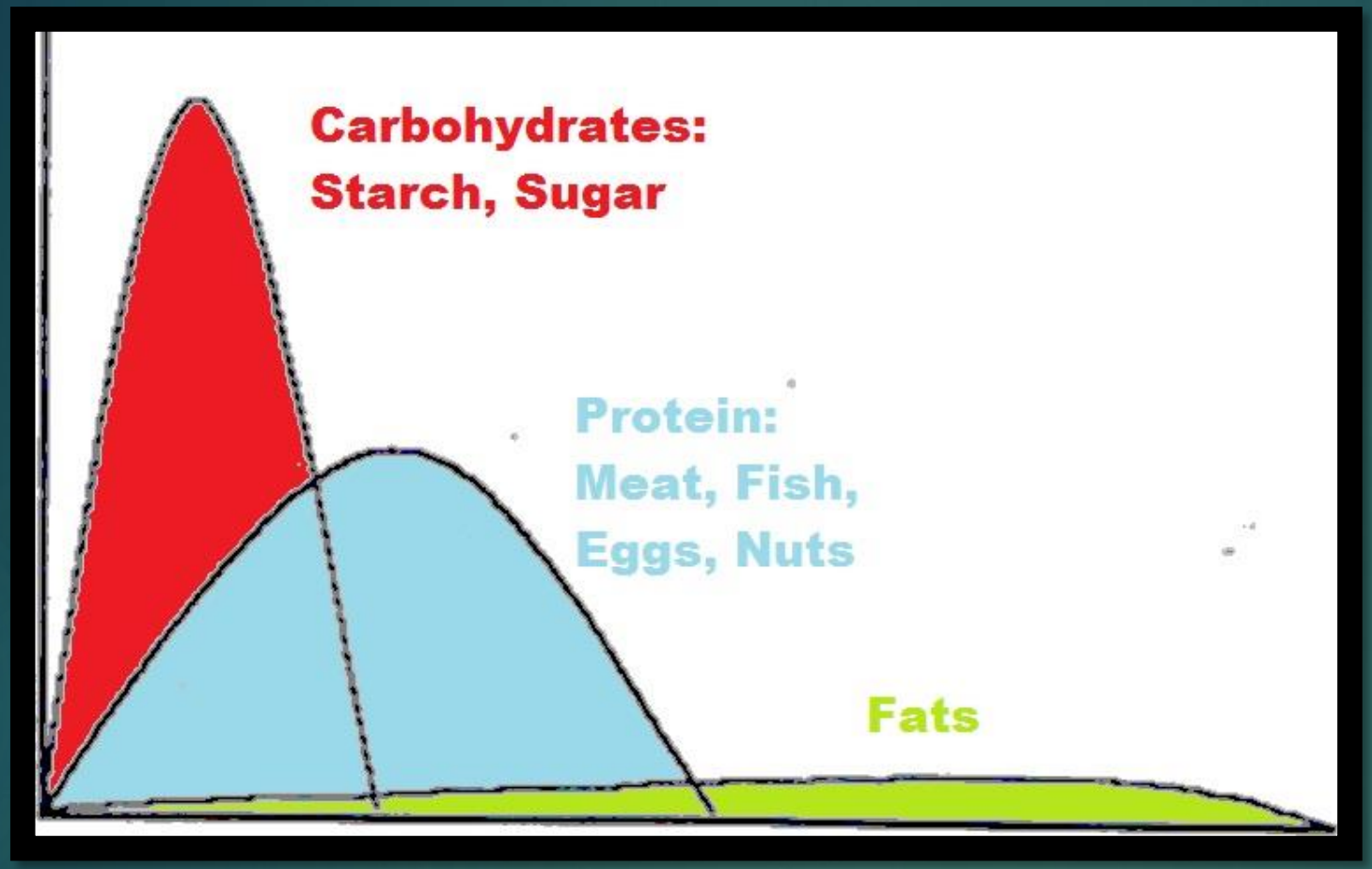
Diabetes Cases By Age Group

A UCLA study finds that **55%** of adults in **California** have either diabetes or prediabetes.[2]



(Note: California is a state that typically ranks near the top in longevity demographics.)

Source: 2013-14 California Health Interview Survey



Starches and sugars are quick burning and require the body to increase insulin production. High insulin levels blocks access to stored fat.

Key Point

Visceral (internal organ/abdominal) adipose (fat) tissue is most closely associated with the metabolic syndrome which is a constellation of conditions that place people at high risk for coronary artery disease.

There is no such thing as

"Borderline Diabetes"

It's all a matter of a progressive loss of glucose control, increasing levels of insulin and the consequent glycosylation damage to the body's proteins

INSULIN IN THE ACTUAL CULPRIT!

Historical features of Insulin Resistance – both sexes:

- Chronic fatigue
- Weight gain, multiple diets – Candida
- May have symptoms depending on whether a bedtime snack eaten
- May wake in the night needing a snack
- May wake anorexic and go as long as possible without eating
- Frequently snacks once fast is broken
- Sick if goes too long without eating
- Intolerant of dietary sugar: high and crash
- Craves sugar and starch
- Craves artificial sweeteners
- Uses lots of caffeine or stimulants
- Runs out of energy in afternoon
- Usually wake up feeling exhausted
- Usually have symptoms of reduced adrenal reserve
- Usually have symptoms of low thyroid
- Frequently on statin drug for lipids
- Frequently take antihypertensives
- Often use antidepressants

Mendelson S. *Metabolic syndrome and*…Academic Press 2008

Historical features – Women

- Change of habitus in early life
- Early menarche or development
- Teenage menstrual problems
  - Resulting in missed school
  - Requiring OCP to regulate
- Intolerance of OCP side effects
- Post-cholecystectomy (stones)
- Gestational diabetes
- Hypertension with pregnancy
- Excessive weight gain with pregnancy
- Baby's birth weight over 9 pounds
- Baby's birth weight under 5lbs 6oz

- Difficulty losing "baby fat"
- Premenstrual syndrome
- Irregularity and menorrhagia
- Dysmenorrhea and clots
- Hirsutism
- Thinning hair on head
- Loss of libido

Dunaif: "Syndrome XX"

Trends Endocrinol Metab. 2003 Oct;14(8):365-70

Historical features - Men

- Loss of libido
- Erectile dysfunction
- Prostatic symptoms
- Increased belly fat
- Gynecomastia
- Only man in his family with hair
- Decreased stamina

All results of low testosterone to E2 ratio

Other findings and symptoms of insulin resistance

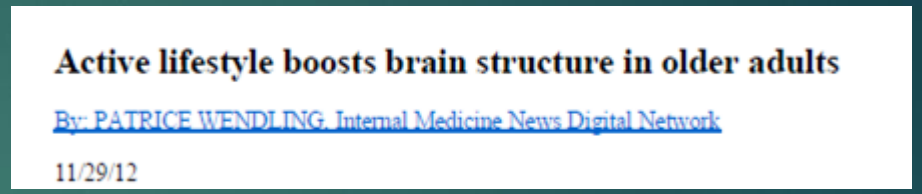
- Acrochordons (skin tags)
- Sleep apnea (even in slender people)
- Hyperglycemia and then hypoglycemia leading to more eating of carbs ~ a vicious cycle

The above leads to irregular eating patterns

- Frequent snacking
- Meniere's syndrome
- Migraine headache

Behaviors that promote insulin resistance

- Physical inactivity
- Lack of sleep
- Poor sleep
- Stressful lifestyle
- Electromagnetic pollution
- The “Standard American Diet” SAD



Active lifestyle boosts brain structure in older adults

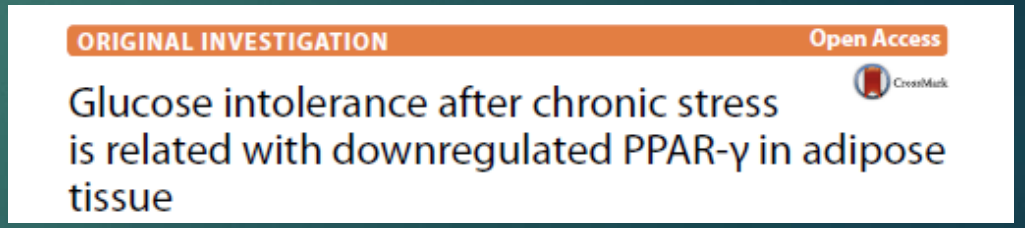
By: PATRICE WENDLING, Internal Medicine News Digital Network

11/29/12

Medical News & Perspectives

Resetting the Circadian Clock Might Boost Metabolic Health

Bridget M. Kuehn



ORIGINAL INVESTIGATION Open Access

Glucose intolerance after chronic stress is related with downregulated PPAR-γ in adipose tissue

How does increased sugar in the blood lead to the tissue damage we see in diabetes and the metabolic syndrome?

Elevated Sugar

↓

Deposition of sugar in proteins (Glycosylation)

↓

Over time, AGES develop

(Advanced Glycosylation End Products)

↓

Over time, “irreversible” changes occur in these glycosylated molecules known as RAGES

(Reactive Advanced Glycosylation End Products)

RAGES are the ultimate inflammatory destructive substances underlying the pathologies of diabetes and the metabolic syndrome:

Arteriosclerosis

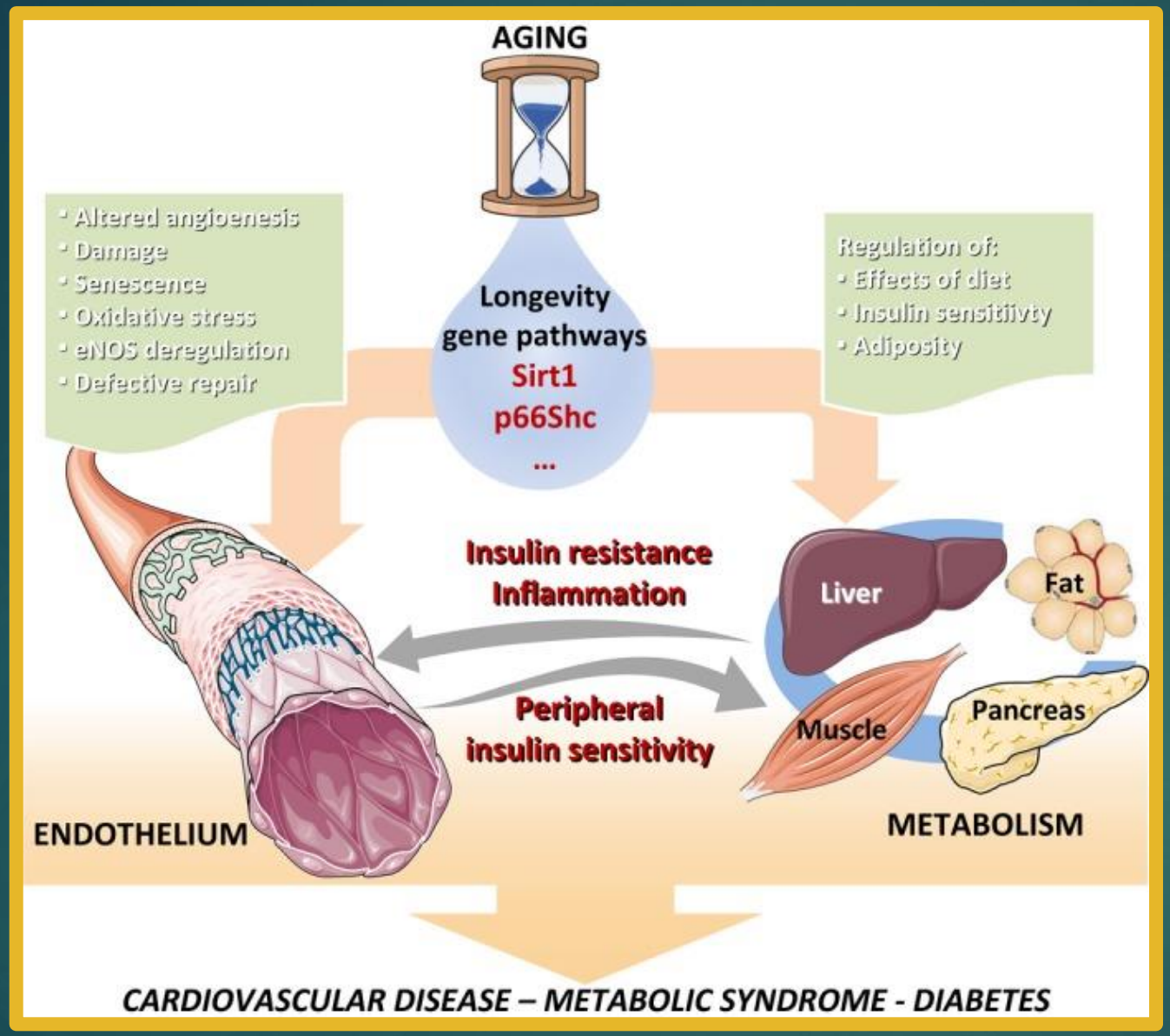
Kidney failure

Nerve damage

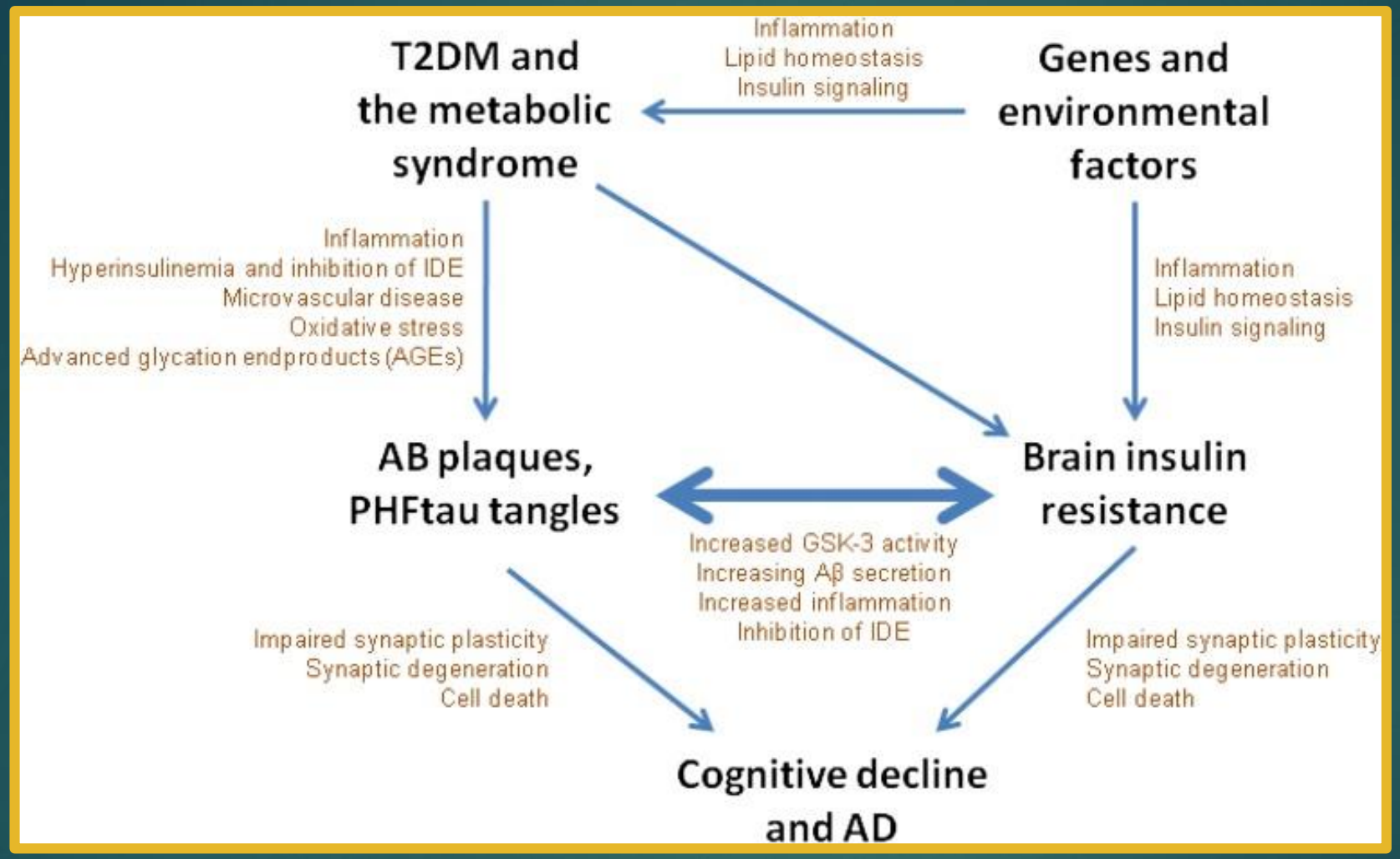
Retinal disorders/blindness

DEMENTIA

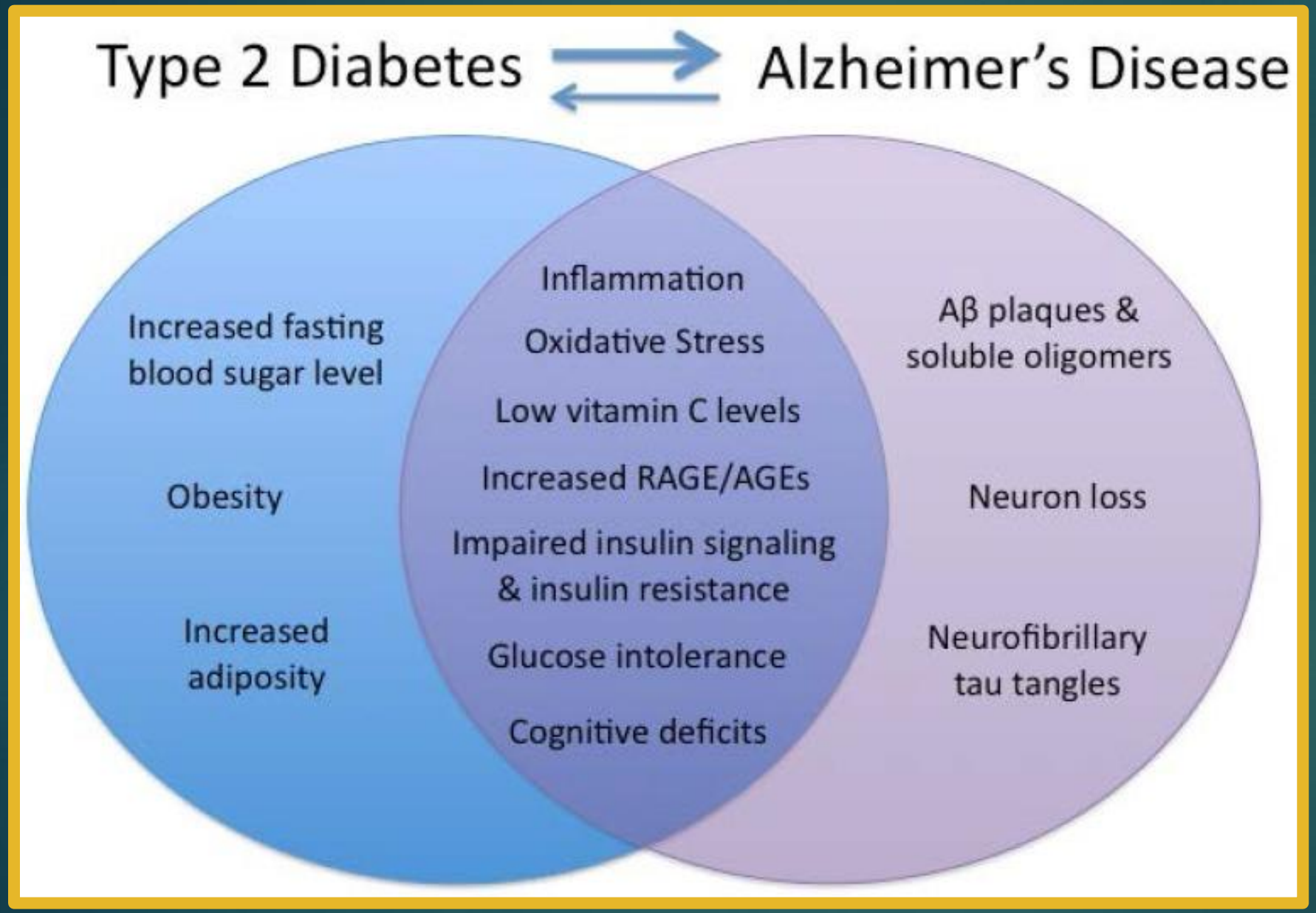
This means the person is not turning glucose into energy, but instead is turning it into fats and the non-used sugar is attaching to protein causing malfunction of such proteins leading to disease.



J Am Heart Assoc. 2013 Jun; 2(3): e000262.



Diabetes. 2014 Jul; 63(7): 2253–2261.



Is Alzheimer's Disease the new Type 3 Diabetes?
Scary.

Nutrients. 2015 Sep; 7(9): 7332–7357.

Hindawi Publishing Corporation
Oxidative Medicine and Cellular Longevity
Volume 2015, Article ID 985845, 14 pages
http://dx.doi.org/10.1155/2015/985845

Hindawi

Review Article

Diabetes and Alzheimer Disease, Two Overlapping Pathologies with the Same Background: Oxidative Stress

Sergio Rosales-Corral,[1] Dun-Xian Tan,[2] Lucien Manchester,[2] and Russel J. Reiter[2]

[1]*Centro de Investigación Biomédica de Occidente, Instituto Mexicano del Seguro Social, Sierra Mojada 800, Colonia Independencia, 44340 Guadalajara, JAL., Mexico*
[2]*Department of Structural Biology, The University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, USA*

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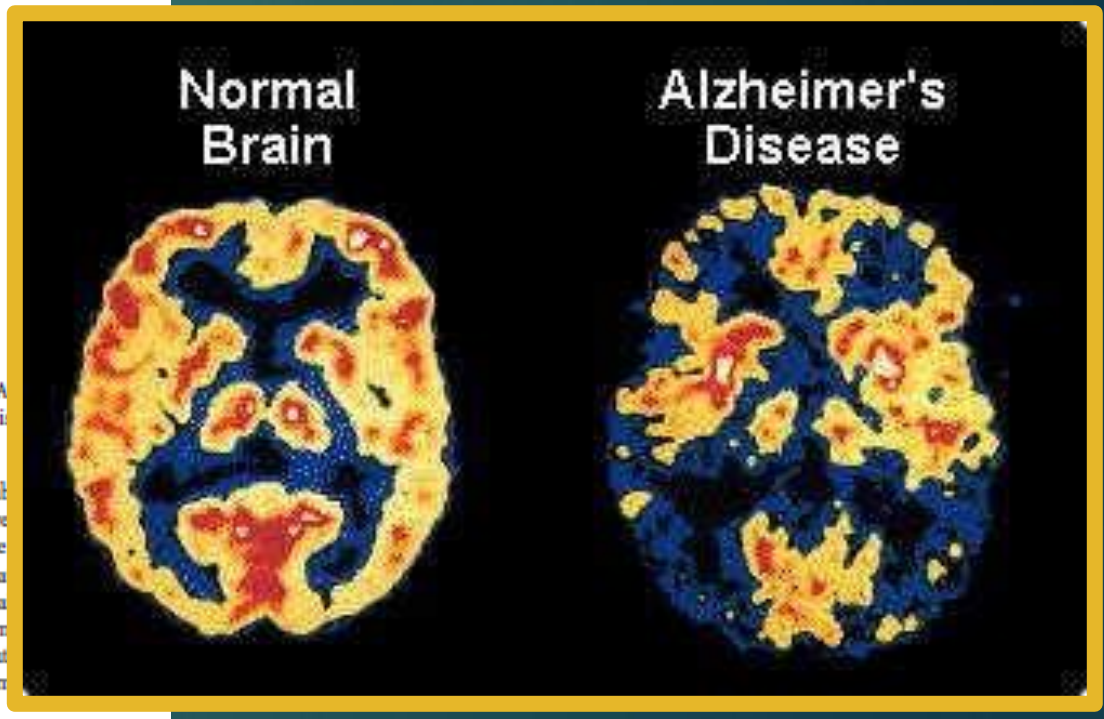
Academic Editor: Angel Catalá

There are several oxidative stress-related pathways interconnecting Alzheimer's disease and type II diabetes, two pul
problems worldwide. Coincidences are so compelling that it is attractive to speculate they are the same disorder. Howe
pathological mechanisms as observed in diabetes are not necessarily the same mechanisms related to Alzheimer's or the
related to Alzheimer's pathology. Oxidative stress is inherent to Alzheimer's and feeds a vicious cycle with other key pa
features, such as inflammation and Ca^{2+} dysregulation. Alzheimer's pathology by itself may lead to insulin resistance in bra
resistance being an intervening variable in the neurodegenerative disorder. Hyperglycemia and insulin resistance from
overlapping with the Alzheimer's pathology, aggravate the progression of the neurodegenerative processes, indeed. But
pathophysiological background is behind the consequences, oxidative stress. We emphasize oxidative stress and its detrim
in some key regulatory enzymes.

"Diabetes is a disease which often shows itself in families in which insanity prevails."
Sir Henry Maudsley, The Pathology of Mind, 1879.



NEUROLOGY

Brain atrophy is evident in patients with prediabetes

BY MICHELE G. SULLIVAN
AT EASD 2016

MUNICH – Brain changes suggestive of cerebral microvascular dysfunction are already apparent in patients with prediabetes.

The changes – increased white matter hyperintensities and decreased white matter volume – are even more pronounced in subjects with type 2 diabetes, Marnix van Agtmaal, MD, said at the annual meeting of the European Association for the Study of Diabetes. Patients with frank diabetes also showed an increase in intracranial cerebrospinal fluid – a correlate of the decrease in brain volume, said Dr. van Agtmaal of Maastricht (the Netherlands) University Medical Center.

The changes, visible on Tesla MRI using T1, T2, and FLAIR weighted images, are probably caused by diabetes-related endothelial dysfunction, he said.

"The brain is highly dependent on properly functioning microcirculation. This is critical, since the brain has high energy demand and no energy reserve. In prediabetes and type 2 diabetes, microvascular endothelial dysfunction occurs. This leads to cerebral hypoperfusion, which in turns causes chronic ischemia. This contributes to small vessel disease leading to brain atrophy and, eventually, cognitive decline and dementia."

Michele G. Sullivan/Frontline Medical News

Dr. Marnix van Agtmaal reported he has seen brain changes suggestive of cerebral microvascular dysfunction in patients with prediabetes on Tesla MRI.

The 2,251 subjects in the analysis were drawn from the Maastricht study, an ongoing observational study of people with type 2 diabetes.

Among the group, 350 had prediabetes, defined as impaired fasting glucose, impaired glucose tolerance, or a combination of the two. Type 2 diabetes was present in 528. The rest had healthy glucose metabolism.

As the cohort progressed from healthy glucose metabolism to pre

about 1.25 mL, and those with diabetes, about 2 mL. In both the partially and fully adjusted models, this relationship was somewhat attenuated, but it remained significant for both prediabetes and diabetes.

The crude model also found that both diabetes groups had significantly lower white
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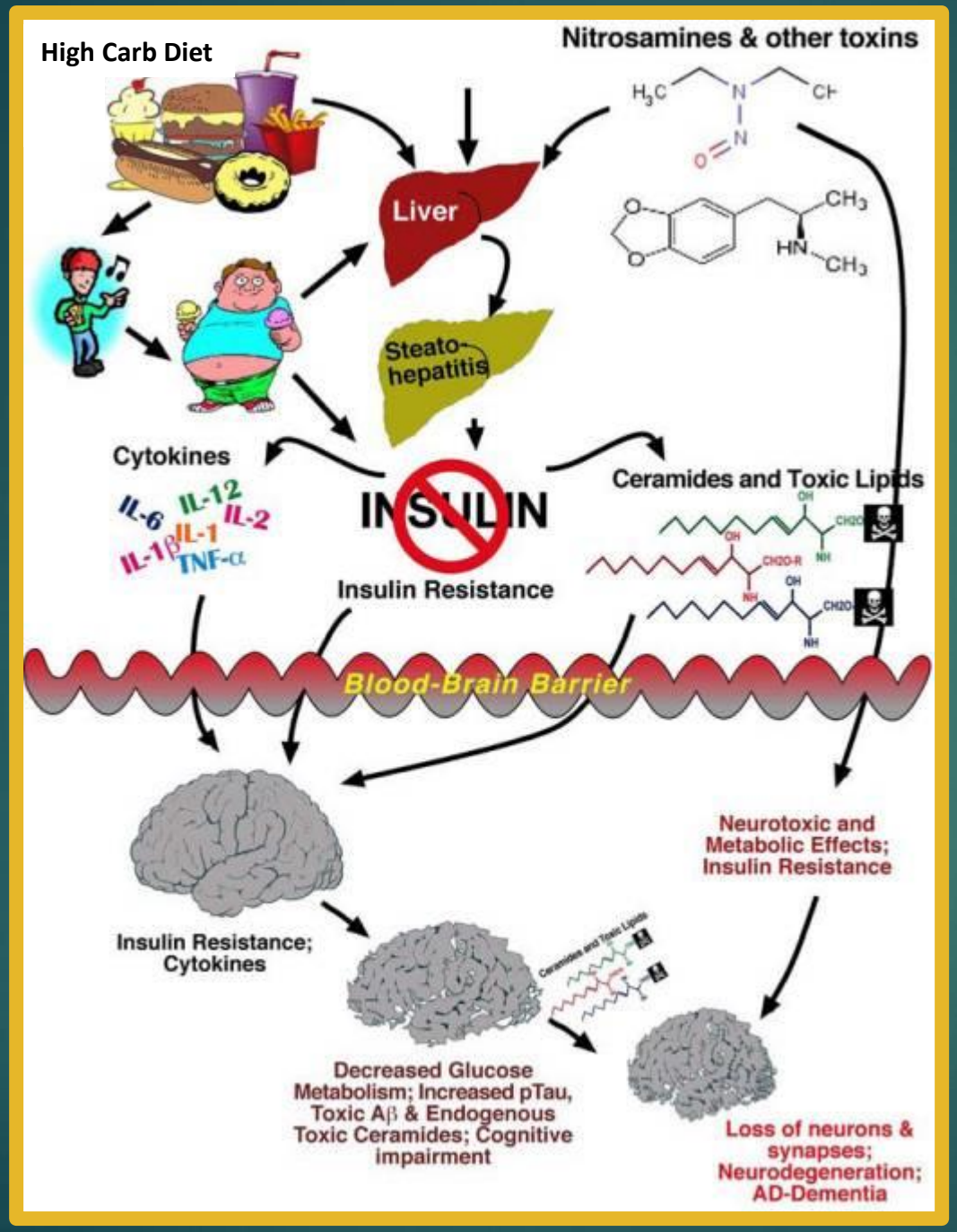
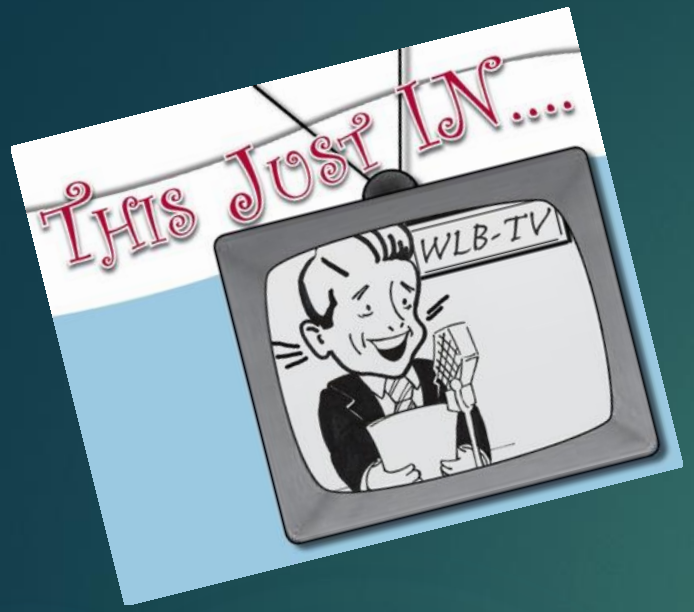
> "Further analysis will also look at cognitive decline and the development of dementia in the group. We also intend to look at associations with **other outcomes of cerebral dysfunction**, including depression."

Family Practice News. October 1, 2016



Curr Alzheimer Res. 2012 Jan; 9(1): 35–66.



Family Practice News, September 15, 2017

NEUROLOGY

Fueling the Alzheimer's brain, upping cognition with fat

BY MICHELE G. SULLIVAN
AT AAIC 2017

LONDON – A 3-month diet that includes 70% fat improved cognition in Alzhei-

fying diet can help prevent Alzheimer's and may even help AD patients think and function better. But this research has largely focused on the heart-healthy diets already proven successful in pre-

fat to protein and carbs is more critical than the source of the fat.

MIND was designed to prevent the cardiovascular and endocrine disorders than predispose to dementia over the

What's more, Dr. Cunnane said, these decrements are region specifi Deficits in glucose metabolism hit t thalamus, and temporal and parieta cortices – all pathologically importa in AD – particularly hard. The brai

Peer-reviewed scientific articles relating "insulin resistance" to a variety of diseases and conditions in scientific journals on pubmed.org-the largest biomedical library in existence:

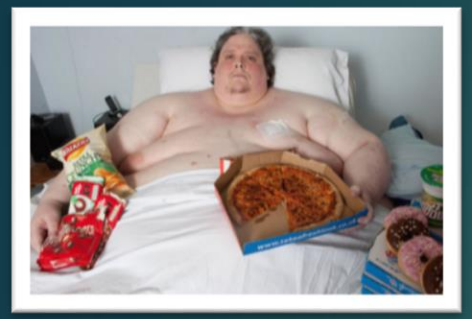
- obesity-38,900 papers
- cancer-9239 papers
- coronary heart disease-5,276 papers
- aging-4,355 papers
- kidney disease-3,977 papers
- stroke- 1,812 papers
- depression- 1,059 papers
- PCOS(polycystic ovary disease)-3,158 papers
- infertility-796 papers
- dementia-683 papers
- cognition-647 papers
- eye diseases-103 papers
- autism-22 papers
- fibromyalgia-18 papers
- fatigue-233 papers
- asthma-193 papers
- Alzheimer's disease-559 papers
- osteoporosis-561 papers
- neuropathy-419 papers
- anxiety-311 papers
- autoimmune disease-292 papers
- osteoarthritis-139 diseases
- muscle weakness-104 diseases

Other Insulin Effects in Insulin Resistance (beyond escorting sugar into cells)

- Makes cells fatter
- Stimulates growth hormone receptors (increase growth of cancers)
- Aldosterone effects (retain sodium, loses potassium, raises blood pressure)
- Fat metabolism (increased triglycerides, decreased HDL)
- Increases fat storage in liver (lipotoxicity, fatty liver)
- Damages vascular endothelium (retinopathy, nephropathy, neuropathy, coronary artery disease, large vessel disease, premature labor)
- Causes fat storage and inhibits fat breakdown
- Influences and deranges a variety of other hormones (thyroid, adrenal, sex hormones)

The medical literature strongly supports a relationship between:

- Insulin resistance and increase of fat levels in blood, artery lining and adipose tissue stores
- Increased production of tissue-damaging free radicals
- Increased calcium inside cells
- Dysfunction of the endothelial layer of blood vessels – especially small ones
- Vasoconstriction leading to increased blood pressure and loss of blood flow to tissues
- Decreased total body magnesium and lowered intra-cellular magnesium



Ingestion of sugar/carbohydrates

High carb diet

Low carb diet

Excess Insulin

Insulin release by pancreas beta cells

Sugar transported into cells *via* insulin receptors and GLUT (Glucose transporters)

"Insulin Resistance"

Sugar burned efficiently for energy and heat

Increased production of free radicals

Less energy and heat productions

Damage to cell structures

Inflammation

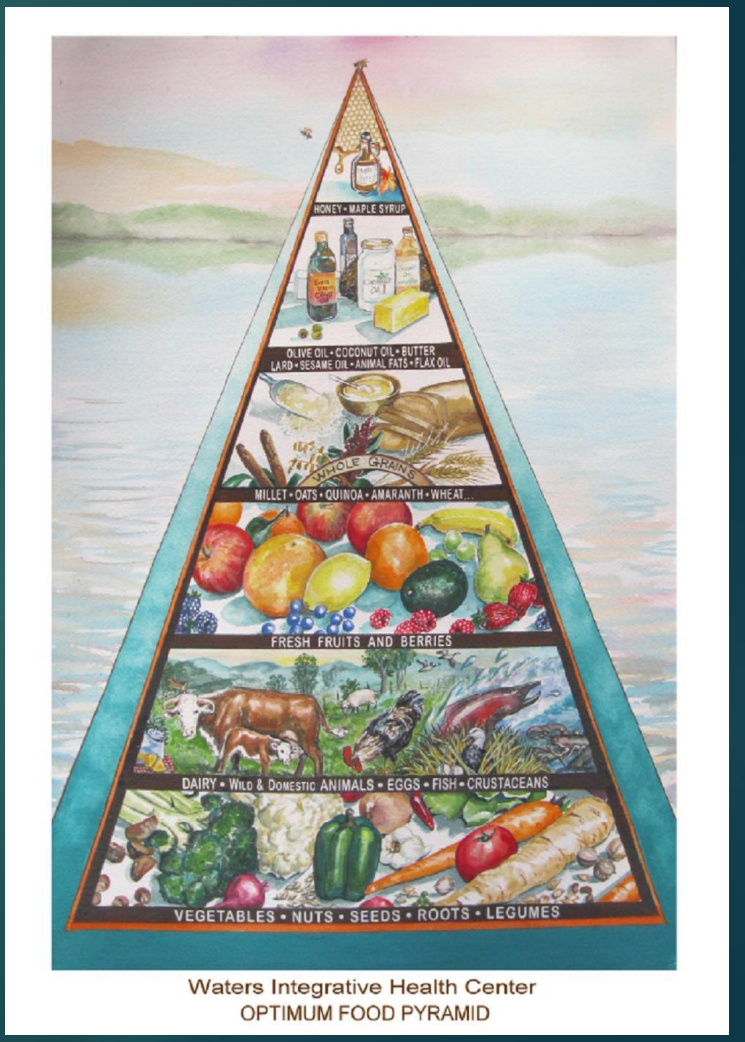
Weight gain and feeling cold

HEALTH AND HAPPINESS!

Coronary Artery Disease
Generalized Vascular Disease
Brain Atrophy / Dementia
Kidney Damage
Neuropathy
Retinopathy

What can we do?

- Diet, Digestion, Detox
- Exercise
- Stress
- Sleep



Let’s talk diet first…

We need to help you reset insulin receptors so you burn calories again!

- Paleolithic Diet with low sugar intake
- Ketogenic Diet
- Plant Paradox Diet
- Paleo/Keto/Plant Paradox Diet

We will guide you through the dietary regimens and help you choose what will work for you.

Paleolithic Diet with low sugar intake

- The Paleolithic diet (also called the paleo diet, caveman diet or stone-age diet) is based mainly on foods presumed to have been available to Paleolithic humans.
- Wide variability exists in the way the diet is interpreted. However, the diet typically includes vegetables, fruits, nuts, roots, and meat while excluding foods such as dairy products, grains, sugar, legumes, processed oils, salt, and alcohol or coffee.
- The diet is based on avoiding not just modern processed foods, but rather the foods that humans began eating after the Neolithic Revolution when humans transitioned from hunter-gatherer lifestyles to settled agriculture.
- Low sugar intake would mean limiting fruits, maple syrup, honey, etc.

Ketogenic Diet

- The ketogenic diet is a high-fat, adequate-protein, low-carbohydrate diet.
- The diet forces the body to burn fats rather than carbohydrates. Normally, the carbohydrates contained in food are converted into glucose, which is then transported around the body and is particularly important in fueling brain-function. However, if there is very little carbohydrate in the diet, the liver converts fat into fatty acids and ketone bodies. The ketone bodies pass into the brain and replace glucose as an energy source. An elevated level of ketone bodies in the blood is known as ketosis.
- Excludes high-carbohydrate foods such as starchy fruits and vegetables, bread, pasta, grains and sugar, while increasing the consumption of foods high in fat such as nuts, cream and butter.

Plant Paradox Diet

What do antelope do to avoid being eaten by cheetas?

What do plants do to avoid being eaten by antelope (and insects, humans, etc.)?

Lectins (from legere = to select) Proteins that bind carbohydrates

- Lectins cause insulin resistance which stimulates fat storage.
- Lectins cause Leptin resistance which makes you eat more. ← A BAD COMBINATION!
  - Leptin is the satiety hormone in the arcuate nucleus of the hypothalamus
  - Made in fat cells and is released when we eat to regulate energy homeostatis
  - A decreased sensitivity to Leptin occurs in obesity
- They bind to carbohydrates containing proteins on the gut and other mucosal surfaces and cause inflammation and thus "leaky" membranes. It is believed that this is the basis of systemic inflammation and autoimmunity.
- It is becoming clear that plant lectins are poisons to the predators of plants – including humans.
  - The main Lectins that are causing ill health are gluten and related molecules in cereal grains. The Lectins in other plant are less problematic for most people.

The Plant Paradox by Steven Gundry MD 2017

Lectins, continued

- Lectins occur in high concentrations in cereal grains, legumes, seeds, nuts and solanaceous vegetables.
- They decrease in the seeds of fruit as they mature.
- Some are highly toxic such as Ricin in the castor bean – two molecules can kill
- Uncooked, five kidney beans could clot all of your blood and kill you.
- Soybeans are very high in a powerful lectin known as soybean agglutinin (SBA)

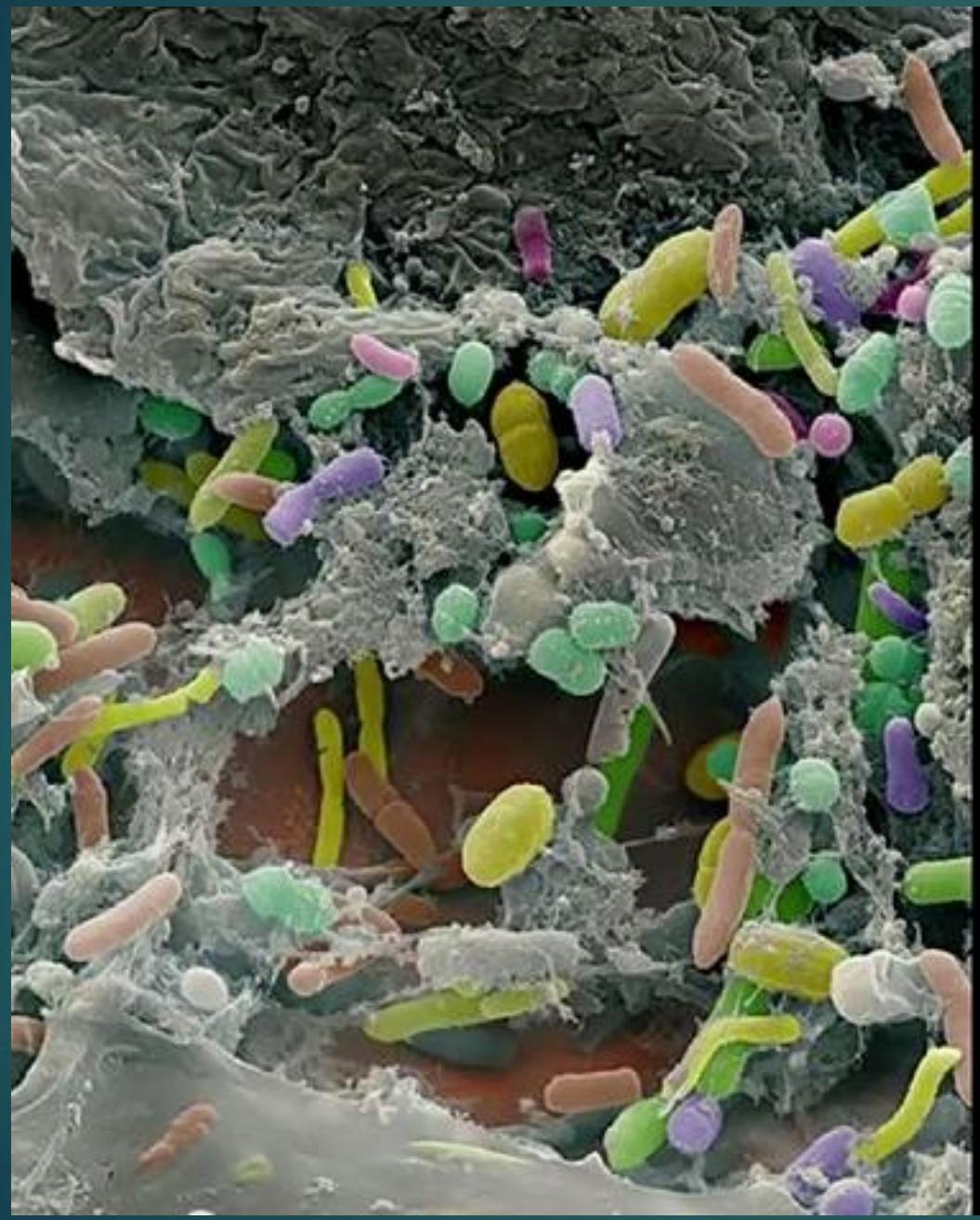
Schoeller, DA, et al. Entrainment of the diurnal rhythm of plasma leptin to meal timing. 1997

Herbs that help correct insulin resistance and inflammation

- Berberine
- Curcumin
- Green Tea Extract
- Cinnamon
- Bergamot
- OPC (Pine bark)
- Chocolate Polyphenols
- Boswellia
- Silymarin

Digestion: The Microbiome
A newly described “virtual organ”

- About 500 species of micro-organisms
- Ten time larger number of these single-celled creatures than the number of cells in our body
- They make 100's of compounds that regulate many body processes
- We give them a home and a part of our food in return for vitamins, short-chain fatty acids, processed plant pigments, etc.



Your flora is your friend

- Food digestion
- Protection against pathogens
- Provides essential nutrients (e.g. vitamins)
- 'Trains' your immune system
- *Disturbance* of flora is linked to disease

VIEWPOINT

The Microbiome and Risk for Obesity and Diabetes

Anthony L. Komaroff, MD
Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts.

Obesity and type 2 diabetes mellitus are influenced both by genes and lifestyle. That is not news. However, the genes in the human microbiome also may play an important role, and that is news.

It has been known for decades that gut bacteria synthesize essential vitamins and amino acids and help degrade toxins. During the past decade, it has become clear that the influence of the microbiome on health may be even more profound.

Beginning at the moment of birth, each human increasingly coexists with microbes. By the time individuals reach adulthood, they are colonized by many more microbial cells than the roughly 13 trillion human cells. More important still, these microbial cells (the microbiota), collectively, have exponentially more genes (the microbiome) than do human cells, around 250 to 800 times more.

Moreover, many genes in the human microbiome generate proteins, including hormones, neurotransmitters, and molecules of inflammation, that can enter the circulation and affect health. In light of this, it is reasonable to question whether the genes of the microbiome might play a greater role in health than do human genes. Recent evidence suggests that the microbiome may affect the probability of many major diseases, including obesity and diabetes.

Obesity

How could microbiota in the gut affect obesity? First, microbiota could influence the calories the body absorbs. Body weight is not affected by the calories that are ingested, but rather by the calories that are absorbed. Simple sugars in the diet are easily absorbed, and human enzymes convert starches into simple sugars, but human enzymes fail to digest many dietary polysaccharides. Microbial enzymes can turn those polysaccharides into digestible sources of energy, particularly monosaccharides and short-chain fatty acids.

> Recent evidence suggests that the microbiome may affect the probability of many major diseases, including obesity and diabetes.

About 90% of gut bacteria are in 1 of 2 phyla: Bacteroidetes and Firmicutes. Firmicutes generate more harvestable energy than Bacteroidetes. Obese humans have relatively more Firmicutes, as do rodents placed on a high-fat diet.[1]

Various experiments suggest that microbiota may powerfully affect obesity in mammals. For example:

- Gut microbiota from obese mice and from lean mice were transplanted into germ-free, lean mice, all of whom had the same daily caloric intake. Over the next 2 weeks, the mice receiving microbiota from obese mice became obese, whereas those receiving microbiota from lean mice remained lean.[1]
- Gut microbiota from conventionally raised animals were placed in the guts of lean germ-free mice. Without any increase in daily caloric intake, the body fat content of the animals increased by 60% within 14 days and they developed insulin resistance.[2]
- Obese mice underwent Roux-en-Y gastric bypass (RYGB) surgery or sham surgery. Mice that underwent RYGB surgery had the expected weight loss and a characteristic change in the gut microbiome, whereas mice that underwent the sham surgery did not. Transfer of bacteria from mice that underwent RYGB surgery to mice that underwent the sham surgery resulted in weight loss, although not as great as seen following RYGB surgery.[3]
- Investigators studied human twin pairs (mostly monozygotic) in which 1 person was obese. Feces from the fat twins and feces from the lean twins were fed to the germ-free mice, which were of normal weight. The mice fed feces from the fat twins became fat; those fed feces from the lean twins remained lean. The fat and lean mice were then housed together. Mice eat each other's feces. Gradually, the obese mice became lean and their gut flora came to resemble the flora of the lean mice. This finding suggests that the flora of lean mice may be able to dominate the flora of obese mice.[4]

These experiments suggest that the composition of gut microbiota can influence obesity. However, other experiments suggest that obesity can influence the composition of gut microbiota. For example, when obese people diet and lose weight, the proportion of Bacteroidetes increases relative to Firmicutes.[4] Conversely, when obese people resume their previous diets and gain weight, the proportion of Firmicutes increases.[4] These experiments suggest that the microbiome may be a reflection of obesity (or leanness), as well as a cause of it. In addition, low-grade gut inflammation caused by gut microbiota may increase the risk of obesity along with the risk of type 2 diabetes.

Type 2 Diabetes Mellitus

Given the increased risk of developing type 2 diabetes in obesity, it is not surprising that the microbiome might also influence type 2 diabetes. However, more than enhanced absorption of carbohydrates may be involved.

Relatively high ratios of Firmicutes to Bacteroidetes not only influence carbohydrate metabolism, but also alter the production of short-chain fatty acids. In particular, acetate production is increased and butyrate production decreased. A recent study[5] found that

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It is plausible that the human microbiome may affect the risk of obesity and type 2 diabetes and other diseases such as atherosclerosis, and that manipulations of the microbiome might reduce that risk.

The 7 Deadly Disruptors of Health

1. Antibiotics – They damage the organ called the Microbiome
2. Non-steroidal anti-inflammatory Drugs (NSAIDS) – Motrin, Aleve, etc. – They damage the intestinal permeability barrier, open the pathway to invading toxins including bacterial products.
3. Stomach Acid Blockers / Proton Pump Inhibitors – Nexium, Prilosec, etc. – Stomach acid protects us from swallowed micro-organisms and predigested proteins. Their use is correlated with kidney disease, dementia. They block proton pumps all over the body, including those in the mitochondria.
4. Artificial Sweeteners – They make your body think you ate sugar and thus more insulin is produced making you gain weight. Also, Splenda kills your microbiomal residents. Aspartame acts as a neurotoxin in some people.

The Plant Paradox by Steven Gundry MD 2017

The 7 Deadly Disruptors of Health, *continued*

5. Endocrine Disruptors – Plasticizers, Artificial hormones (BC pills, Premarin, Provers, etc.), Pesticides, Preservatives, Sun Screens, various cosmetic components, halogenated compounds, (Fluorine, Bromine, Chlorine) – These substances alter Thyroid and steroid hormone receptor function leading to obesity, male and female reproductive problems, hormone deficiencies, cancers, prostate problems, impaired neuroendocrine development (think autism spectrum disorders) and sexual confusion. The triclosan in hand sanitizers and toothpaste promotes obesity by altering gut flora and causes bladder cancer. Sunscreen blocks vitamin D production. The phthalates have been shown to damage sperm DNA, attach to estrogen and thyroid hormone receptors causing premature sexual development in girls and obesity.

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HHS Public Access
Author manuscript
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Published in final edited form as:
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Persistent organic pollutants in young adults and changes in glucose related metabolism over a 23-year follow-up

Jose R. Suarez-Lopez[a,*], **Duk-Hee Lee**[b], **Miquel Porta**[c], **Michael W. Steffes**[d], and **David R. Jacobs Jr.**[e]

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Abstract

Objectives—Substantial evidence associates persistent organic pollutants (POP) with metabolic disturbances related to diabetes, but longitudinal studies with repeated measures are scarce. We aimed to characterize the association between background exposures to POPs with repeated measures of glucose homeostasis over 23-years.

Methods—Within the Coronary Artery Risk Development in Young Adults study (year 0 ages: 18–30 years), we measured POPs in serum obtained in 1987–88 (follow-up year 2) in 90 non-diabetic controls and 90 cases diabetes-free at year 2 who became diabetic by year 20. We analyzed 32 POPs detectable in ≥75% of participants and created summary scores for 32 POPs, 23 polychlorinated biphenyls (PCB), and 8 organochlorine pesticides (OCP). Dependent variables were measures of glucose homeostasis at years 0–25 (up to 8 examinations). We explored associations using repeated measures regression adjusted for race, sex, concurrent body mass index (BMI), examination center and period, separately for cases and controls.

*Corresponding author.

Competing financial interests
None.

Institutional review board approval for research
The study was approved by the institutional review boards of the University of Minnesota, University of Alabama at Birmingham, Northwestern University, and the Division of Research at Kaiser Permanente Health Care Plan. Participants signed informed consent at every examination.

Objectives—We aimed to characterize the association between background exposures to POPs with repeated measures of glucose homeostasis over 23-years.

Methods—We measured POPs in serum obtained in 1987–88 (follow-up year 2) in 90 nondiabetic controls and 90 cases diabetes-free at year 2 who became diabetic by year 20. We analyzed 32 POPs detectable in ≥75% of participants and created summary scores for 32 POPs, 23 polychlorinated biphenyls (PCB), and 8 organochlorine pesticides (OCP).

Conclusions—Glucose homeostasis may worsen after decades of exposure to PCBs and OCPs at background environmental levels, independent of BMI and after participants reached the 5th decade of life.

Environ Res. 2015 February ; 137: 485–494.
doi:10.1016/j.envres.2014.11.001.

The 7 Deadly Disruptors of Health, *continued*

6. Genetically Modified Food and the Associated Herbicide Round-Up – Alien proteins are probably in underpinning of a lot of allergy and auto-immunity. Glyphosphate (Round-up's active ingredient) is turning out to be disastrous for health. It damages our microbiomal residues, has been linked to cancer, kidney disease (El Salvador banned it), liver disease and other conditions. The EU is progressively restricting its use and is returning GMO, Round-up laden grains for use as animal feed.
7. Exposure to Blue Light – This wavelength suppresses melatonin production and thus disturbs sleep. It also stimulates ghrelin and cortisol production, which increases hunger and wakes us up. Blue light stimulates the long days of summer and makes us eat excessively thereby contributing to obesity. We now live in endless summer because of artificial light.

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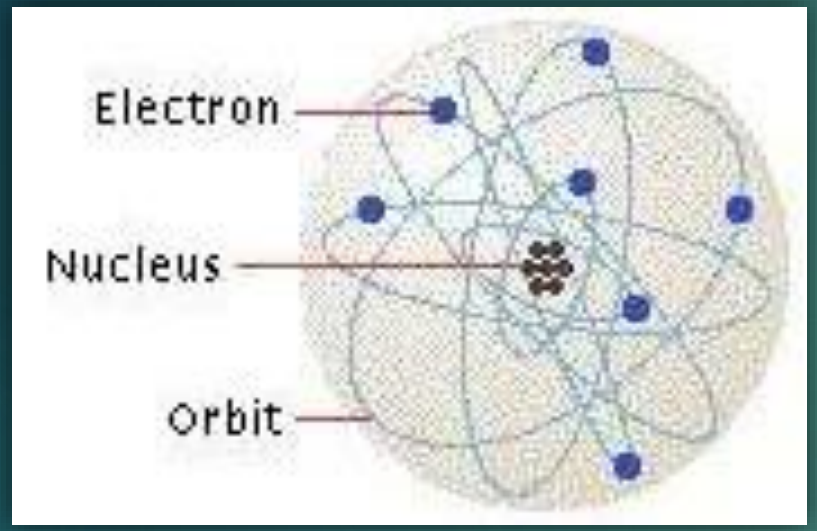
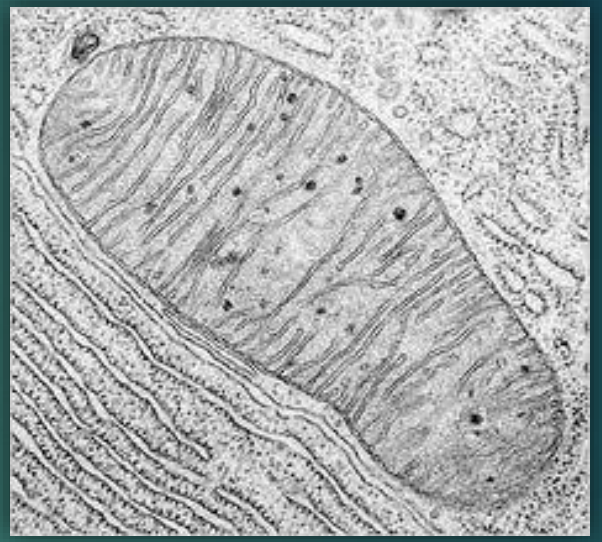
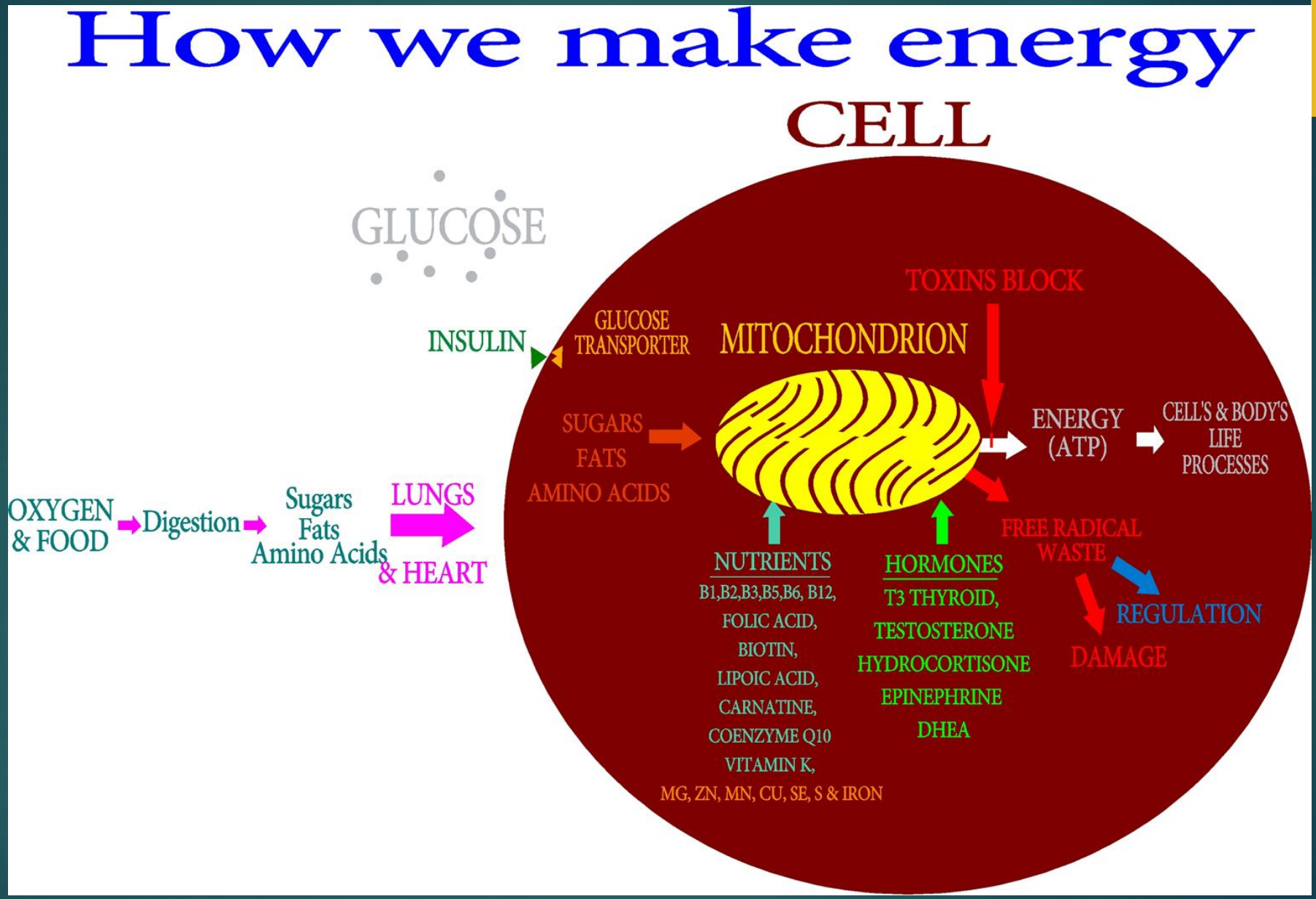
The Mighty Mitochondrion

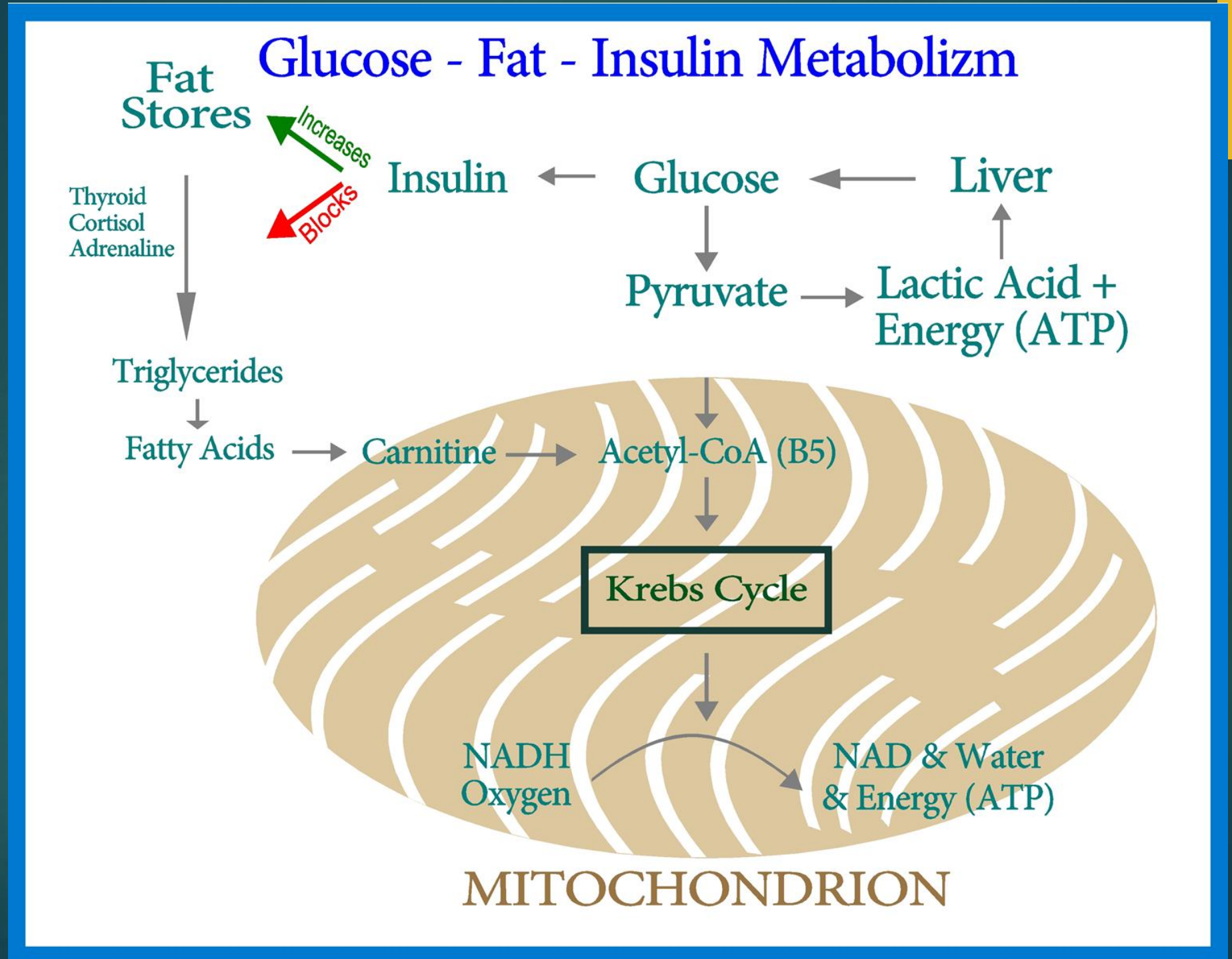




- How we burn food calories and oxygen to make the energy for life's processes.
- Mitochondria are the furnaces of our cells and make up 10% of body weight

How we make energy




Glucose - Fat - Insulin Metabolizm
Fat Stores
Increases
Blocks
Insulin
Glucose
Liver
Thyroid
Cortisol
Adrenaline
Pyruvate
Lactic Acid + Energy (ATP)
Triglycerides
Fatty Acids
Carnitine
Acetyl-CoA (B5)
Krebs Cycle
NADH
Oxygen
NAD & Water
& Energy (ATP)
MITOCHONDRION

ANTIOXIDANTS & REDOX SIGNALING
Volume 12, Number 4, 2010
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DOI: 10.1089/ars.2009.2531

Mitochondrial Dysfunction in Diabetes: From Molecular Mechanisms to Functional Significance and Therapeutic Opportunities

William I. Sivitz and Mark A. Yorek

Abstract

Given their essential function in aerobic metabolism, mitochondria are intuitively of interest in regard to the pathophysiology of diabetes. Qualitative, quantitative, and functional perturbations in mitochondria have been identified and affect the cause and complications of diabetes. Moreover, as a consequence of fuel oxidation, mitochondria generate considerable reactive oxygen species (ROS). Evidence is accumulating that these radicals *per se* are important in the pathophysiology of diabetes and its complications. In this review, we first present basic concepts underlying mitochondrial physiology. We then address mitochondrial function and ROS as related to diabetes. We consider different forms of diabetes and address both insulin secretion and insulin sensitivity. We also address the role of mitochondrial uncoupling and coenzyme Q. Finally, we address the potential for targeting mitochondria in the therapy of diabetes. *Antioxid. Redox Signal.* 12, 537–577.

Reviewing Editors: *Nader Abraham, David Busija, Anonymous, Alexander Galkin, Thomas Kietzmann, Renu Kowluru, Matthew Picklo, and Yuichiro J. Suzuki*

Department of Internal Medicine, Division of Endocrinology and Metabolism, Iowa City Veterans Affairs Medical Center and the University of Iowa, Iowa City, Iowa.

Qualitative, quantitative, and functional perturbations in mitochondria have been identified and affect the cause and complications of diabetes. Moreover, as a consequence of fuel oxidation, mitochondria generate considerable reactive oxygen species (ROS). Evidence is accumulating that these radicals
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Department of Internal Medicine, Division of Endocrinology and Metabolism,
Iowa City Veterans Affairs Medical Center and the University of Iowa, Iowa City, Iowa.

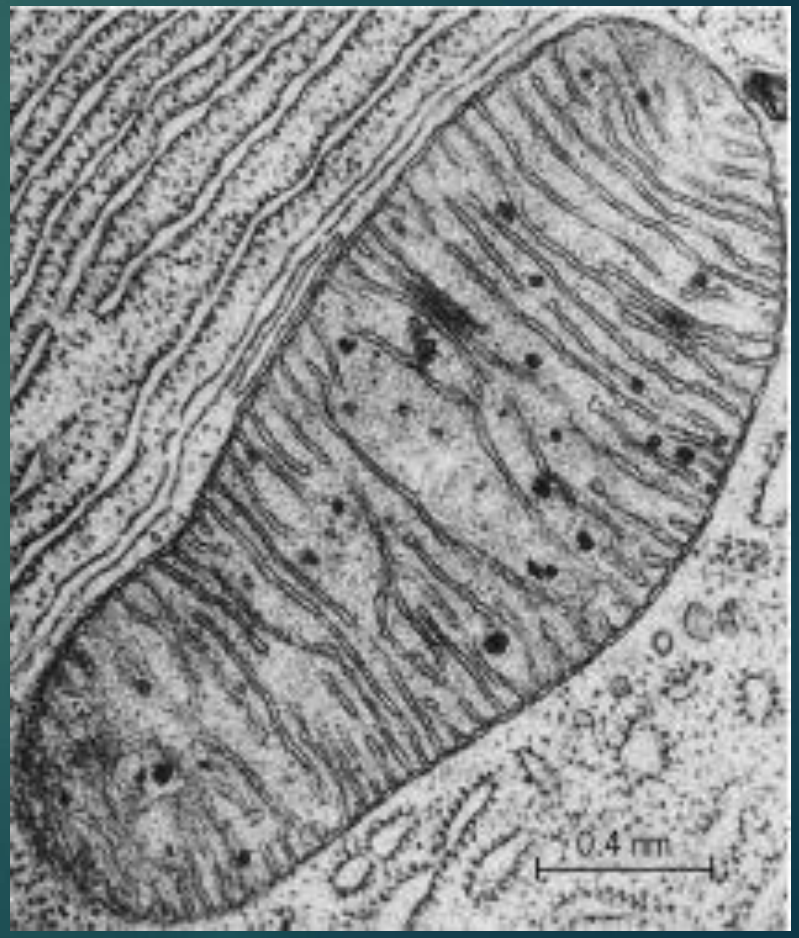
Factors that Inhibit Mitochondrial Function

- Heavy Metals (lead, mercury, cadmium, arsenic, etc.)
- Excess Iron
- Pesticides
- Herbicides
- Pharmaceutical drugs
- Solvents
- Vaccine – components
- Self-generated Waste Products
- Plasticizers
- PVC’s
- PCB's
- Industrial Chemicals

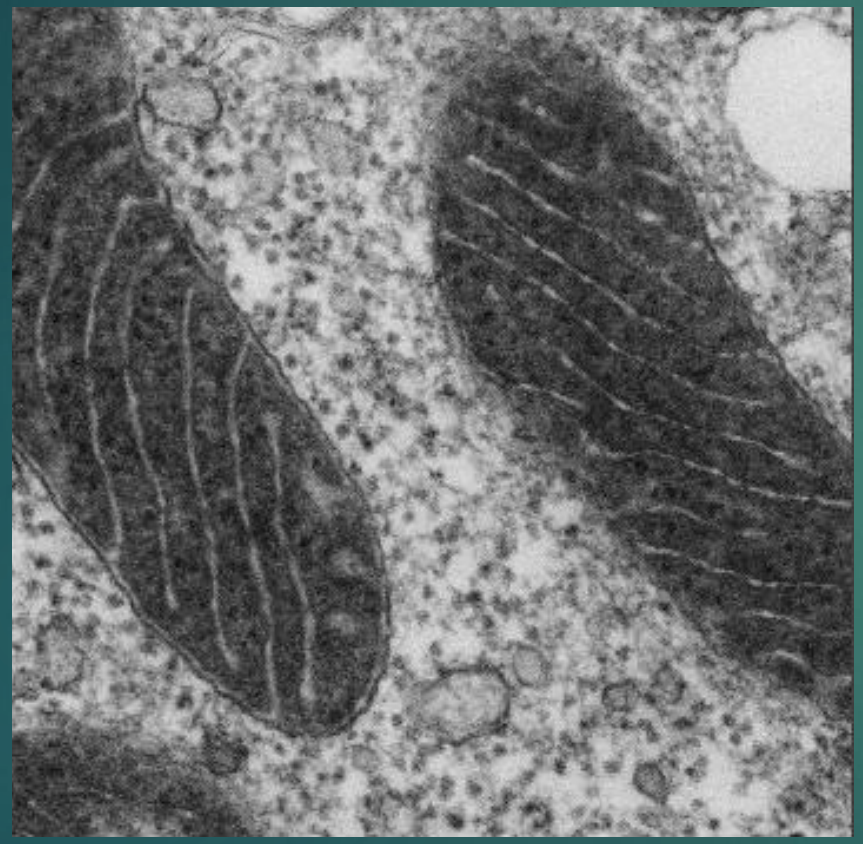
Mitochondria Get Sick and Swell

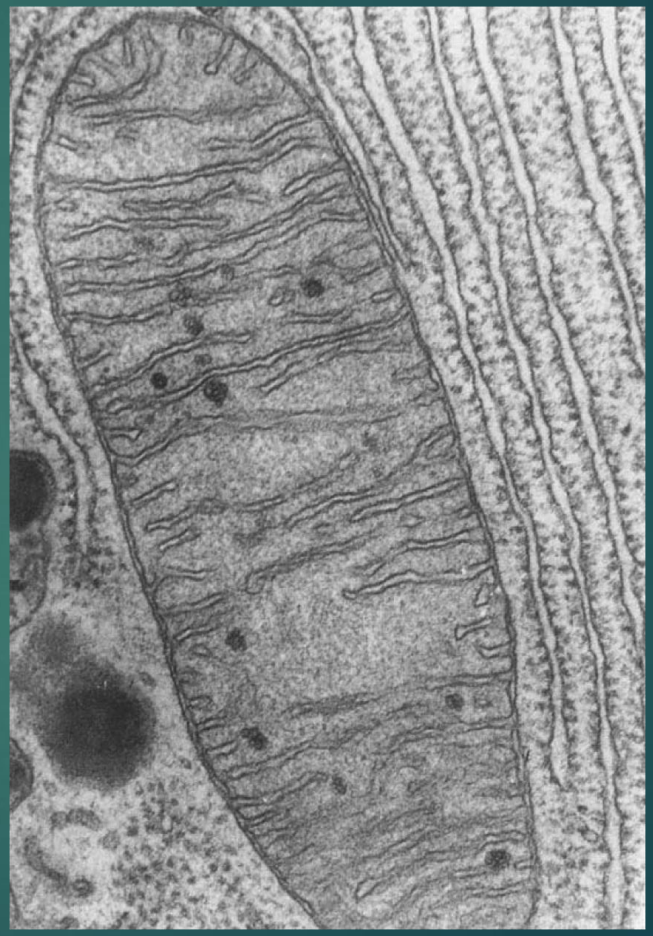
- Toxic metals such as lead

  (changing protein pattern)
- Free radicals
- Calcium influx
- Contamination from a damaged mitochondrion to others in the cell
- Disruption of oxidative phosphorylation sequential arrangement



Calcium as “Toxic” Element





Metals, Toxicity and Oxidative Stress

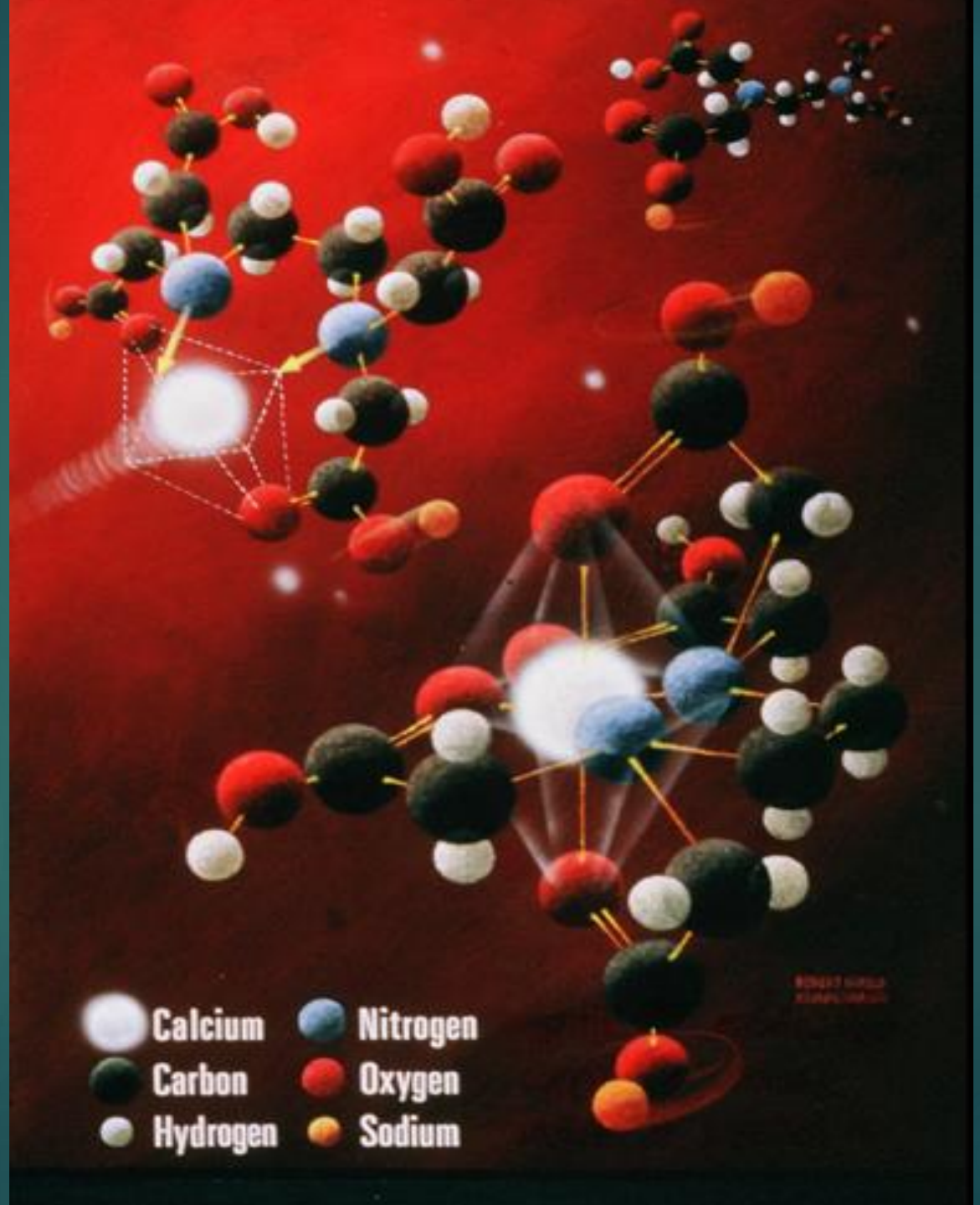
"The unifying factor in determining toxicity and carcinogenicity for all these metals is the generation of reactive oxygen and nitrogen species [free radicals!]. … Antioxidants (both enzymatic and non-enzymatic [such as chelation]) provide protection against deleterious metal-mediated free radical attacks."

Valko M, Morris, H and Cronin M. Current Medicinal Chemistry, 2005

Ferritin Levels Deadly

NOT high cholesterol, *NOT* high blood sugar, *NOT* high blood pressure, *NOT* high-whatever … Salonen showed that, after tobacco totals, high blood *ferritin* correlated most closely with myocardial infarction – when higher than 200 µg/ml = double risk

Salonen JT, Nyyssonen K, et al. Circulation, 1991


Calcium
Nitrogen
Carbon
Oxygen
Hydrogen
Sodium

Lead and Cardiovascular Health

- Lead inhibits repair of arterial endothelial barrier *and* nitric oxide (NO) production
- NO relaxes smooth muscles, inhibits platelet aggregation, mediates bactericidal/antiviral activity of macrophages and decreases inflammatory response

Am Cardiol (1999)83:1488-90; Annu Rev Physiol (1995)57:771-90;
Prog Cardiovasc Dis (1995)38:155-66; Adv Immunol (1995)60:323-71

Lead and Hypertension in Peri- and Post-Menopausal Women

- 3rd NHANES Survey (1988-1994)
- Follow up of 2,165 women, 40-59yo
- Conclusion: “At blood Lead levels well below occupational exposure limit guidelines, blood Lead positively associated with Blood Pressure, and both systolic and diastolic hypertension”

JAMA (2003)289:1525-32

Lead is a Calcium Antagonist

- A primary mechanism of Lead neurotoxicity is disruption of Calcium metabolism
- Lead enters cells through Calcium channels, keeps Calcium channels open about 5X longer than Calcium
- This allows excess calcium to enter cells and “jam up” cellular machinery

Lead Neurotoxicity

- Activates phosphokinase c (>binding than Ca)
  - Increased neuronal responsiveness: distractibility, impulsivity and motor restlessness
- Inhibits NMDA receptor-more than at a physiological level
- Alters metabolism of dopaminergic, cholinergic, and glutamatergic neurotransmitters (NT)
- Aberrant Ca homeostasis, increased spontaneous NT release (hyperexcitability of nerve cells)
- Disrupts microtubule metabolism- “Railroad tracks” of the cell

Excellent Review: Ann Rev Pharmacol Toxicol (1995)35:391

Lead Burden: Neurological Symptoms

- Behavioral toxicant
- Sensory deficits (visual/auditory)
- Motor deficits/cognitive dysfunction
- Memory loss, depression, confusion
- “Aged” population at increased risk due to mobilization from bone (osteoporosis)(release of a lifetime of stored lead in bone)

Osteoporosis Int (1999)9

Immunological Biomarkers of Metal-induced Neurotoxicity

- Exposure to Lead or MeHg induces reactive astrogliosis, neuronal degradation and secondary demyelinization which increases release of proteins

  Neurotypic: neurofilament triplet proteins (NFs)

  Gliotypic: glial fibraillary acidic protein (GFAP)

  Myelin: myelin basic protein (MBP

Neurotoxicol (1996)17; Neurotoxicol (1994)15

Biomarkers of Neurotoxicity (cont'd)

- Neural "antigens" are mitogenic to lymphocytes and elicit autoimmune responses
  - *Anti-GFAP, Anti-NFs, Anti-MBP*
- Magnitude of serum Ab response correlates with extent of neurological damage
- Sensitive indicator of neurotoxic EFFECTS of metals

Environ Hlth Perspect (1999)107; Neurotoxicol (1996)17

Metals Compromise Innate Immunity

Arsenic, Mercury, Cadmium, Lead, Nickel and Tin

- Decreased numbers and activities of Macrophages and Natural Killer cells
- Decreased Resistance to bacteria, protozoa and fungi
- Decreased Antiviral and tumericidal activity

Int Arch Occup Envir Hlth (1997)68; Toxicol of Metals (1996); Immunol Today (1992)13; Toxicol (1991)67

Metals and Cancer

Arsenic, Cadmium, Nickel, Lead, Tin, Beryllium

- Mechanisms include: free radical production, DNA-protein cross-links, altered DNA polymerase, decreased SOD, depletion of rGSH and Se, inhibition of “clean up” apoptosis
- Indirect: immunosuppression that allows chemical initiation -> neoplasia

Lead and Cancers

- Renal, brain (gliomas), lung, stomach
- Synergistic with other carcinogens
- Chromasomal aberrations / gene expression, decreased DNA repair
- Specific Lead-binding proteins -> renal tubules, nuclear Lead inclusion bodies
- Pb_{cyt} -> $Pb_{nucleus}$ (binds chromatin)

Am J Ind Med (2000)38:295; Toxicologist (1994)14:14

Journal of Advancement in Medicine
Volume 2, Numbers 1/2, Spring/Summer 1989

Ninety Percent Reduction in Cancer Mortality after Chelation Therapy With EDTA

Walter Blumer, M.D. and Elmer Cranton, M.D.

ABSTRACT

Mortality from cancer was reduced 90% during an 18-year follow-up of 59 patients treated with Calcium-EDTA. Only one of 59 treated patients (1.7%) died of cancer while 30 of 172 non treated control subjects (17.6%) died of cancer (P=0.002). Death from atherosclerosis was also reduced. Treated patients had no evidence of cancer at the time of entry into this study. Observations relate only to long-term prevention of death from malignant disease, if chelation therapy is begun before clinical evidence of cancer occurs. Control and treated patients lived in the same neighborhood, adjacent to a heavily traveled highway in a small Swiss city. Both groups were exposed to the same amount of lead from automobile exhaust, industrial pollution and other carcinogens. Exposure to carcinogens was no greater for the studied population than exists in most other metropolitan areas throughout the world. Statistical analysis showed EDTA chelation therapy to be the only significant difference between controls and treated patients to explain the marked reduction in cancer mortality.

Edta is well recognized as a therapy for lead toxicity. EDTA also removes other toxic heavy metals and nutritional elements such as iron which promote cancer by catalyzing free radical pathology.

Lead from automobile exhausts, petrochemicals form wear of automobile tires, cadmium, and other carcinogens are present in higher concentrations adjacent to heavily traveled automobile highways. These substances cause cancer and potentate other carcinogens.

It was reported in an earlier paper that cancer mortality among 231 adults living along a heavily traveled highway was higher than among persons living in a traffic-free section of the same city1 Nervous disorders, headaches, fatigue, gastrointestinal disorders, depression, and substance abuse was also observed with higher frequency.2 It was postulated that lead exposure from automobile exhausts might be one cause of this difference.

Beginning in 1961, a group of 59 patients with such symptoms was treated with parenteral doses of Calcium EDTA. Symptoms improved and urinary delta-amino levulinic acid diminished.3

Subsequent to the EDTA chelation therapy, a decrease in cancer mortality was observed. When compared with a control group of untreated patients who did not receive EDTA, many fewer cancer deaths were recorded.4,5 The control group was similar to the treated group in all ways except to the EDTA chelation therapy.

The purpose of this present study is to determine more precisely and to statistically analyze the long-term change in cancer mortality after treatment with EDTA.

What Do Toxic Metals Do?

- Neurotoxic
- Nephrotoxic
- Cardiovascular Disease
- Dysregulation of Immune System
- Compromise Gastrointestinal Integrity
- Compromise Nutritional Status
- Carcinogenic / Mutagenic

Mechanism of Action of EDTA Chelation Therapy

- Removes toxic metals
- Corrects calcium levels in small vessel lining
- Reduces free radical damage to DNA and membrane lipids
- Makes blood platelets less sticky
- Reduces cholesterol and triglyceride levels
- Improves cellular energy production
- Improves endothelial function and thus corrects the micro vascular disorder of insulin resistance
- Corrects magnesium deficiency which is at the heart of insulin resistance syndrome
- Improves kidney function, reduces uric acid, etc.

Chelation Therapy

EDTA History

- 1933 first synthesized in Germany and USA for use in linen dye industry to remove calcium from water.
- 1947 Nickel EDTA used in breast cancer to deliver Nickel to tumors – failure
- 1950's EDTA first used to treat lead encephalopathy in children – successful. Before this use, this was uniformly a fatal disease (no treatment)

Chelation continued…

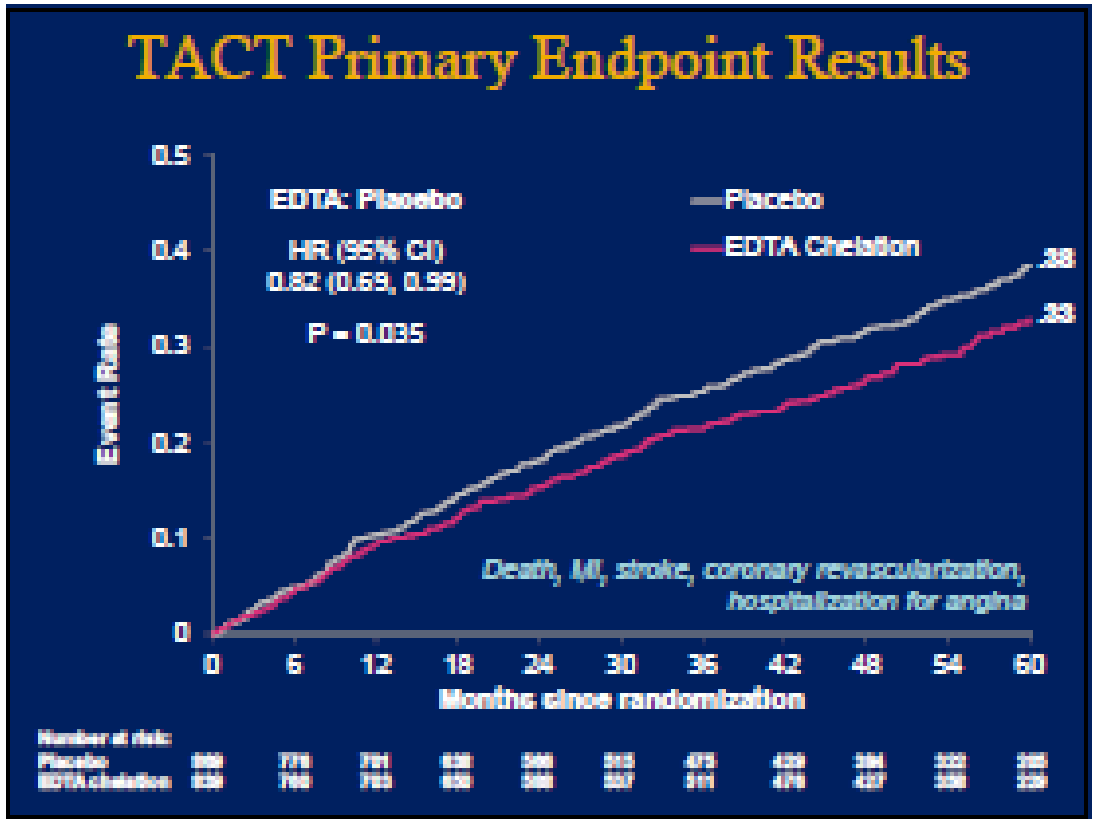
- 1960-70's Wider use of EDTA for lead poisoning and doctors noted that patients with heart and vascular diseases improved.
- 1970-90's Rise of Bypass Surgery and the suppression of EDTA Chelation Therapy
- 2003 EDTA Chelation Therapy shown to be effective in avoiding dialysis in kidney failure patients (double-blind study in Taiwan-New England Journal of Medicine).
- 2004-2012 TACT (Trial to Assess Chelation Therapy)

Lead and Renal Insufficiency

- 24 non-diabetic, chronic renal disease patients
- 27 month placebo/control prospective study
- Ca-Na2-EDTA significantly improved glomerular filtration and decreased progression of renal insufficiency
- Cost comparison
  - Chelation ~ $3,750
  - Hemodialysis ~ $61,000

New Eng J Med (2003)348:277-86

Trial to Assess Chelation Therapy (TACT)



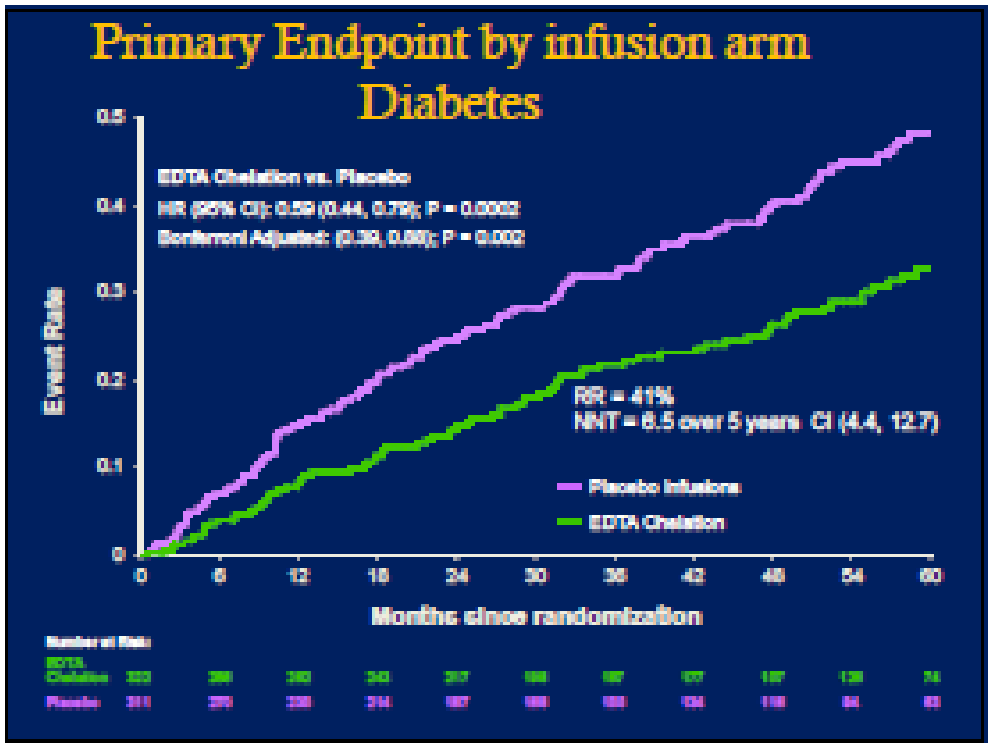
Conclusion 1

- EDTA-based chelation therapy reduces combined cardiovascular events in post MI patients treated with optimal medical therapy (5-year NNT= 18).

The Trial to Assess Chelation Therapy (TACT): Connecting Silos of Scientific Information

Gervasio Lamas, MD

Trial to Assess Chelation Therapy (TACT)

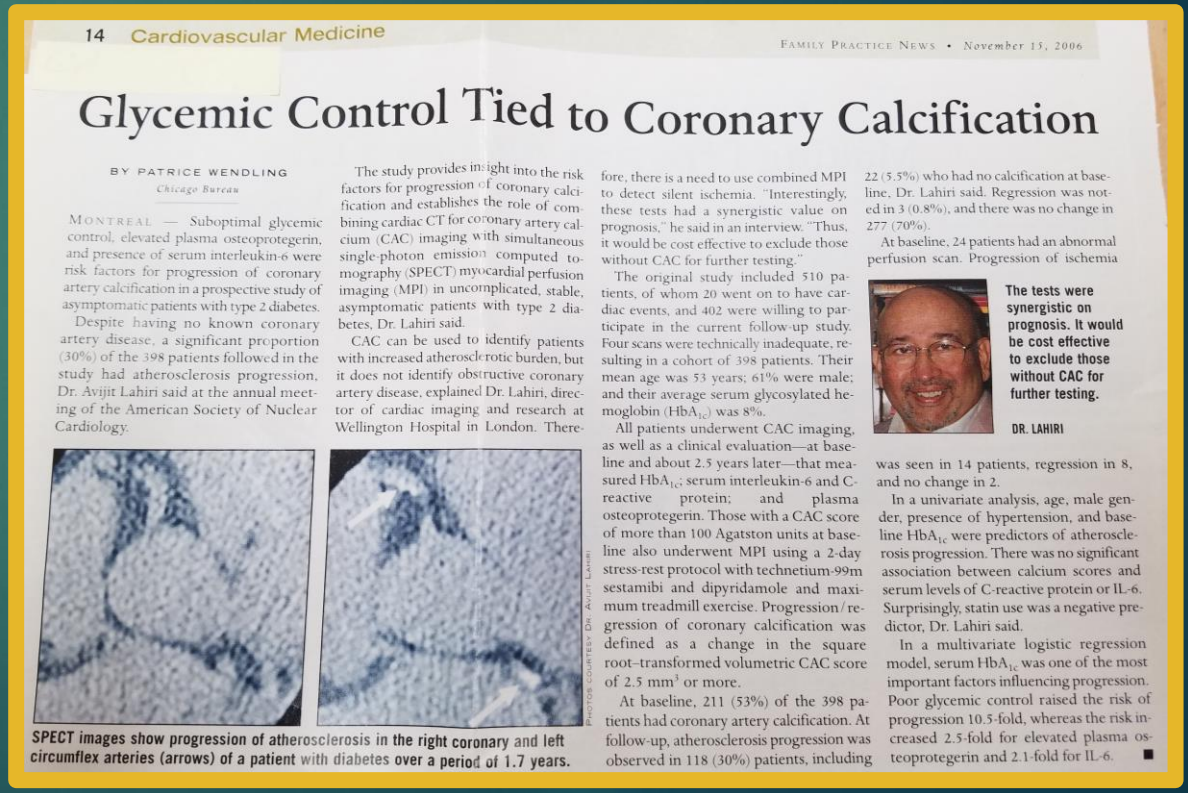


Conclusion 4

- Patients with diabetes demonstrate enhanced efficacy with EDTA chelation. Compared with placebo, EDTA-treated patients demonstrated a 41% reduction in CV endpoints (p=0.0002, 5-year NNT = 7), and a 43% reduction in total mortality (p=0.011, 5-year NNT=12).
- CONTEXT: Statin therapy for secondary prevention (DM): 5 year NNT was 15 for major coronary events.

C. Hancke & K. Flytie (Denmark) J. AdvMed, 1993

- 58 of 65 patients on waiting list for bypass were able to cancel surgery.
- 24 of 27 patients on the waiting list for amputation were able to cancel surgery.



14 Cardiovascular Medicine

FAMILY PRACTICE NEWS • November 15, 2006

Glycemic Control Tied to Coronary Calcification

BY PATRICE WENDLING
Chicago Bureau

MONTREAL — Suboptimal glycemic control, elevated plasma osteoprotegerin, and presence of serum interleukin-6 were risk factors for progression of coronary artery calcification in a prospective study of asymptomatic patients with type 2 diabetes.

Despite having no known coronary artery disease, a significant proportion (30%) of the 398 patients followed in the study had atherosclerosis progression, Dr. Avijit Lahiri said at the annual meeting of the American Society of Nuclear Cardiology.

The study provides insight into the risk factors for progression of coronary calcification and establishes the role of combining cardiac CT for coronary artery calcium (CAC) imaging with simultaneous single-photon emission computed tomography (SPECT) myocardial perfusion imaging (MPI) in uncomplicated, stable, asymptomatic patients with type 2 diabetes, Dr. Lahiri said.

CAC can be used to identify patients with increased atherosclerotic burden, but it does not identify obstructive coronary artery disease, explained Dr. Lahiri, director of cardiac imaging and research at Wellington Hospital in London. Therefore, there is a need to use combined MPI to detect silent ischemia. "Interestingly, these tests had a synergistic value on prognosis," he said in an interview. "Thus, it would be cost effective to exclude those without CAC for further testing."

The original study included 510 patients, of whom 20 went on to have cardiac events, and 402 were willing to participate in the current follow-up study. Four scans were technically inadequate, resulting in a cohort of 398 patients. Their mean age was 53 years; 61% were male; and their average serum glycosylated hemoglobin (HbA_{1c}) was 8%.

All patients underwent CAC imaging, as well as a clinical evaluation—at baseline and about 2.5 years later—that measured HbA_{1c}, serum interleukin-6 and C-reactive protein; and plasma osteoprotegerin. Those with a CAC score of more than 100 Agatston units at baseline also underwent MPI using a 2-day stress-rest protocol with technetium-99m sestamibi and dipyridamole and maximum treadmill exercise. Progression/regression of coronary calcification was defined as a change in the square root–transformed volumetric CAC score of 2.5 mm^3 or more.

At baseline, 211 (53%) of the 398 patients had coronary artery calcification. At follow-up, atherosclerosis progression was observed in 118 (30%) patients, including 22 (5.5%) who had no calcification at baseline, Dr. Lahiri said. Regression was noted in 3 (0.8%), and there was no change in 277 (70%).

At baseline, 24 patients had an abnormal perfusion scan. Progression of ischemia was seen in 14 patients, regression in 8, and no change in 2.

The tests were synergistic on prognosis. It would be cost effective to exclude those without CAC for further testing.

DR. LAHIRI

In a univariate analysis, age, male gender, presence of hypertension, and baseline HbA_{1c} were predictors of atherosclerosis progression. There was no significant association between calcium scores and serum levels of C-reactive protein or IL-6. Surprisingly, statin use was a negative predictor, Dr. Lahiri said.

In a multivariate logistic regression model, serum HbA_{1c} was one of the most important factors influencing progression. Poor glycemic control raised the risk of progression 10.5-fold, whereas the risk increased 2.5-fold for elevated plasma osteoprotegerin and 2.1-fold for IL-6. ■

PHOTOS COURTESY DR. AVIJIT LAHIRI

SPECT images show progression of atherosclerosis in the right coronary and left circumflex arteries (arrows) of a patient with diabetes over a period of 1.7 years.

Available online at www.sciencedirect.com
SCIENCE DIRECT
Free Radical Biology & Medicine 38 (2005) 1565–1570
www.elsevier.com/locate/freeradbiomed

Original Contribution

Acute prooxidant effects of vitamin C in EDTA chelation therapy and long-term antioxidant benefits of therapy☆

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Abstract

Chelation therapy is thought to not only remove contaminating metals but also to decrease free radical production. EDTA chelation therapy, containing high doses of vitamin C as an antioxidant, is often used in the treatment of diseases such as diabetes and cardiovascular diseases but the effectiveness of this treatment may be variable and its efficacy has not been demonstrated conclusively. The objective of this work was to determine if the vitamin C added to standard chelation therapy cocktails was prooxidant. We administered a standard EDTA cocktail solution with or without 5 g of sodium ascorbate. One hour following the standard chelation therapy, there were highly significant prooxidant effects on lipids, proteins, and DNA associated with decreased activities of RBC glutathione peroxidase and superoxide dismutase while in the absence of sodium ascorbate, there were no acute signs of oxidative damage. After 16 sessions of standard chelation therapy, the acute prooxidant effects of vitamin C remained, but, even in the absence of nutrient supplements, there were beneficial long-term antioxidant effects of chelation therapy and plasma peroxide levels decreased. In conclusion, multiple sessions of EDTA chelation therapy protect lipids against oxidative damage. However, standard high amounts of vitamin C added to EDTA chelation solutions also display short term prooxidant effects. The added benefits of lower levels of vitamin C in chelation therapy need to be documented.
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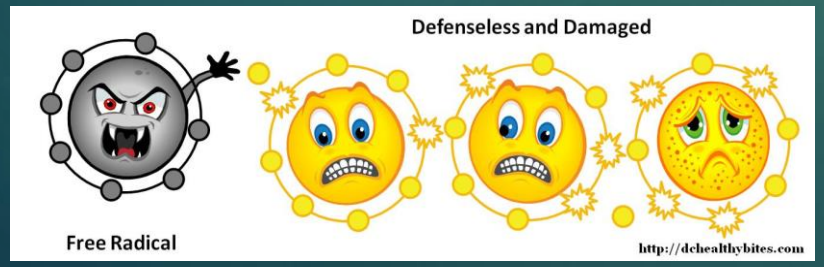
Keywords: Reactive oxygen species; Ascorbic acid; Antioxidant; Prooxidant; EDTA chelation



Original Research

Alternative Medicine Review Volume 14, Number 1 2009

EDTA Chelation Therapy, without Added Vitamin C, Decreases Oxidative DNA Damage and Lipid Peroxidation

Anne Marie Roussel, PhD; Isabelle Hininger-Favier, PhD; Robert S. Waters, MD; Mireille Osman, MS; Karen Fernholz, RN; and Richard A. Anderson, PhD

Abstract
Chelation therapy is thought to not only remove contaminating metals but also to decrease free radical production. However, in standard ethylene diamine tetraacetic acid (EDTA) chelation therapy, high doses of vitamin C with potential pro-oxidant effects are often added to the chelation solution. The authors demonstrated previously that the intravenous administration of the standard chelation cocktail, containing high amounts of vitamin C, resulted in an acute transitory pro-oxidant burst that should be avoided in the treatment of pathologies at risk of increased oxidative stress such as diabetes and cardiovascular disease. The current study was designed to determine the acute and chronic biochemical effects of chelation therapy on accepted clinical, antioxidant variables. An EDTA chelation cocktail not containing ascorbic acid was administered to six adult patients for five weeks (10 sessions of chelation therapy); antioxidant indicators were monitored. Immediately after the initial chelation session, in contrast with the data previously reported with the standard cocktail containing high doses of

Introduction

Antioxidants that decrease cardiovascular risk factors and improve endothelial function[1] are recommended to prevent and treat cardiovascular disease (CVD) and diabetes.[2,3] Ethylene diamine tetraacetic acid (EDTA) chelation therapy, in combination with vitamins and minerals, is proposed to have antioxidant properties[4] and considered to be a complementary therapy for patients with coronary artery disease.[5] However, this claim is controversial.[6,7] A recent review evaluating EDTA chelation therapy in treating cardiovascular diseases did not support the evidence of beneficial effects.[8] No clinical trials with a large enough study group have been completed to provide sufficient statistical power to determine the clinical effectiveness of EDTA chelation therapy.

EDTA might act to prevent metal-induced free radical production, but the direct *in vivo* effects of EDTA chelation therapy on oxidative stress markers

Biol Trace Elem Res (2008) 124:243–250
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Intravenous Magnesium Sulfate With and Without EDTA as a Magnesium Load Test—Is Magnesium Deficiency Widespread?

Robert S. Waters · Karen Fernholz · Noella A. Bryden · Richard A. Anderson

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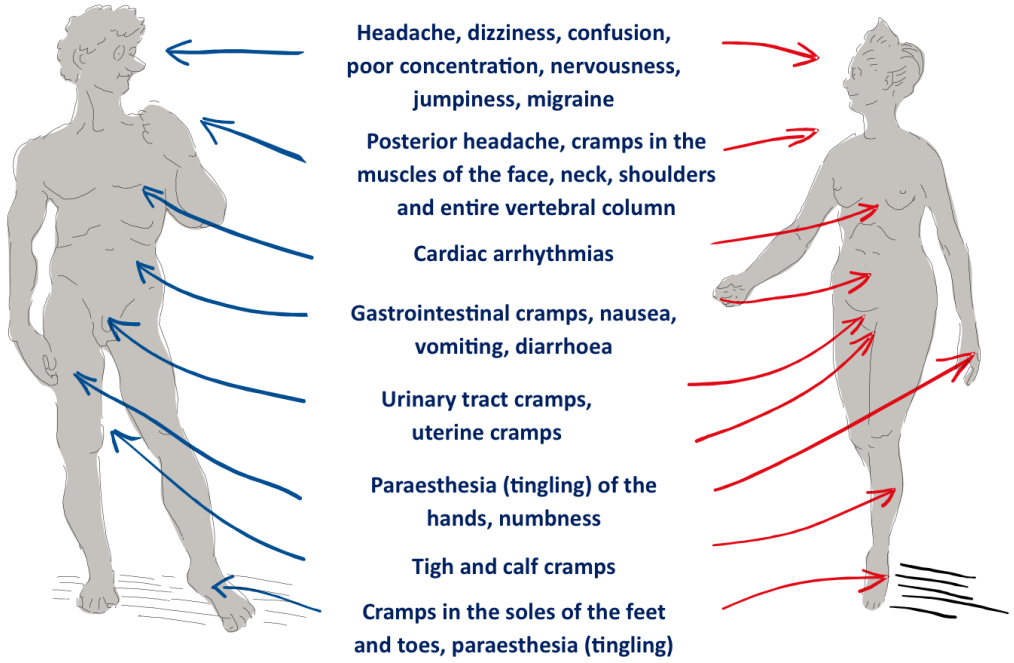
Abstract Serum/plasma measurements do not reflect magnesium deficits in clinical situations, and magnesium load tests are used as a more accurate method to identify magnesium deficiency in a variety of disease states as well as in subclinical conditions. The objective of this study was to determine if people are indeed magnesium deficient or if the apparent magnesium deficiency is due to the composition of the infusate used in the load test. Magnesium load tests were performed on seven patients using three different Mg solution infusions—a Mg–EDTA (ethylene diamine tetraacetic acid)-nutrient cocktail used in EDTA chelation therapy containing several components including vitamins and minerals, and the same cocktail without EDTA and an infusion of an identical amount of magnesium in normal saline solution. There was no significant difference in the amount of magnesium retained in the 24 h after infusion among the three infusates. All infusates resulted in very high magnesium retention compared to previous published magnesium load studies. Magnesium deficiency may be widespread, and the relationship of Mg deficiency to related diseases requires further study.

Keywords Magnesium deficiency · EDTA · Metabolic syndrome · Magnesium status · Chelation therapy · Magnesium load test

Symptoms of magnesium deficiency



Magnesium deficiency is universal in insulin resistance syndrome

- Magnesium is required for ATP energy transfer in every biochemical process in all cells of the body
- Magnesium regulates the proper dilatation of the blood vessels to deliver oxygen and nutrients to every cell
- Magnesium participates in calcium chemistry by preventing accumulation of that element in the intracellular space. Calcium accumulation in cellular membranes is the basis of aging
- In addition, magnesium activates the sodium-potassium ATPase pump that controls the balance of electrolytes in cells
- Lowering intracellular magnesium concentration decreases cellular glucose utilization and thus results in peripheral insulin resistance

These effects explain why deficiency of magnesium is related to oxidative stress, proinflammatory state, endothelial dysfunction, platelet aggregation, insulin resistance, hypertension and hyperglycemia

Magnesium deficiency and metabolic syndrome: stress and inflammation may reflect calcium activation

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Abstract. Magnesium (Mg) intake is inadequate in the western diet and metabolic syndrome is highly prevalent in populations around the world. Epidemiological studies suggest that high Mg intake may reduce the risk but the possibility of confounding factors exists, given the strong association between Mg and other beneficial nutriments (vegetables, fibers, cereals). The concept that metabolic syndrome is an inflammatory condition may explain the role of Mg.

Mg deficiency results in a stress effect and increased susceptibility to physiological damage produced by stress. Stress activates the hypothalamic-pituitary-adrenal axis (HPA) axis and the sympathetic nervous system. The activation of the renin-angiotensin-aldosterone system is a factor in the development of insulin resistance by increasing oxidative stress. In both humans and rats, aldosteronism results in an immunostimulatory state and leads to an inflammatory phenotype. Stress response induces the release of large quantities of excitatory amino acids and activates the nuclear factor NFκB, promoting translation of molecules involved in cell regulation, metabolism and apoptosis. The rise in neuropeptides is also well documented. Stress-induced HPA activation has been identified to play an important role in the preferential body fat accumulation but evidence that Mg is involved in body weight regulation is lacking. One of the earliest events in the acute response to stress is endothelial dysfunction. Endothelial cells actively contribute to inflammation by elaborating cytokines, synthesizing chemical mediators and expressing adhesion molecules. Experimental Mg deficiency in rats induces a clinical inflammatory syndrome characterized by leukocyte and macrophage activation, synthesis of inflammatory cytokines and acute phase proteins, extensive production of free radicals. An increase in extracellular Mg concentration decreases inflammatory effects, while reduction in extracellular Mg results in cell activation. The effect of Mg deficiency in the development of insulin resistance in the rat model is well documented. Inflammation occurring during experimental Mg deficiency is the mechanism that induces hypertriglyceridemia and pro-atherogenic changes in lipoprotein metabolism. The presence of endothelial dysfunction and dyslipidemia triggers platelet aggregability, thus increasing the risk of thrombotic events. Oxidative stress contributes to the elevation of blood pressure. The inflammatory syndrome induces activation of several factors, which are dependent on cytosolic Ca activation.

Presented in part at the XII[th] International Magnesium Symposium held to Iasi, Romania, 23-25 September 2009.

Studies are great…
but let's look at real people.

How can we tell if chelation is working?

- Laboratory Data improves
- Scans improve
- Wounds Heal
- More Energy

Case of N.B.

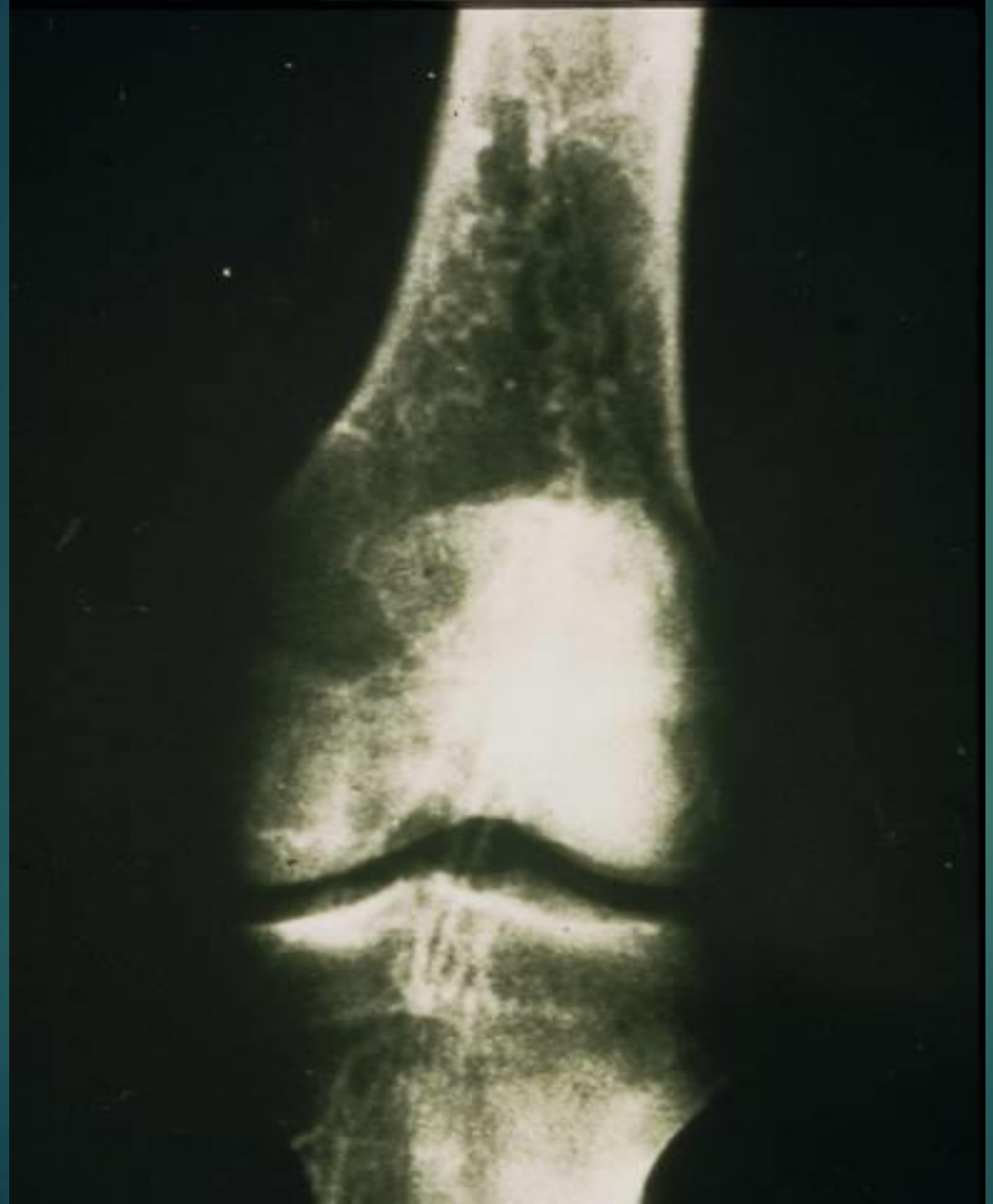
	3/5/80 Before Chelation	1/17/89 After 70 Chelations
Thallium Treadmill:	Non-reversible ischemic zones in the intraventricular septum and portions of the inferior wall of the left ventricle.	Normal exam after peak stress
Treadmill EKG:	ST segment depression B.P. start = 170/95 B.P. at 2 minutes = 240/100	No ST segment depression B.P. start = 132/80 B.P. at 2 minutes = 155/80

Lab Data for Marilyn S.

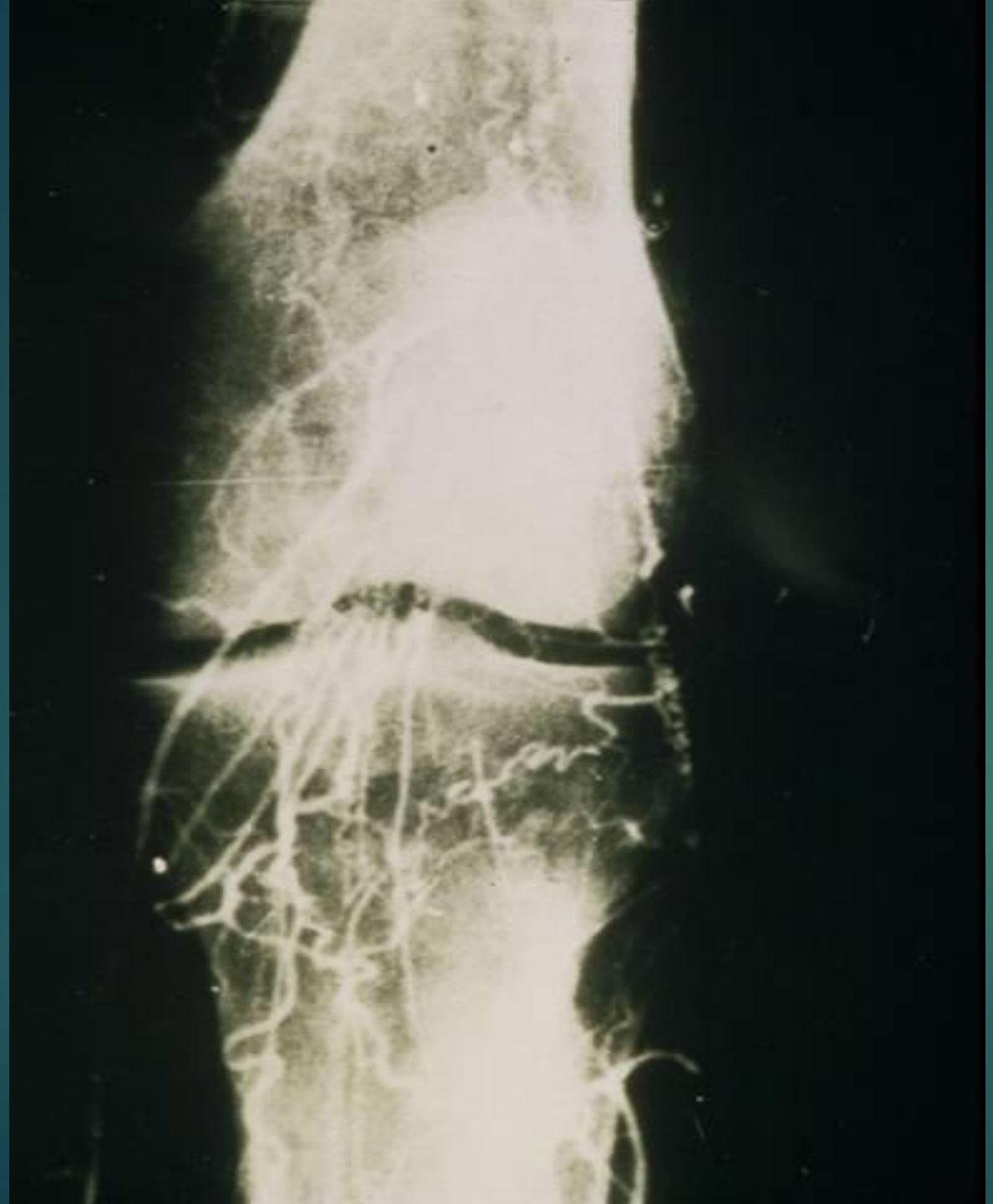
	11/1/1995	1/17/1996
Total Cholesterol	393	165
HDL Cholesterol	33.3	45.6
Cholesterol/HDL	11.8	3.6
Triglycerides	2,647	271

Lab Data for Fred S.

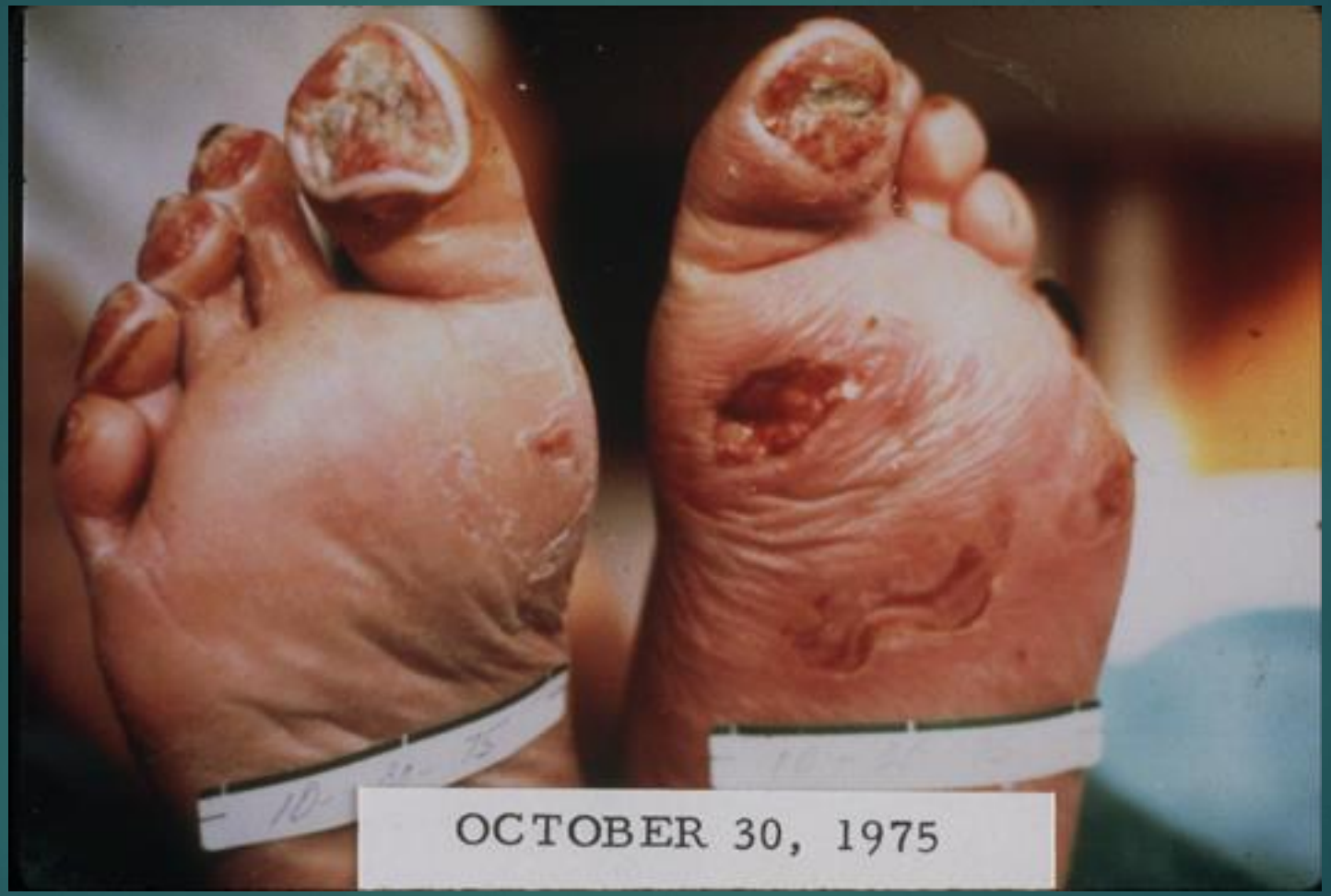
	3/16/2015	8/27/2015
Hemoglobin A1C	12.2	5.7
Blood Sugar	266	97
Ferritin	565	110
C-Reactive Protein	4.1	2.1



This is an angiogram of the leg - note the tiny amount of blood going past the knee joint



This is an angiogram after chelation therapy treatments - note the increase in blood flow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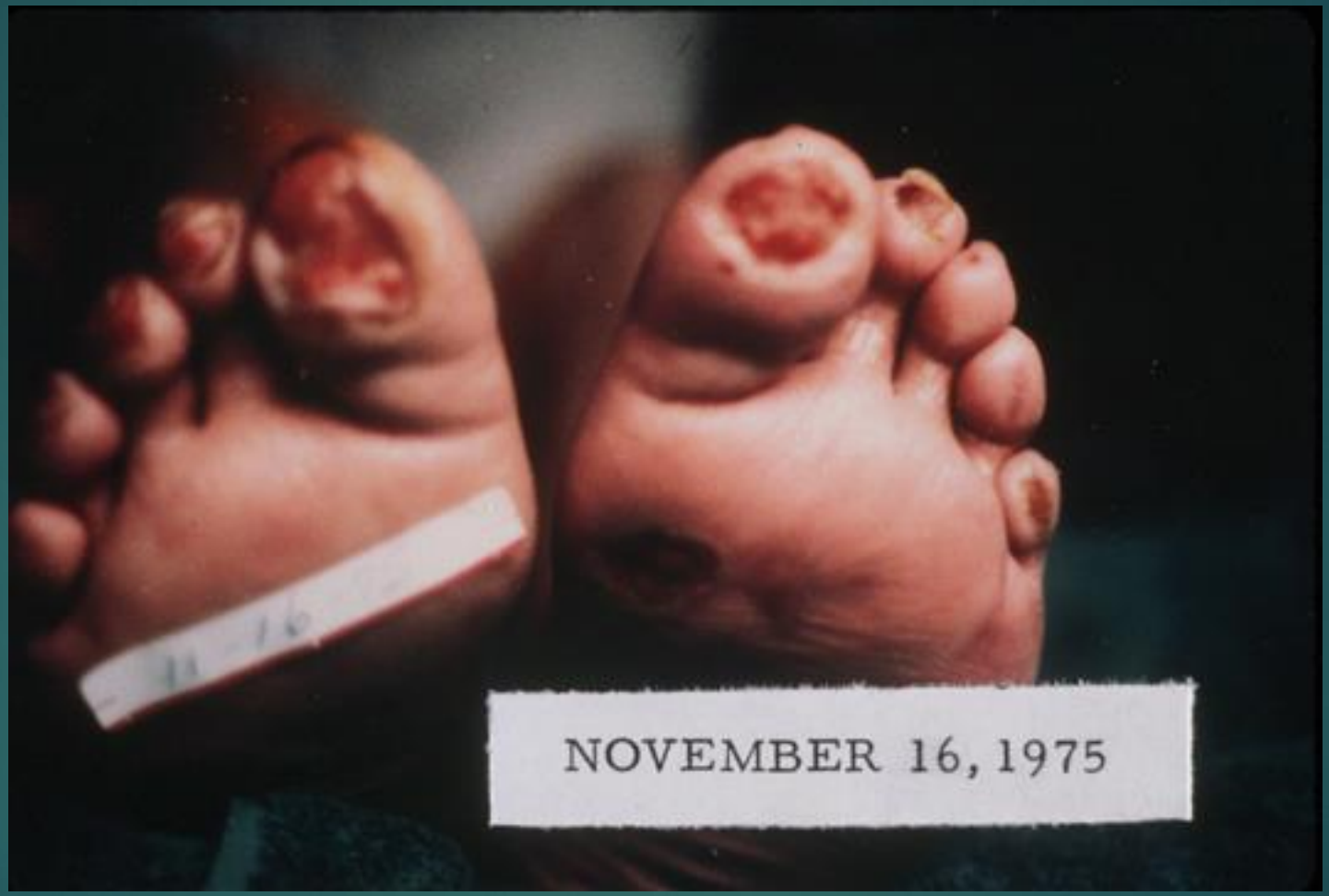
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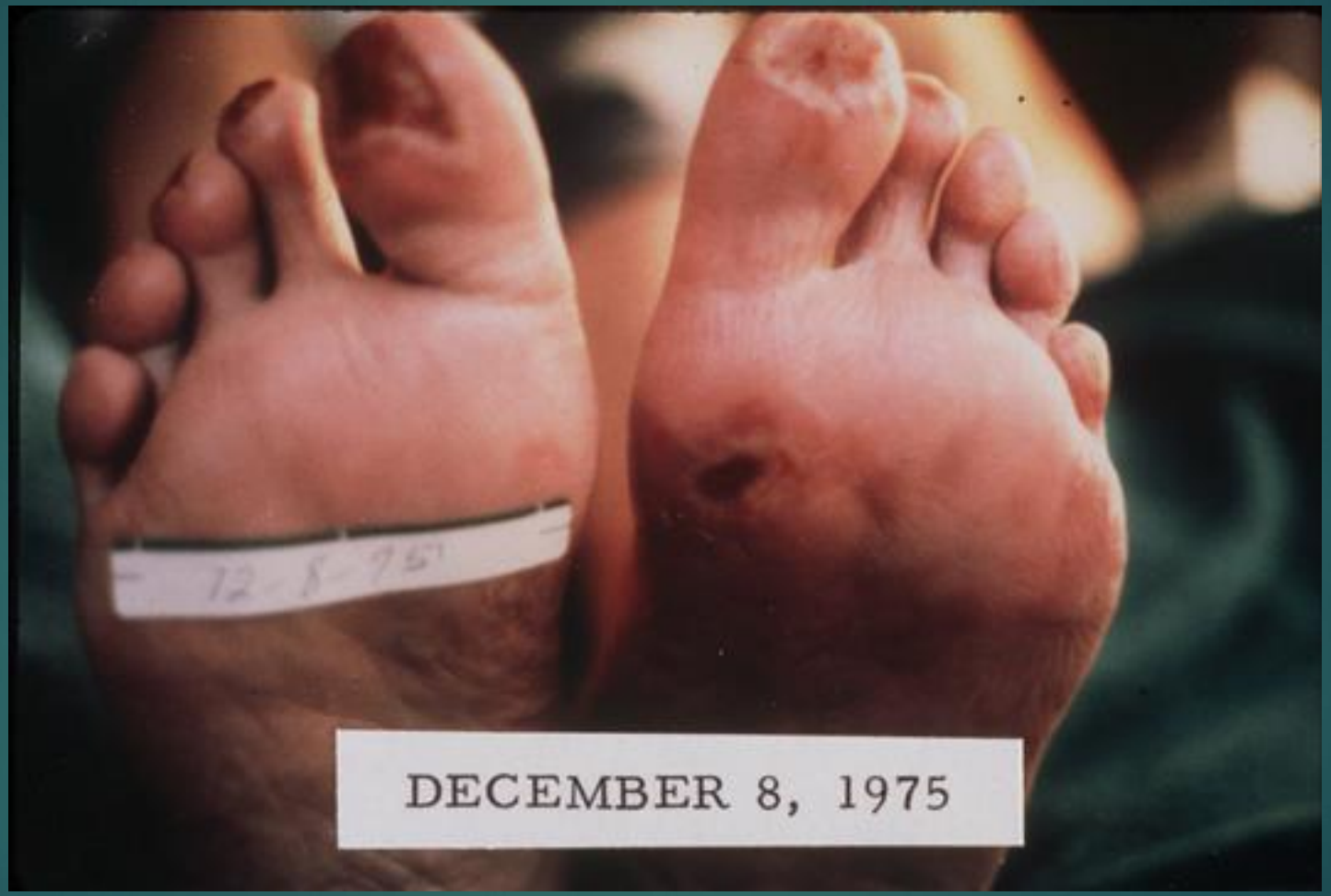
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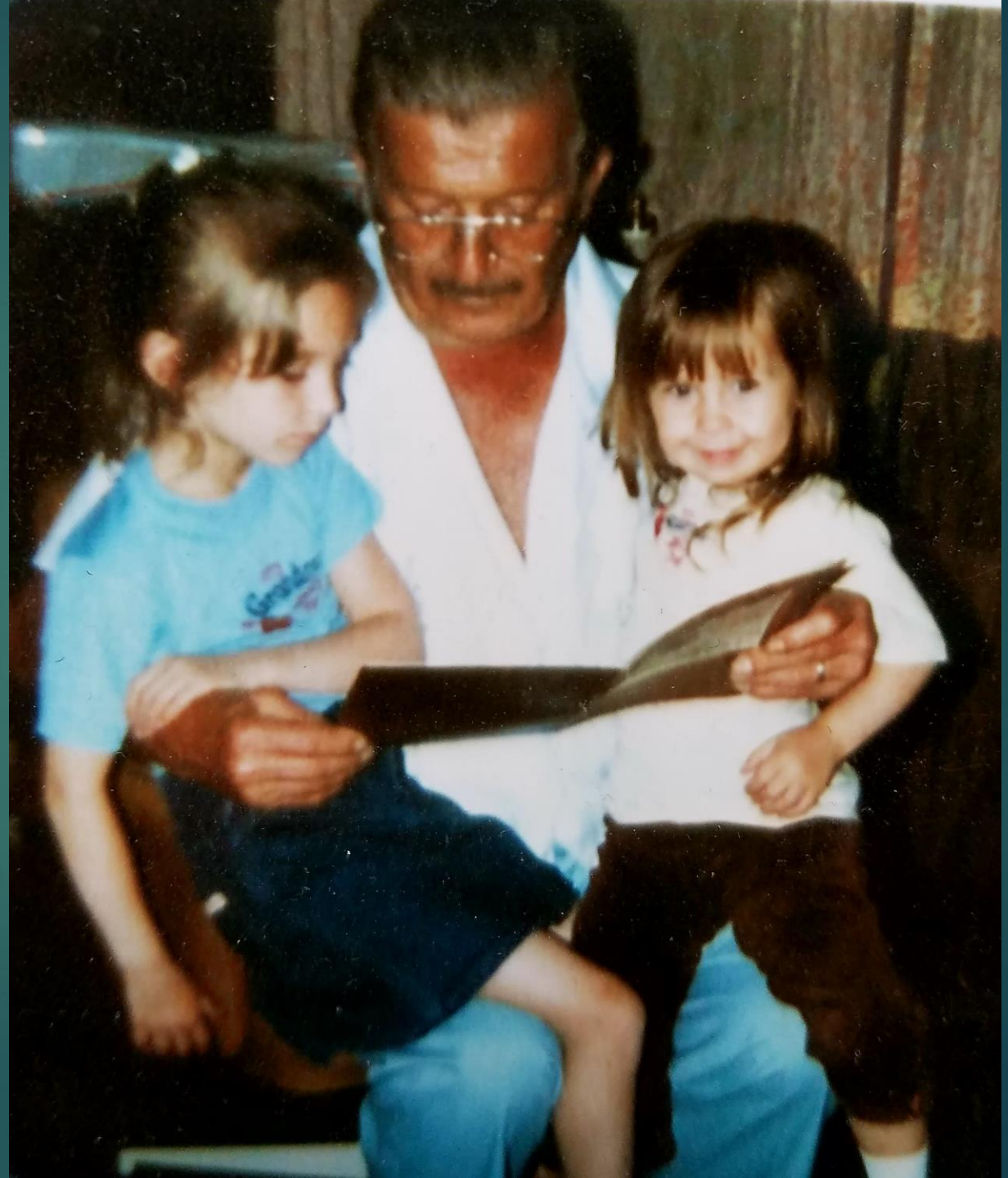
Why do I offer Chelation Therapy?

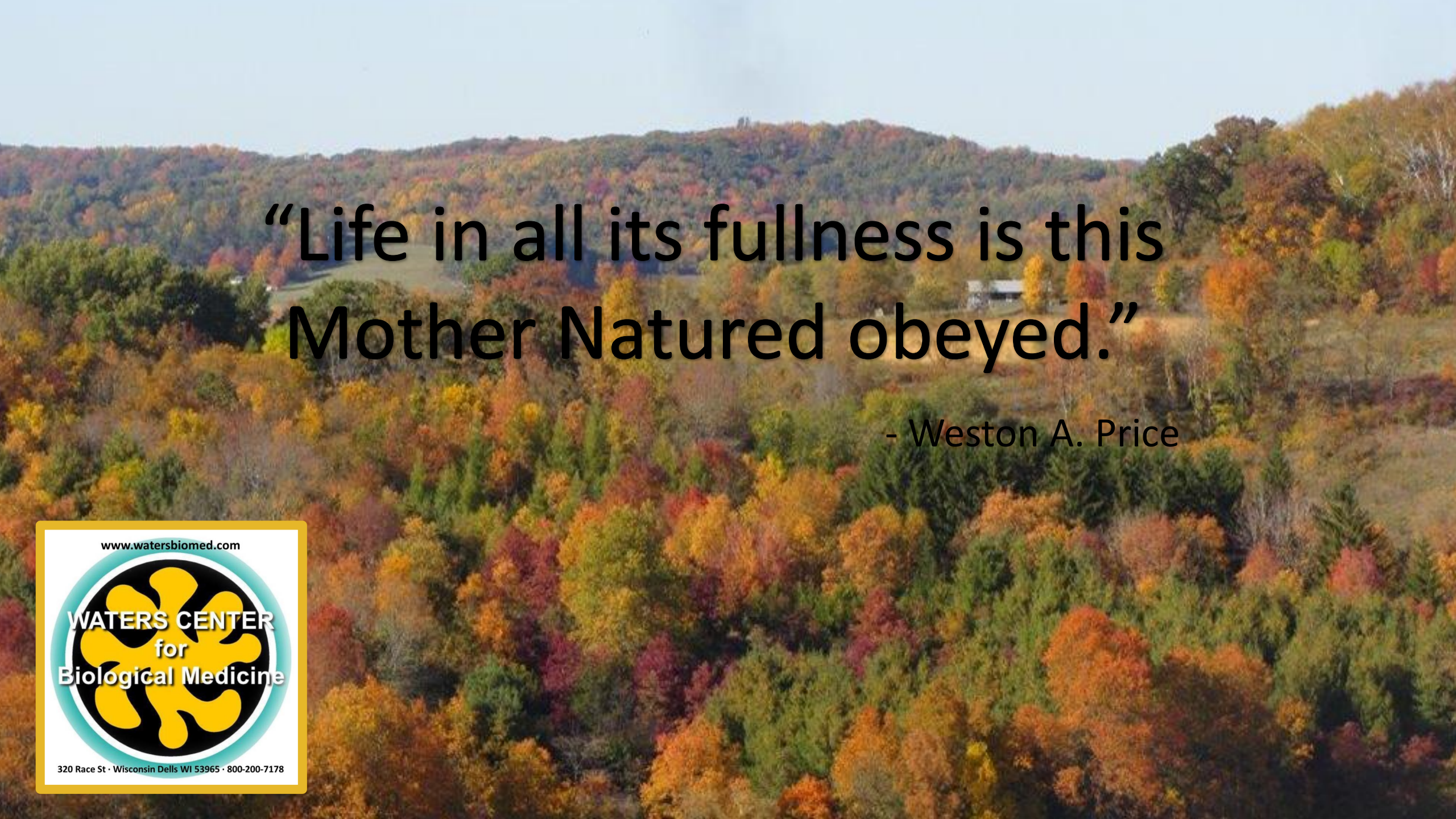


“When health is absent, wisdom cannot reveal itself, art cannot manifest, strength cannot fight, wealth becomes useless, and intelligence cannot be applied.”

~HEROPHILUS 300 BC

Our 9-pronged Program to Reverse Insulin Resistance and Restore Health

1. Low carbohydrate diet – in some cases a ketogenic diet is the most appropriate and the fastest way to reverse insulin resistance and feel well again
2. Begin a regular exercise or body movement program
3. Correct nutritional deficiencies
4. Decrease inflammation and free radical damage with botanical medicine
5. Balance and correct all endocrine gland disorders – thyroid, adrenal glands and sex hormones
6. Normalize sleep
7. Address other lifestyle and environmental stresses
8. Detoxification of organic chemicals with sauna
9. EDTA Chelation Therapy – this treatment addresses all of the components of the diseases of aging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Mother Natured obeyed."
- Weston A. Price
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