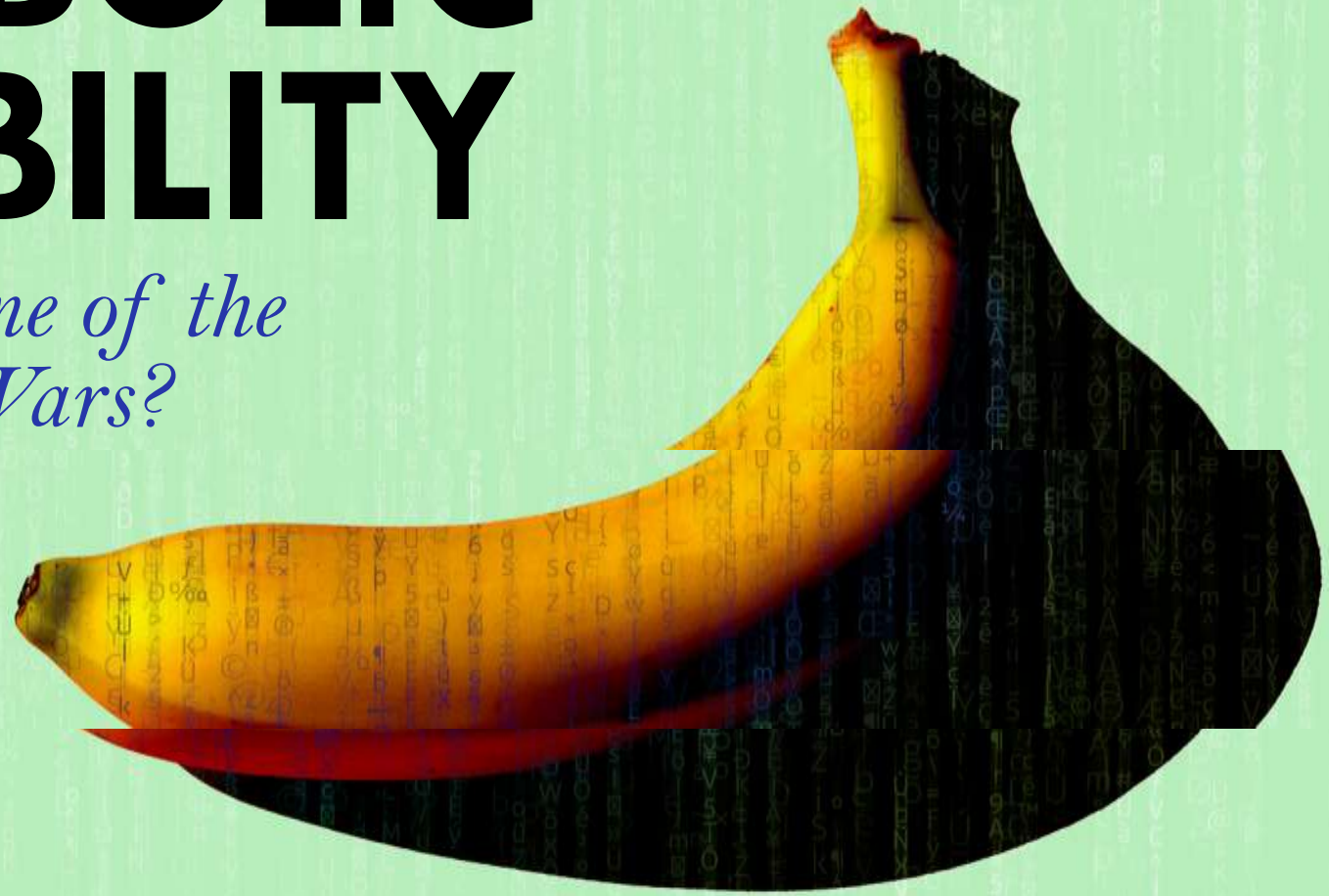
METABOLIC FLEXIBILITY

The Rosetta Stone of the Macronutrient Wars?



ROBB WOLF *revolutionary solutions to modern life*

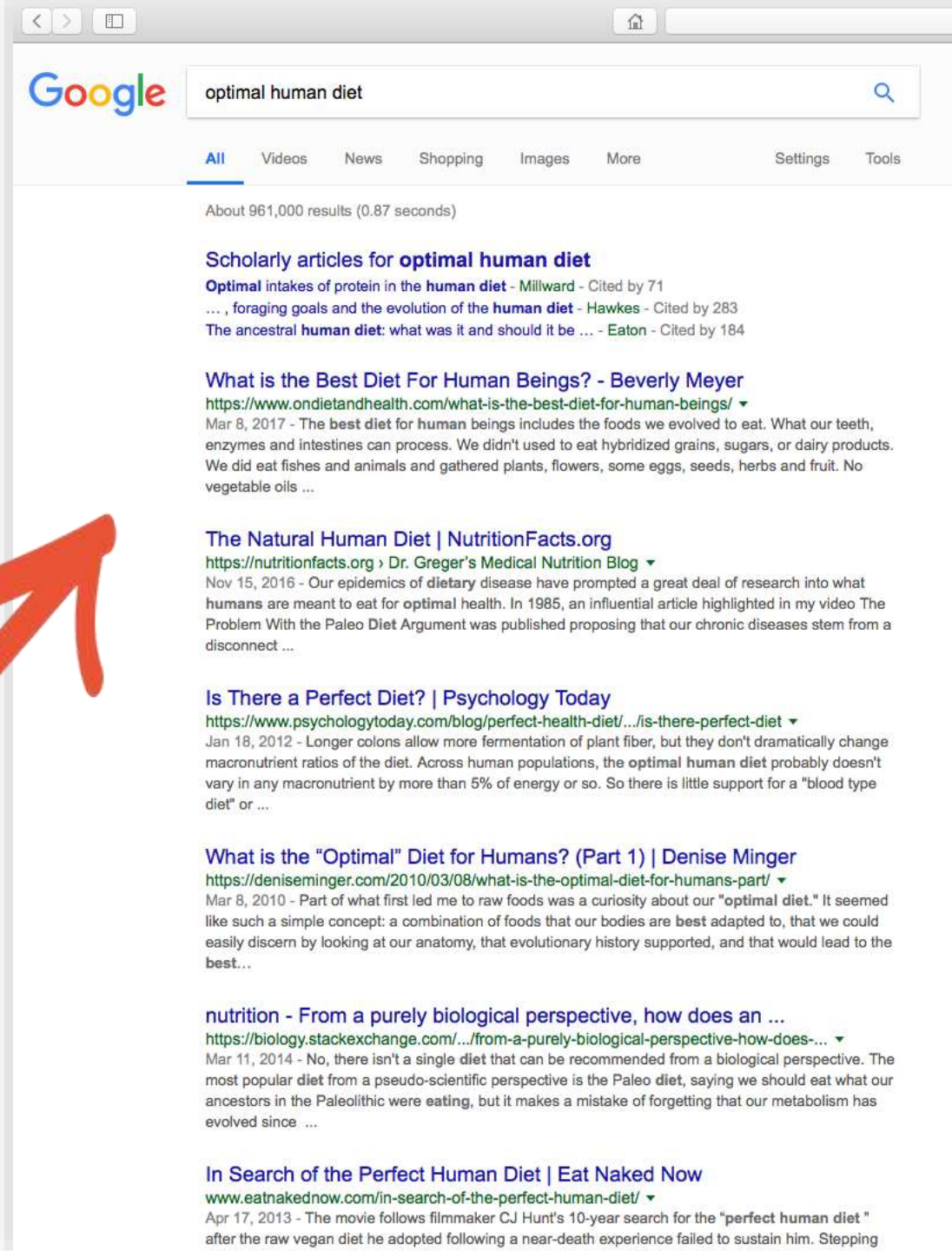
optimal

optimal
optimal **definition**
optimal **blue**
optimal **outbreeding**
optimal **health**
optimal**ly**
optimal **health systems**
optimal **resume**
optimal **nutrition**
optimal **arousal theory**

Only the fringes care about optimal?

Seems a reasonable question, but *really hard to pin down!*

NOTHING in Pubmed



Is there an **OPTIMAL ancestral diet?**

Cultures with “diets” that work

Inuit

Kitavan

Image Source: MVOrionKitava CC BY-SA 3.0, https://en.wikipedia.org/w/index.php?curid=6701695

Okinawan

“Blue-zones”

Images Source:
U.S. Air Force photo/Tech. Sgt. Rey Ramon, http://www.kadena.af.mil/News/Article-Display/Article/418168/okinawan-ancestors-rejoin-families-during-obon/
Anziano Sardo by Jean Bajean - https://www.flickr.com/photos/jeanbajean/4095162162/sizes/o/in/photostream/, CC BY-SA 2.5, https://commons.wikimedia.org/w/index.php?curid=27437962

Commonalities

Largely whole **unprocessed foods**

Many common **lifestyle features** *(much more on this later)*

Oddly missing?
~~**Macronutrients**~~

Outcomes

Population is largely **free of Western degenerative disease**

Cultures with “diets” that *do not* work

US *(Why??)*

Processing

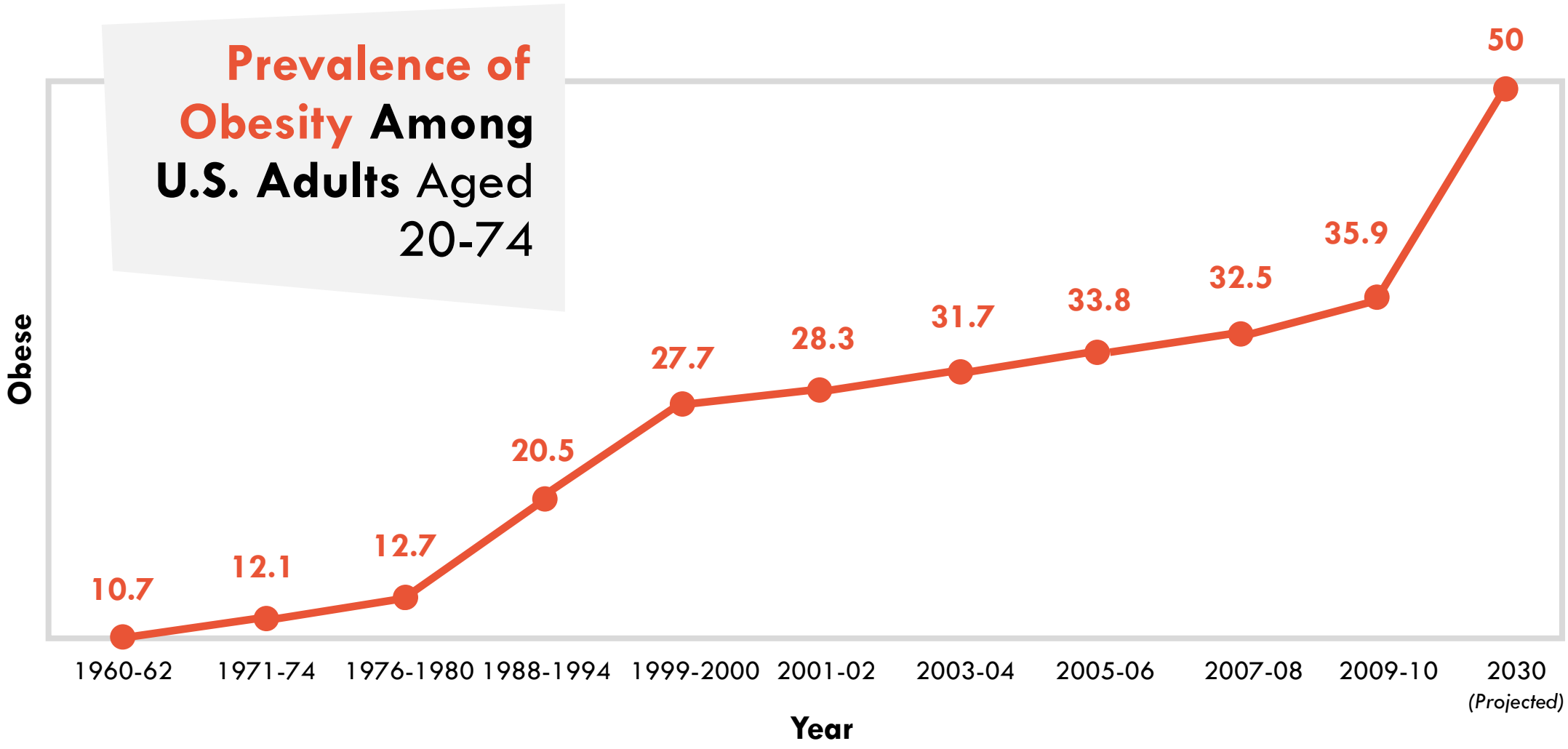
Increasing **palate complexity**

Dedicated engineering to make food **hyperpalatable**

(Betcha can't eat just one)

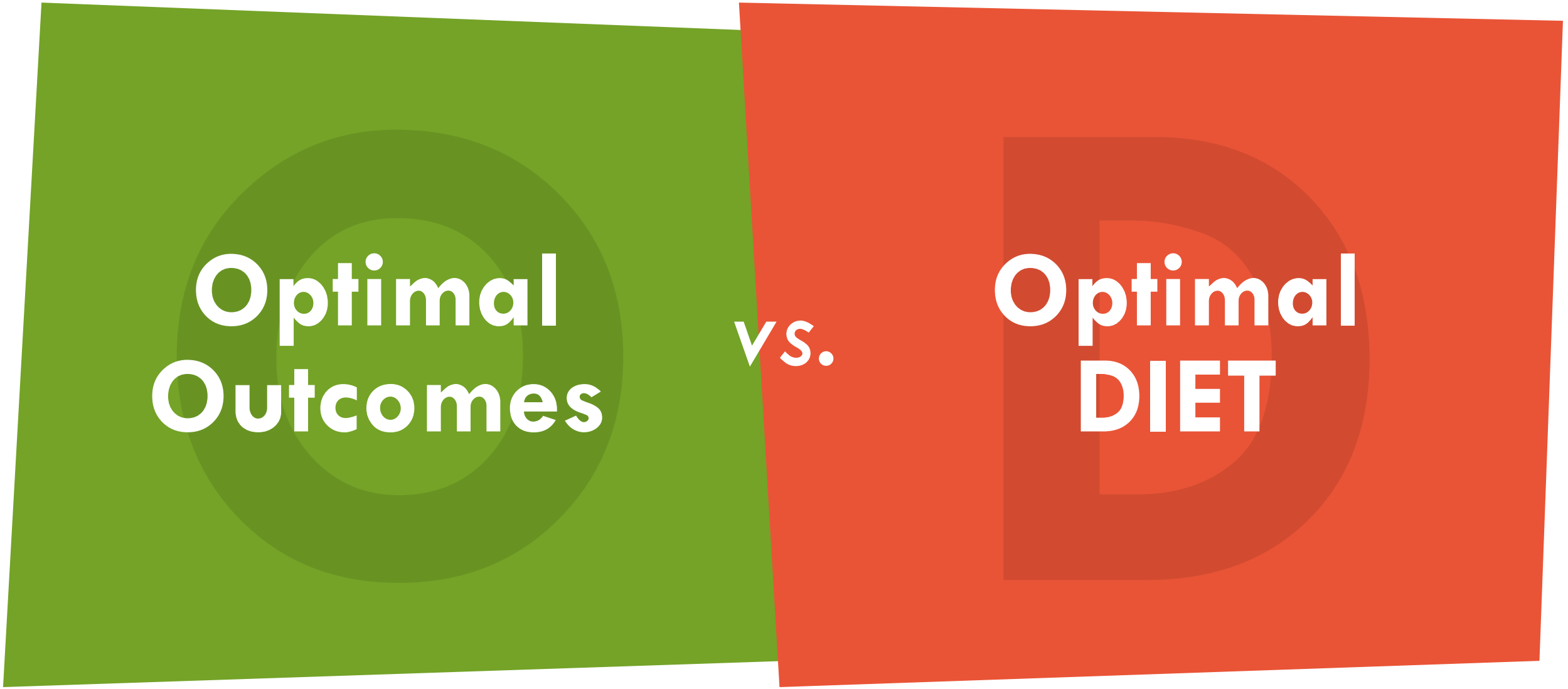

TITANFALL 2
UNLOCK 2XP
+EXCLUSIVE TITAN CONTENT
DEWANDDORITOS.COM
Doritos
BRAND
GUARANTEED FRESH
UNTIL PRINTED DATE
Roulette
ATTENTION
SOME CHIPS ARE VERY HOT
FLAVORED TORTILLA CHIPS
NET WT. 3 OZ. (85 g)
ATTENTION
SOME CHIPS ARE VERY HOT

Outcomes



Reference: Derived from NHANES data http://www.cdc.gov/nchs/data/hestat/obesity_adult_09_10/obesity_adult_09_10.html#table1

Time for

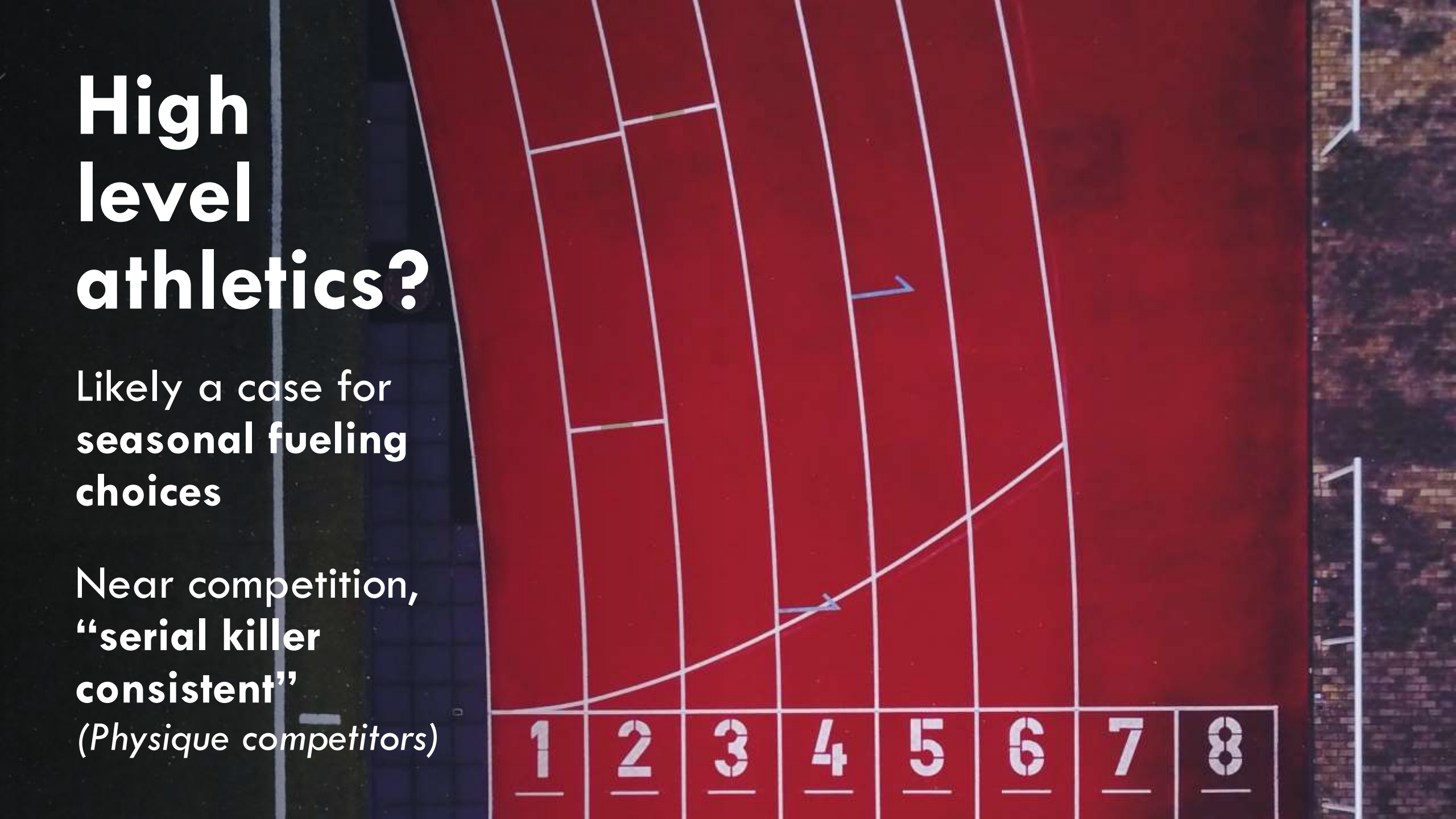


When might a **set macro ratio** make sense?

High level athletics?

Likely a case for **seasonal fueling choices**

Near competition, **“serial killer consistent”**
(Physique competitors)



Illness/conditions that likely benefit from Low Carb

Gut Disbiosis

Mitochondrial Insufficiency

IR/substrate depletion

Key to fat loss?

Appetite *suppression*

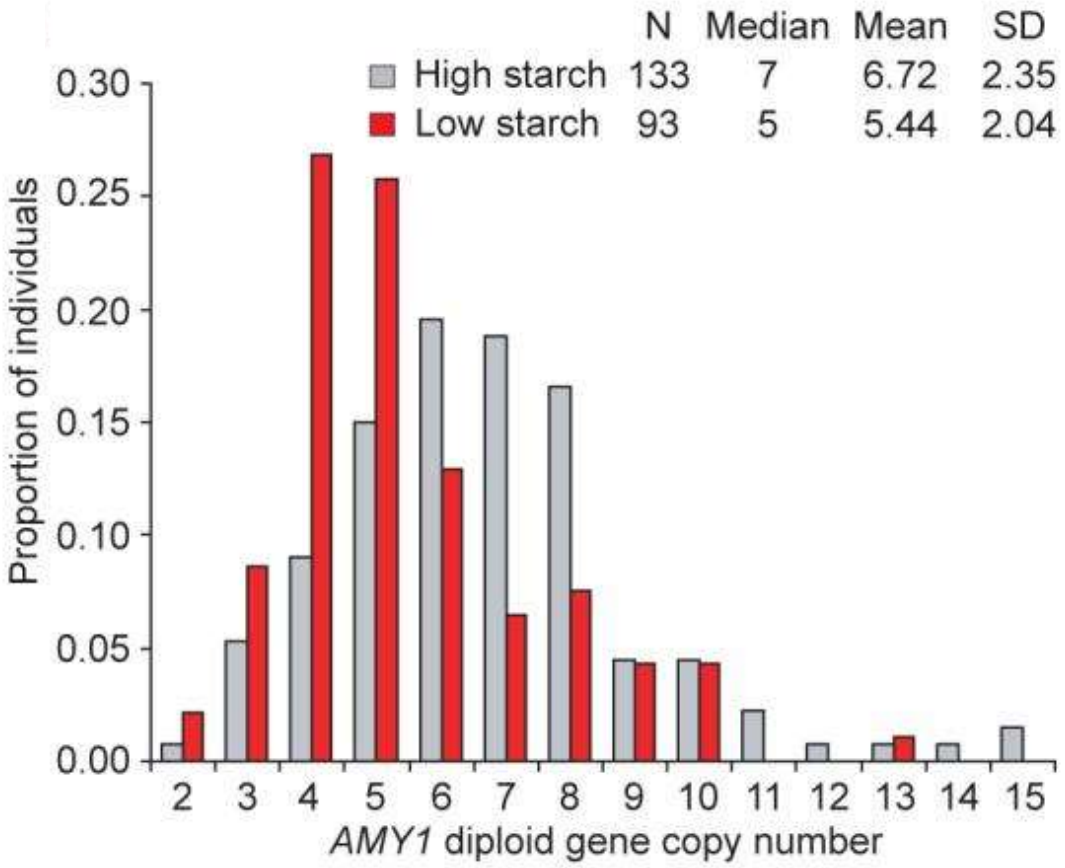
The assumption we must make:

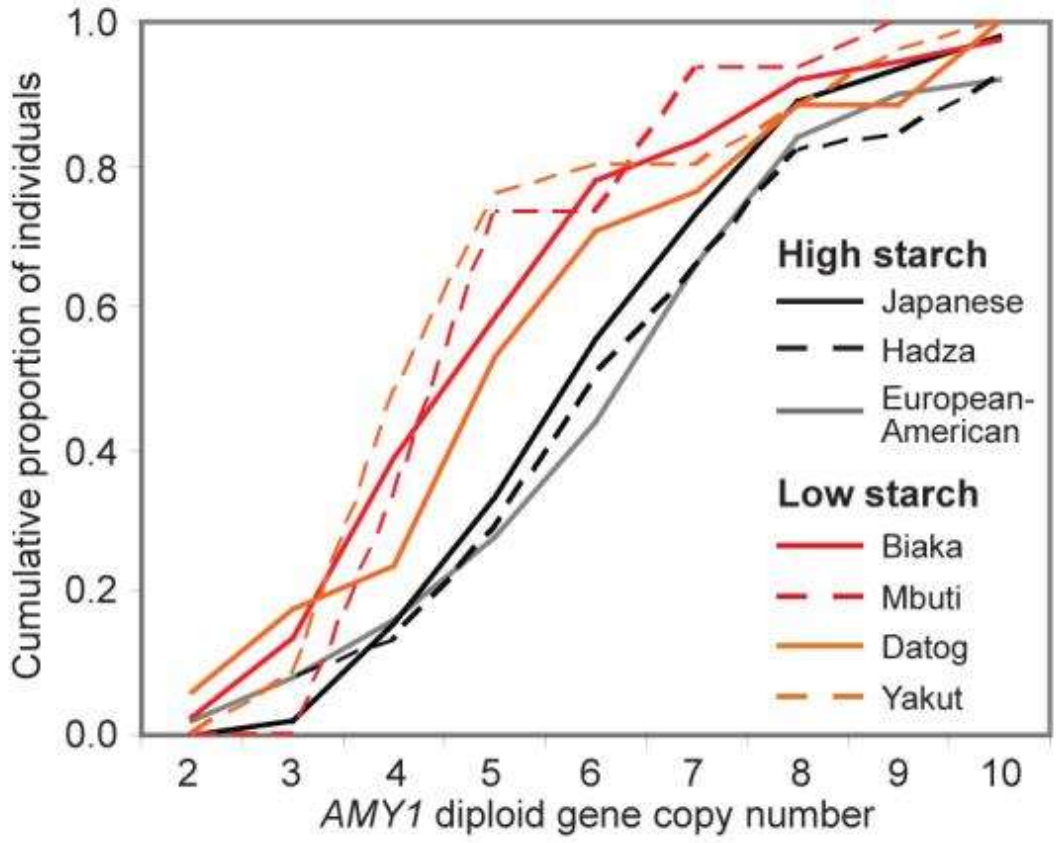
If there is an optimal diet for humans (LC? HC?) we should **see metabolic and genetic predilections FOR this specific approach.**

The Case for Carbs

Humans SHOULD be able to eat carbs,

likely more than all other primates!



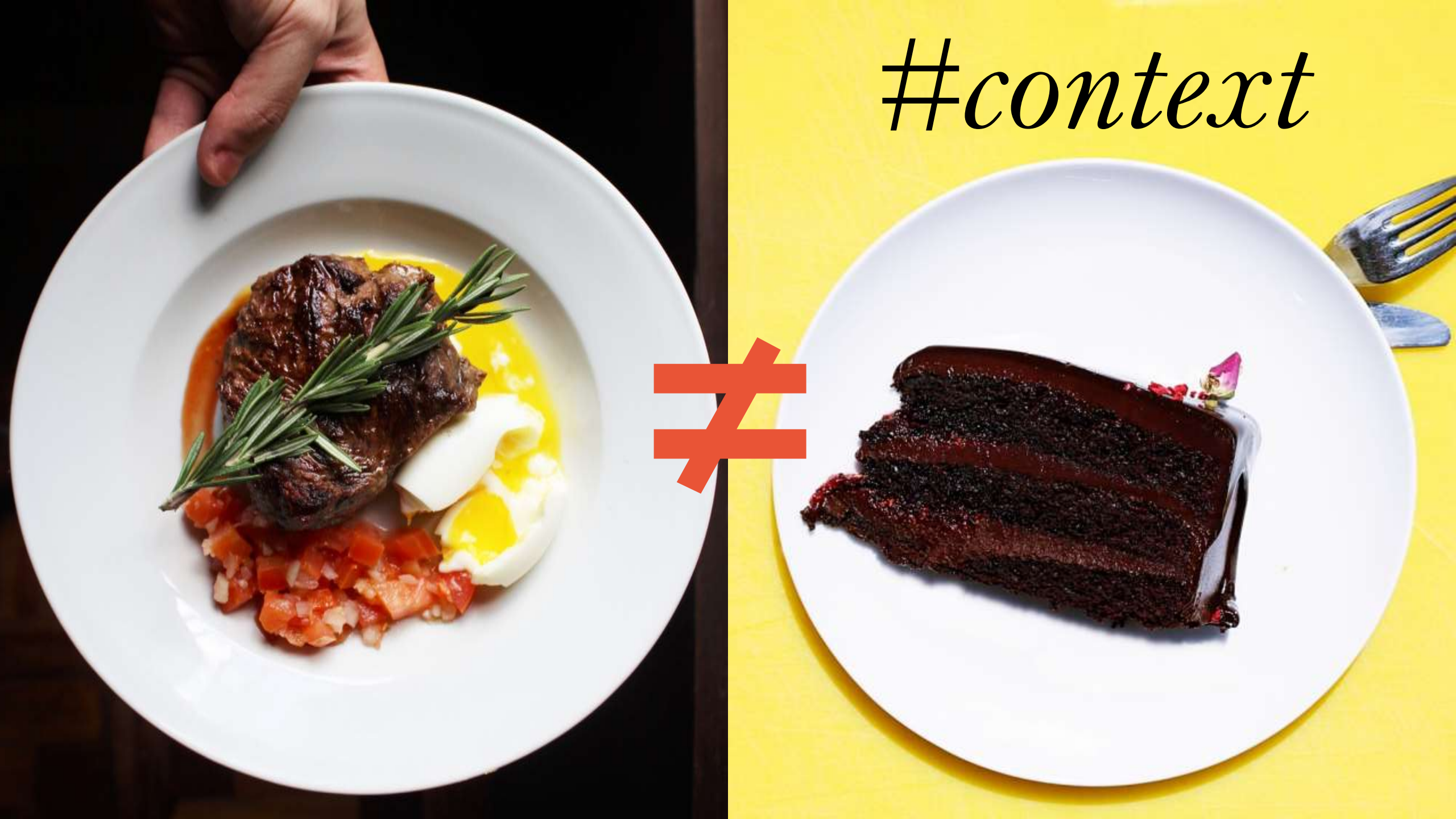


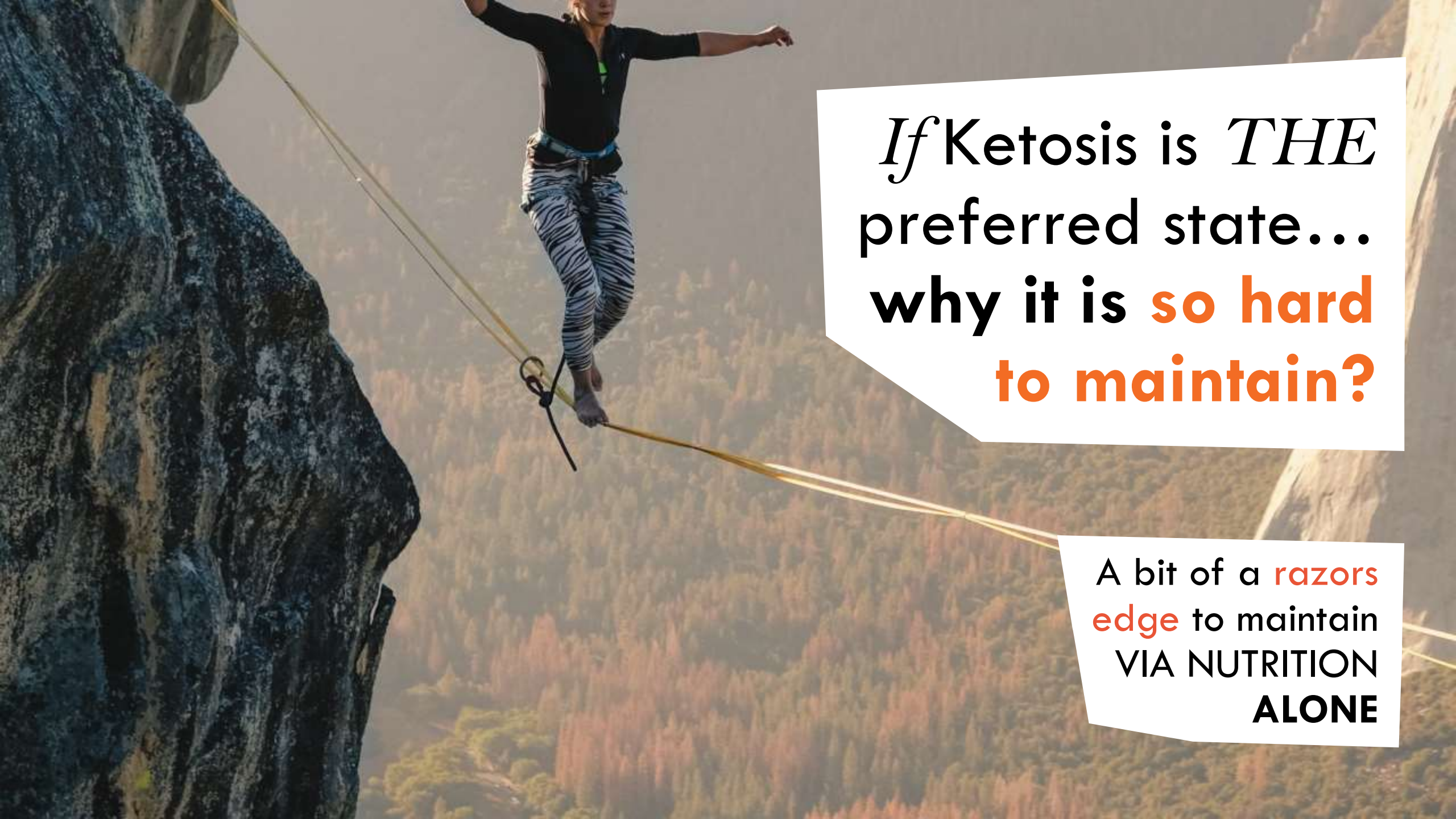
So,
what about
Ketosis?

The Case Against Ketosis, pt. 1

Body actively "tries"
to **get out of Ketosis**

What are the **recommended carb levels per day?**


#context
≠


If Ketosis is *THE* preferred state…
why it is so hard to maintain?
A bit of a razors edge to maintain VIA NUTRITION **ALONE**

The Case Against Ketosis, pt. 2

The Inuit largely **do not USE Ketosis!**

CPT-1a

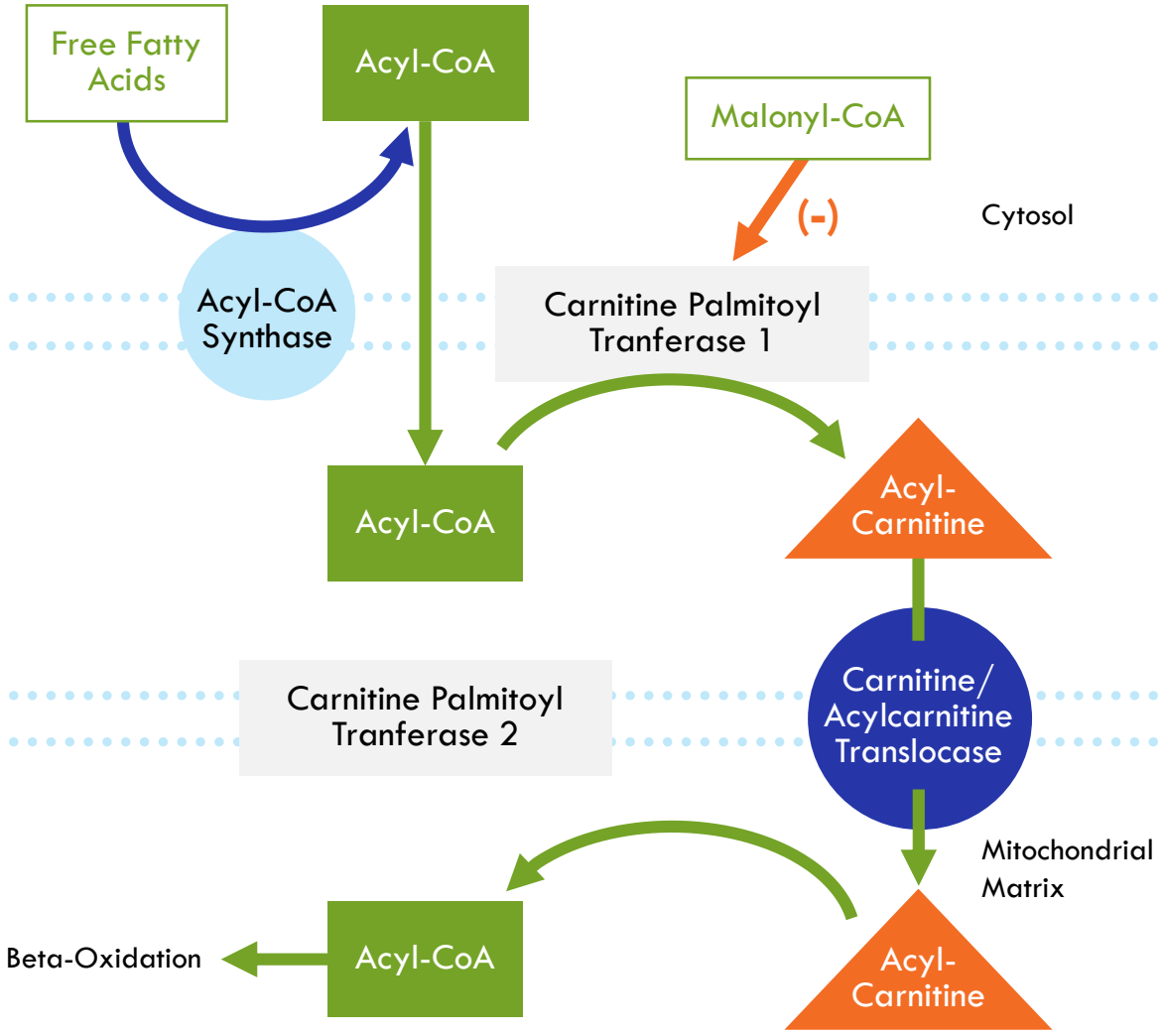
(Carnitine palmitoyltransferase)

Increases **hypoglycemia**

Largely **blocks ability to enter ketosis**

3X increase in **infant mortality!**

Stunning speed of **gene spread**



Mitochondrial carnitine palmitoyltransferase system
Carntine Palmitoyl Transferase (CPT1 and CPT2). The fatty acids are transferred from cell cytoplasm to mitochondrial matrix for beta-oxidation. The CPT1 is activity is regulated by malonyl-CoA feedback inhibition.

Evolutionary impacts on health might be more prevalent than currently appreciated.

– *Florian Clemente*

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News / Gene that once aided survival in the Arctic is found to have negative impact on health today

Discussion | Video and audio | Spotlight on... | Research at Cambridge | Innovation at Cambridge | Research Impact

Gene that once aided survival in the Arctic is found to have negative impact on health today

Millennia-old genetic variant that once provided advantages for survival in cold climates increases risk of hypoglycemia and infant mortality.

In individuals living in the Arctic, researchers have discovered a genetic variant that arose thousands of years ago and most likely provided an evolutionary advantage for processing high-fat diets or for surviving in a cold environment; however, the variant also seems to increase the risk of hypoglycemia, or low blood sugar, and infant mortality in today's northern populations.

> *Evolutionary impacts on health might be more prevalent than currently appreciated*
>
> — *Florian Clemente*

The findings, published online in the *American Journal of Human Genetics*, provide an example of how an initially beneficial genetic change could be detrimental to future generations.

Published

24 Oct 2014

Image

Ig-loos or Snow Villages at Oo-pung-ne-wing

Credit: Toronto Public Library

Share

Email 1 | reddit 0 | Share 0 | Share 0 | Tweet 2 | Share 60 | Like 0

Subjects

Arctic

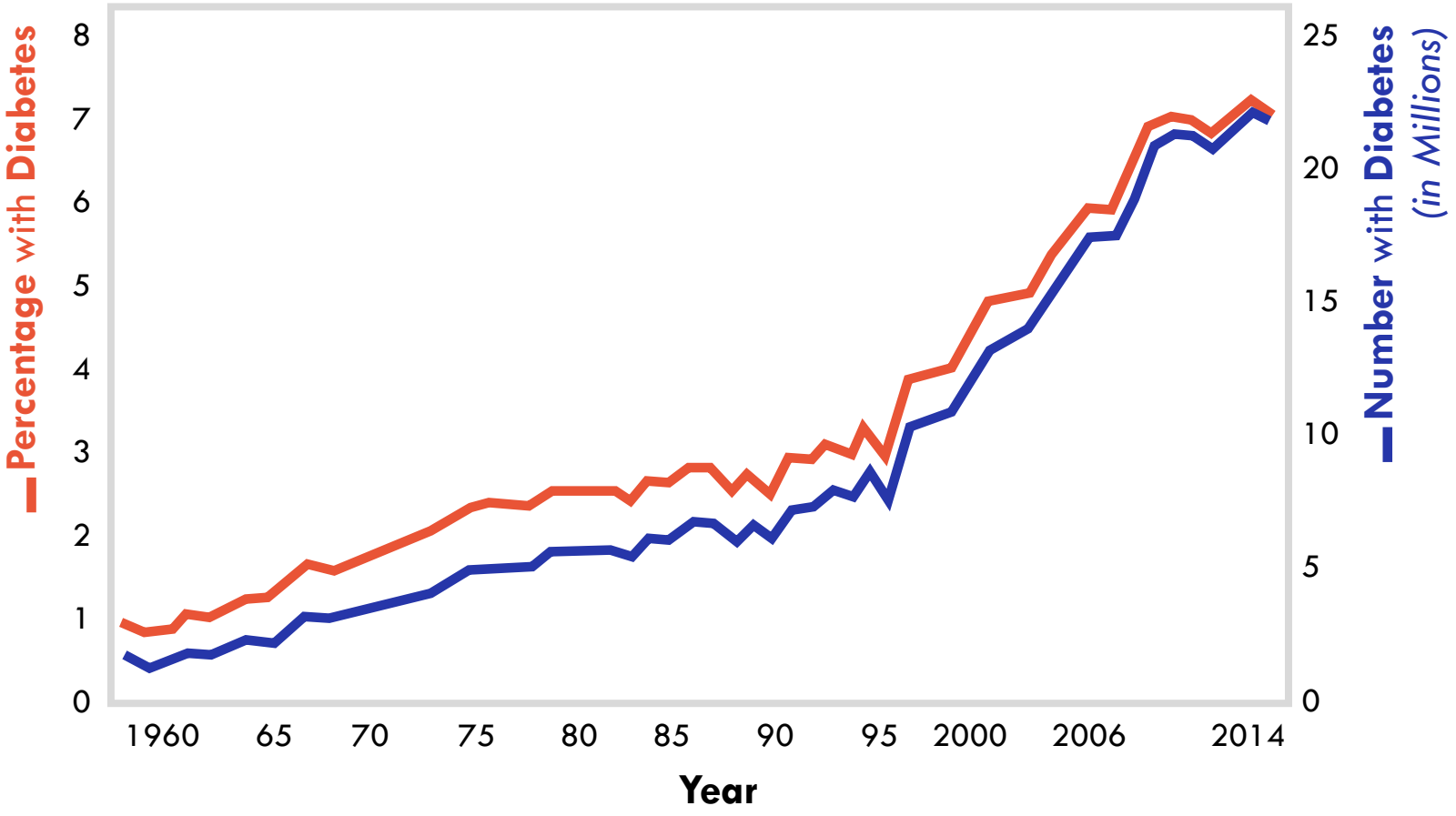
Genetics

Genetic Variant

So,
carbs good,
low carb bad
right?

#context

This is NOT the paleolithic, most of us don't have numbers like HG's



Reference: Centers for Disease Control and Prevention (CDC), "Long-Term Trends in Diabetes," April 2016. Americans diagnosed with diabetes, 1958 through 2014.

There *ARE* **laudable characteristics** of **time restricted feeding, exercise and low carb intake.**


SAY HELLO TO MY LITTLE FRIEND!!!!
Hysteresis.

Interdiscip Top Gerontol. Author manuscript; available in PMC 2009 Oct 1.
Published in final edited form as:
Interdiscip Top Gerontol. 2007; 35: 39–68.
doi: 10.1159/000096555

PMCID: PMC2755292
NIHMSID: NIHMS134099

Glucose Hysteresis as a Mechanism in Dietary Restriction, Aging and Disease

Charles V. Mobbs, Jason Mastaitis, Minhua Zhang, Fumiko Isoda, Hui Cheng, and Kelvin Yen

Author information ▶ Copyright and License information ▶

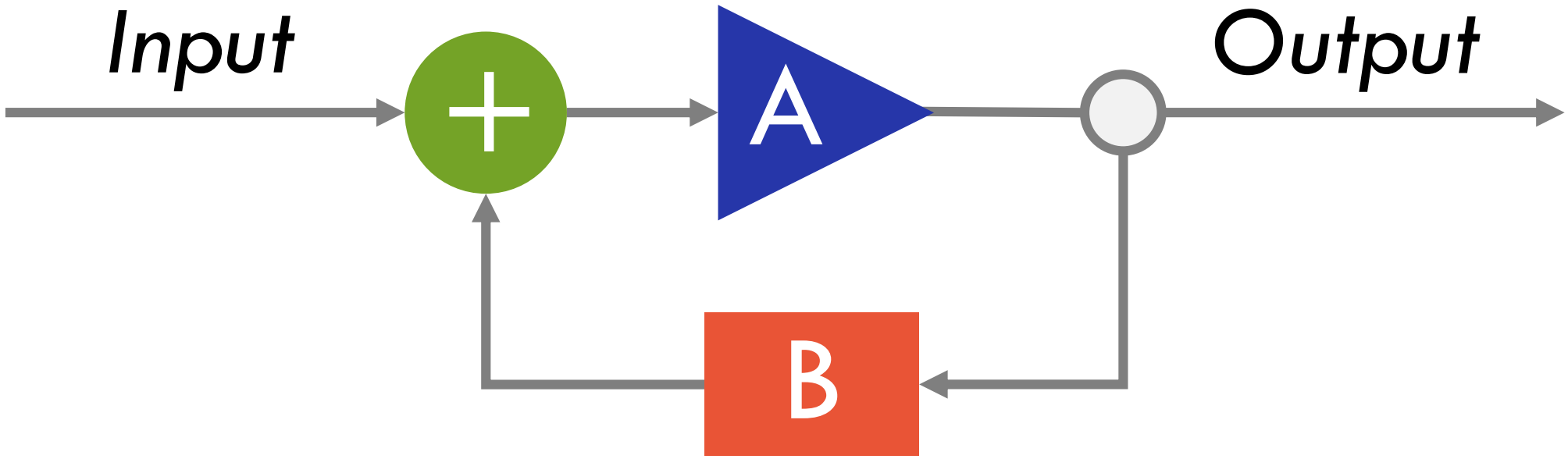
Abstract

Go to:

Elevated blood glucose associated with diabetes produces progressive and apparently irreversible damage to many cell types. Conversely, reduction of glucose extends life span in yeast, and dietary restriction reduces blood glucose. Therefore it has been hypothesized that cumulative toxic effects of glucose drive at least some aspects of the aging process and, conversely, that protective effects of dietary restriction are mediated by a reduction in exposure to glucose. The mechanisms mediating cumulative toxic effects of glucose are suggested by two general principles of metabolic processes, illustrated by the *lac* operon but also observed with glucose-induced gene expression. First, metabolites induce the machinery of their own metabolism. Second, induction of gene expression by metabolites can entail a form of molecular memory called hysteresis. When applied to glucose-regulated gene expression, these two principles suggest a mechanism whereby repetitive exposure to postprandial excursions of glucose leads to an age-related increase in glycolytic capacity (and reduction in β-oxidation of free fatty acids), which in turn leads to an increased generation of oxidative damage and a decreased capacity to respond to oxidative damage, independent of metabolic rate. According to this mechanism, dietary restriction increases life span and reduces pathology by reducing exposure to glucose and therefore delaying the development of glucose-

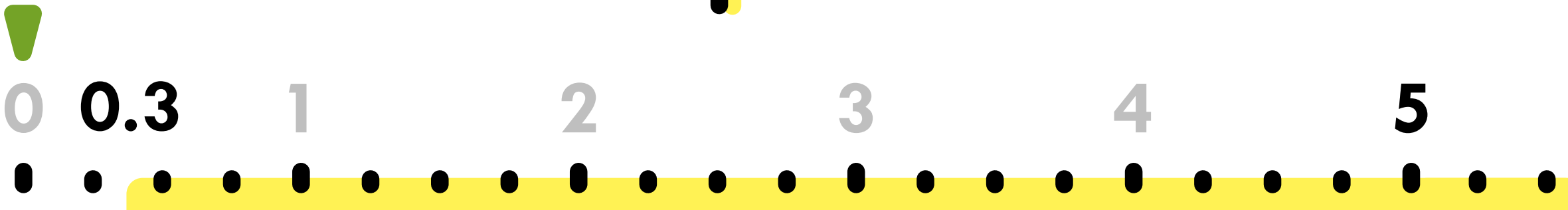
What is Hysteresis?

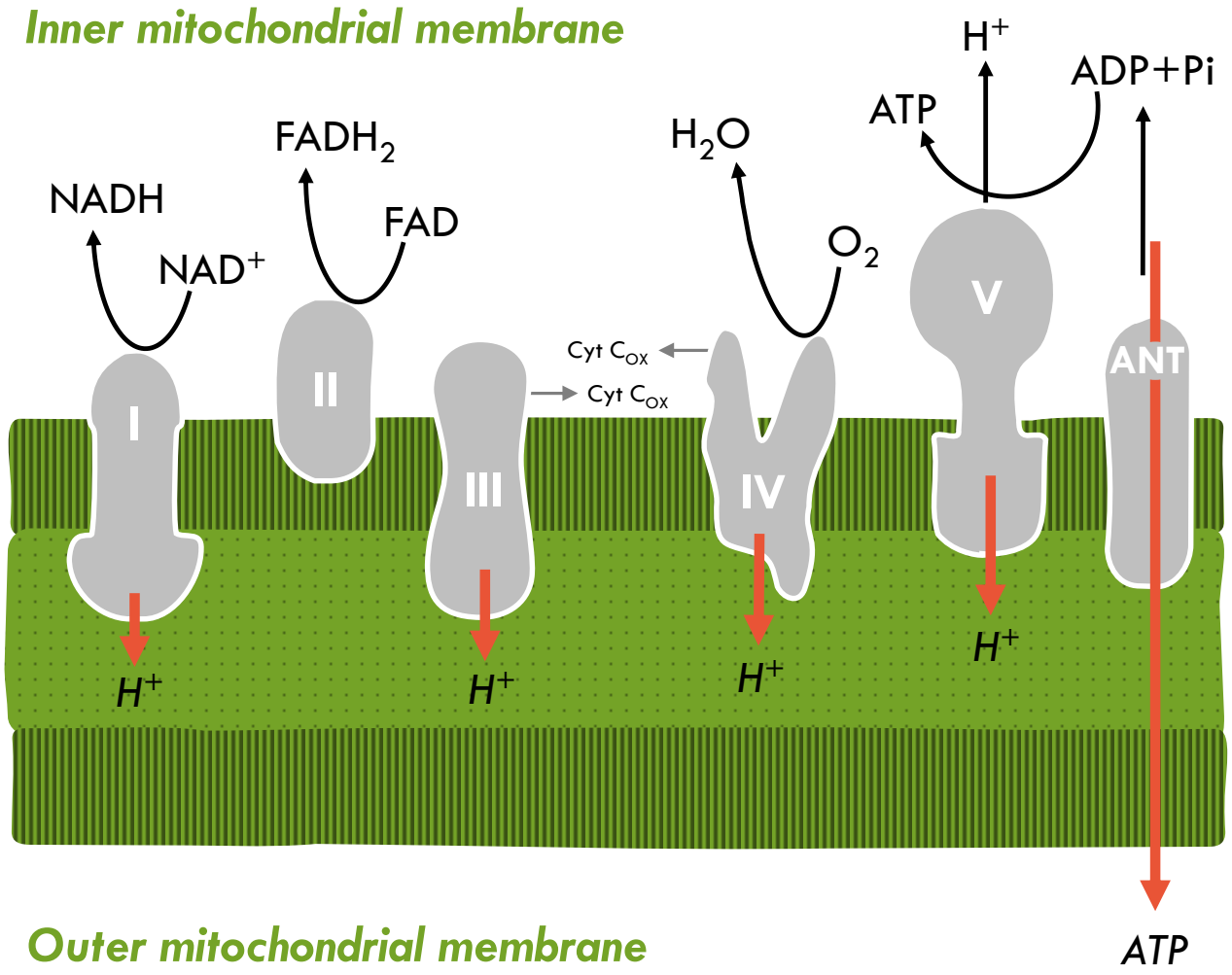
Dependence of the state of a system on its HISTORY
(A type of memory)



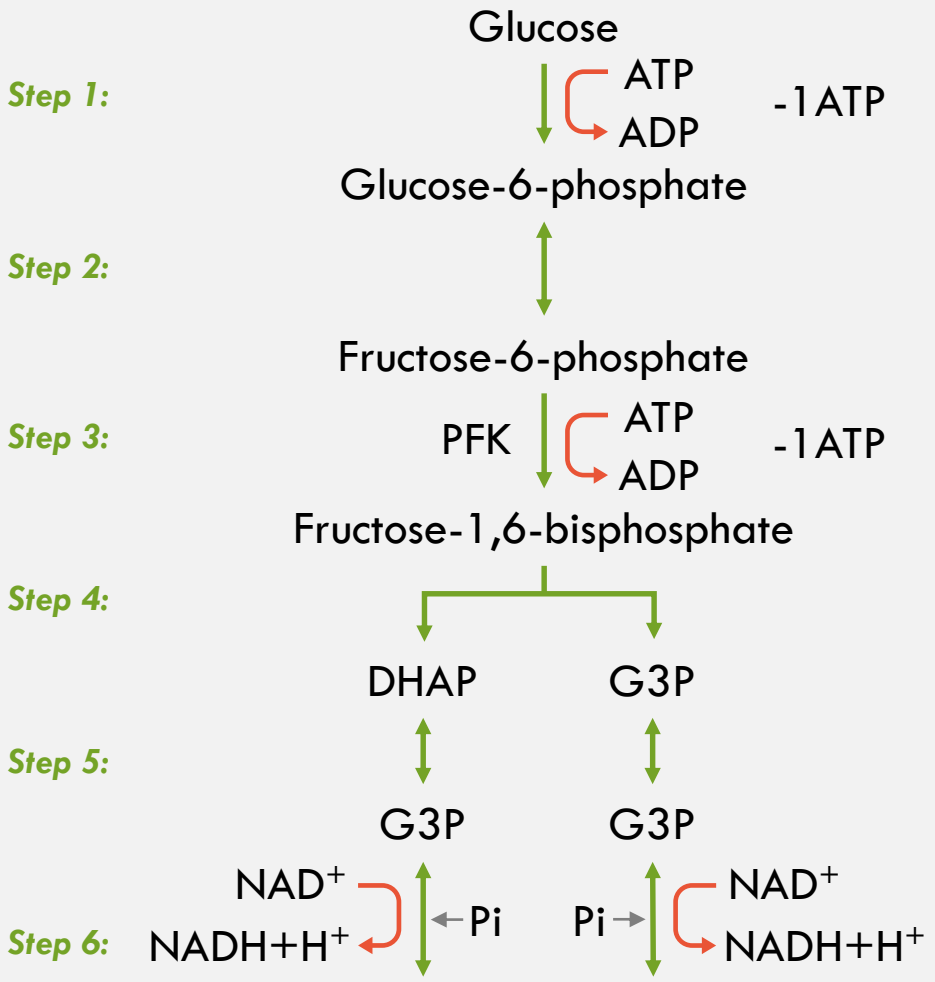
Schmidt Trigger in electronics

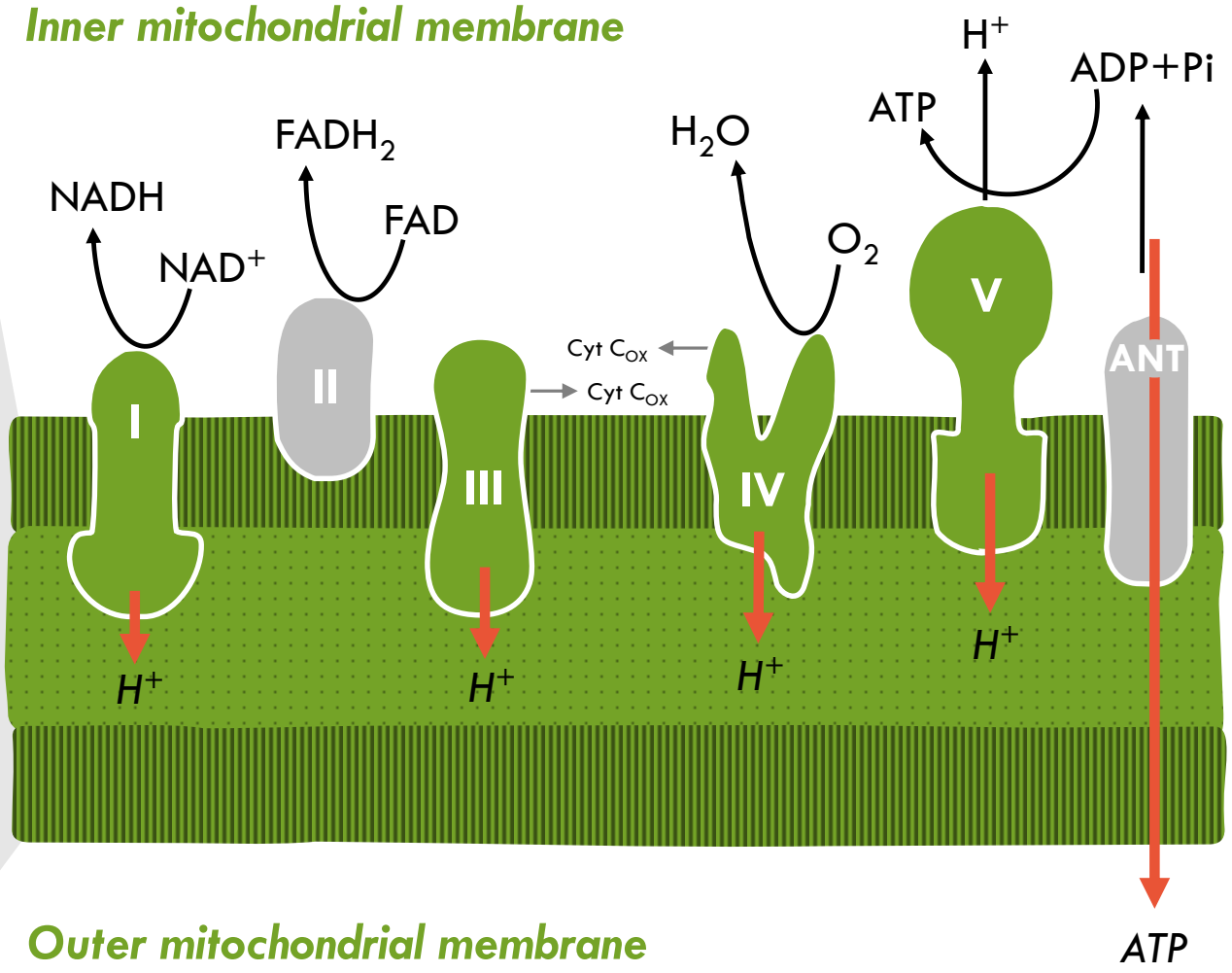
Glucose Hysteresis:
Epigenetics in Action



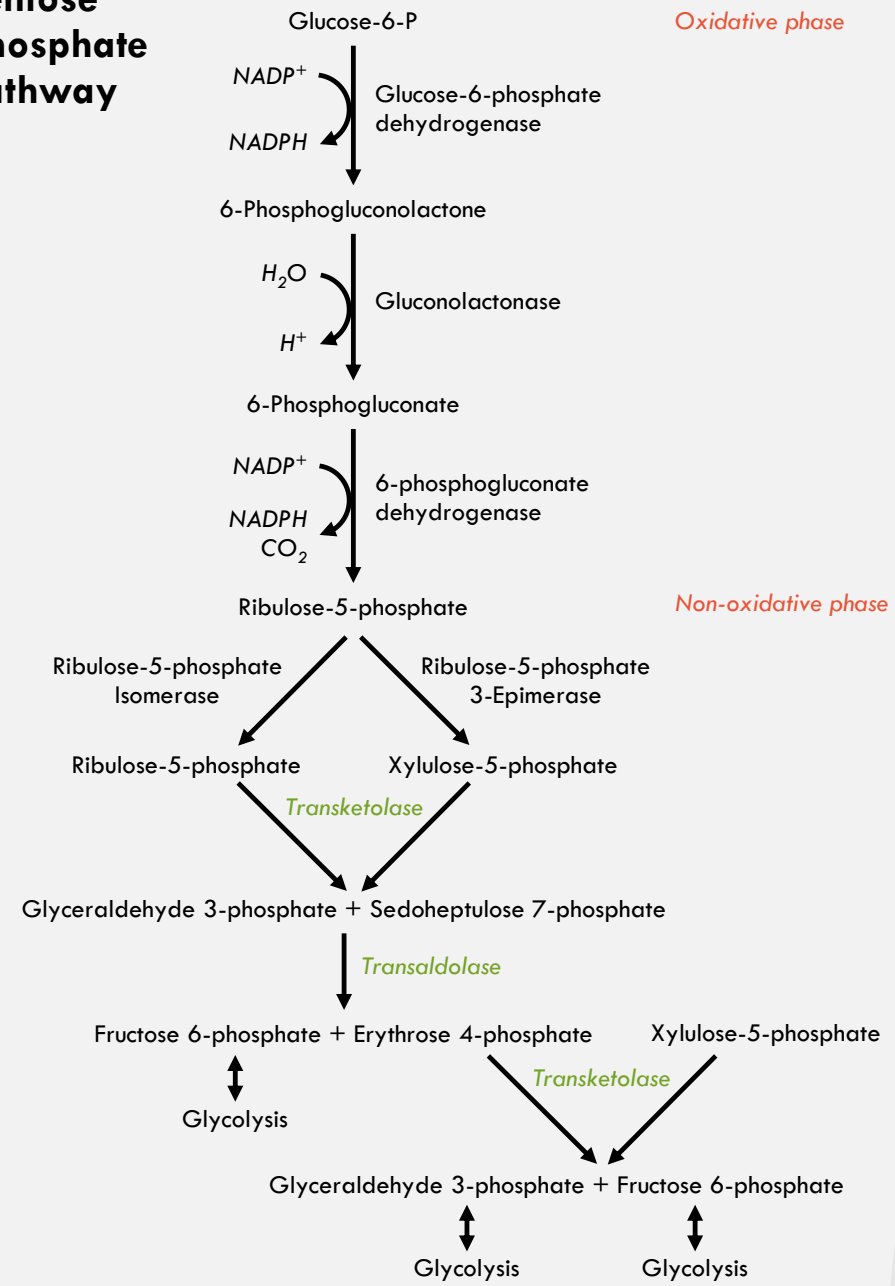

Inner mitochondrial membrane
NADH
NAD+
FADH2
FAD
H2O
O2
H+
ATP
ADP+Pi
I
II
III
IV
V
ANT
Cyt Cox
Cyt Cox
H+
H+
H+
H+
ATP
Outer mitochondrial membrane

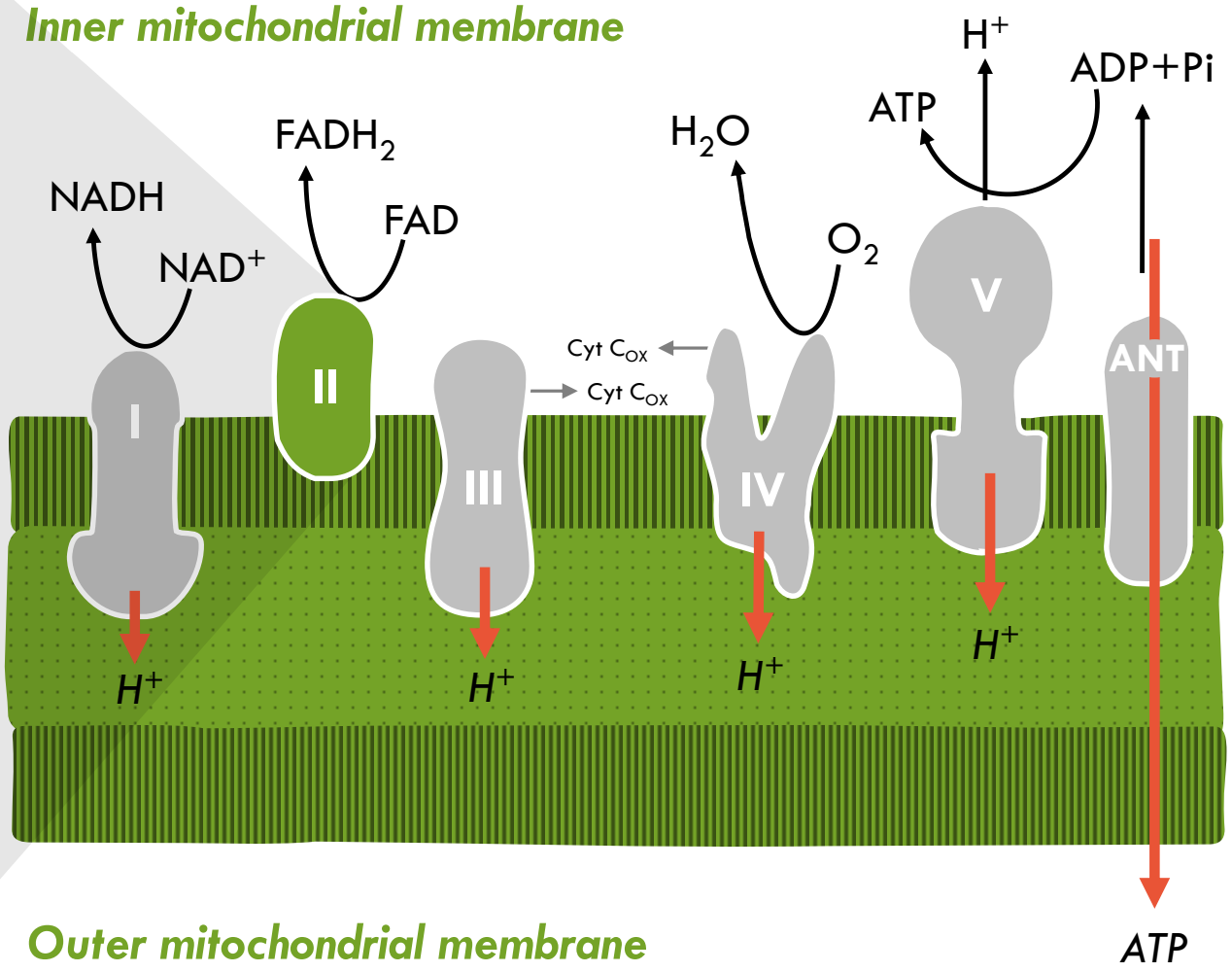
Glycolysis Pathway



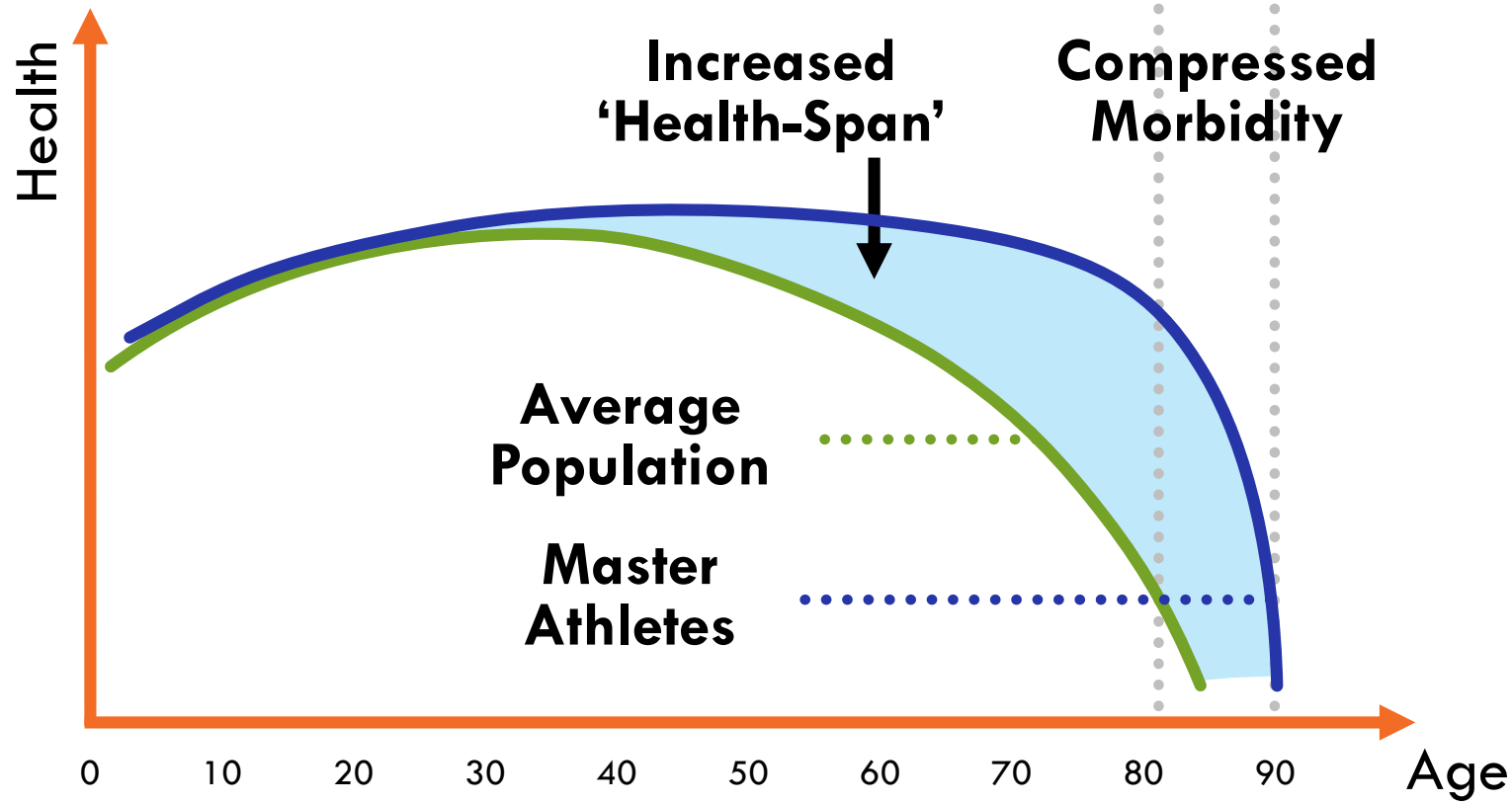


Pentose Phosphate Pathway





Complex 1 *vs.* Complex 2 and **Health Span**



Reference: https://assets.weforum.org/editor/znQ7FmQNtDn_0zlEk0PyVdgMApRGvh_5Pholu6XM4uo.jpg

The Respiratory Quotient (RQ)

The following table shows the **RQ values** for different classes of **respiratory substrate** when they are used for aerobic respiration

RQ value	1.0	0.7	0.9
Respiratory Substrate	**Glucose**	**Fatty acid**	**Protein**

If any degree of anaerobic respiration occurs RQ values significantly above a value of 1.0 are obtained

Kenyan Runners

Pediatr Diabetes. 2015 May;16(3):211-8. doi: 10.1111/pedi.12141. Epub 2014 Apr 23.

Metabolic inflexibility and insulin resistance in obese adolescents with non-alcoholic fatty liver disease.

Lee S[1], Rivera-Vega M, Alsayed HM, Boesch C, Libman I.

Author information

Abstract

BACKGROUND: Non-alcoholic fatty liver disease (NAFLD) is a comorbidity of childhood obesity.

OBJECTIVE: We examined whole-body substrate metabolism and metabolic characteristics in obese adolescents with vs. without NAFLD.

SUBJECTS: Twelve obese (BMI ≥ 95th percentile) adolescents with and without NAFLD [intrahepatic triglyceride (IHTG) ≥5.0% vs. <5.0%] were pair-matched for race, gender, age and % body fat.

METHODS: Insulin sensitivity (IS) was assessed by a 3-h hyperinsulinemic-euglycemic clamp and whole-body substrate oxidation by indirect calorimetry during fasting and insulin-stimulated conditions.

RESULTS: Adolescents with NAFLD had increased ($p < 0.05$) abdominal fat, lipids, and liver enzymes compared with those without NAFLD. Fasting glucose concentration was not different between groups, but fasting insulin concentration was higher ($p < 0.05$) in the NAFLD group compared with those without. Fasting hepatic glucose production and hepatic IS did not differ ($p > 0.1$) between groups. Adolescents with NAFLD had higher ($p < 0.05$) fasting glucose oxidation and a tendency for lower fat oxidation. Adolescents with NAFLD had lower ($p < 0.05$) insulin-stimulated glucose disposal and lower peripheral IS compared with those without NAFLD. Although respiratory quotient (RQ) increased significantly from fasting to insulin-stimulated conditions in both groups (main effect, $p < 0.001$), the increase in RQ was lower in adolescents with NAFLD vs. those without (interaction, $p = 0.037$).

CONCLUSION: NAFLD in obese adolescents is associated with adverse cardiometabolic profile, peripheral insulin resistance and metabolic inflexibility.

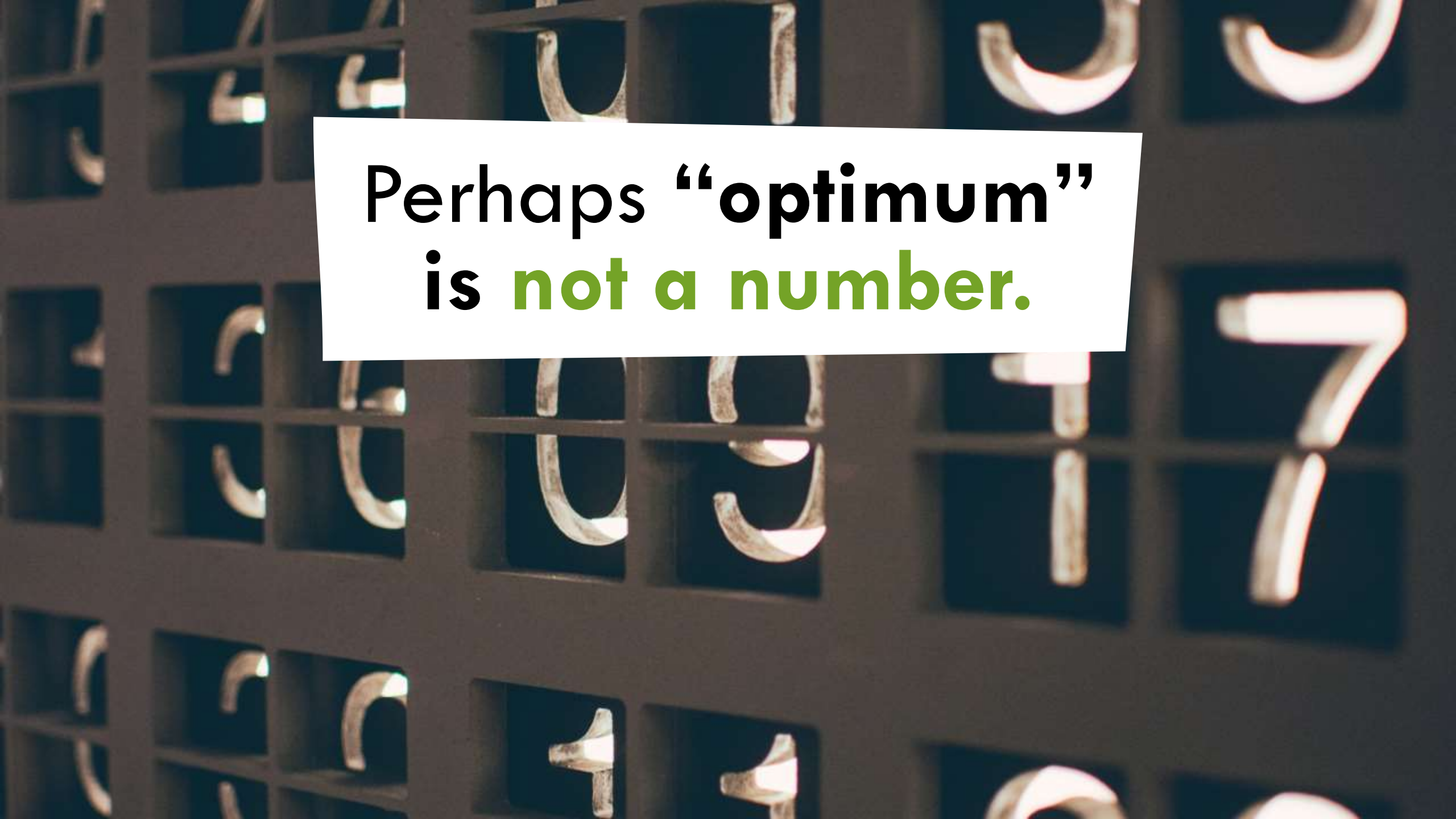
KEYWORDS: childhood obesity; insulin sensitivity; non-alcoholic fatty liver disease; visceral fat

PMID: 24754380 PMCID: PMC4339626 DOI: 10.1111/pedi.12141

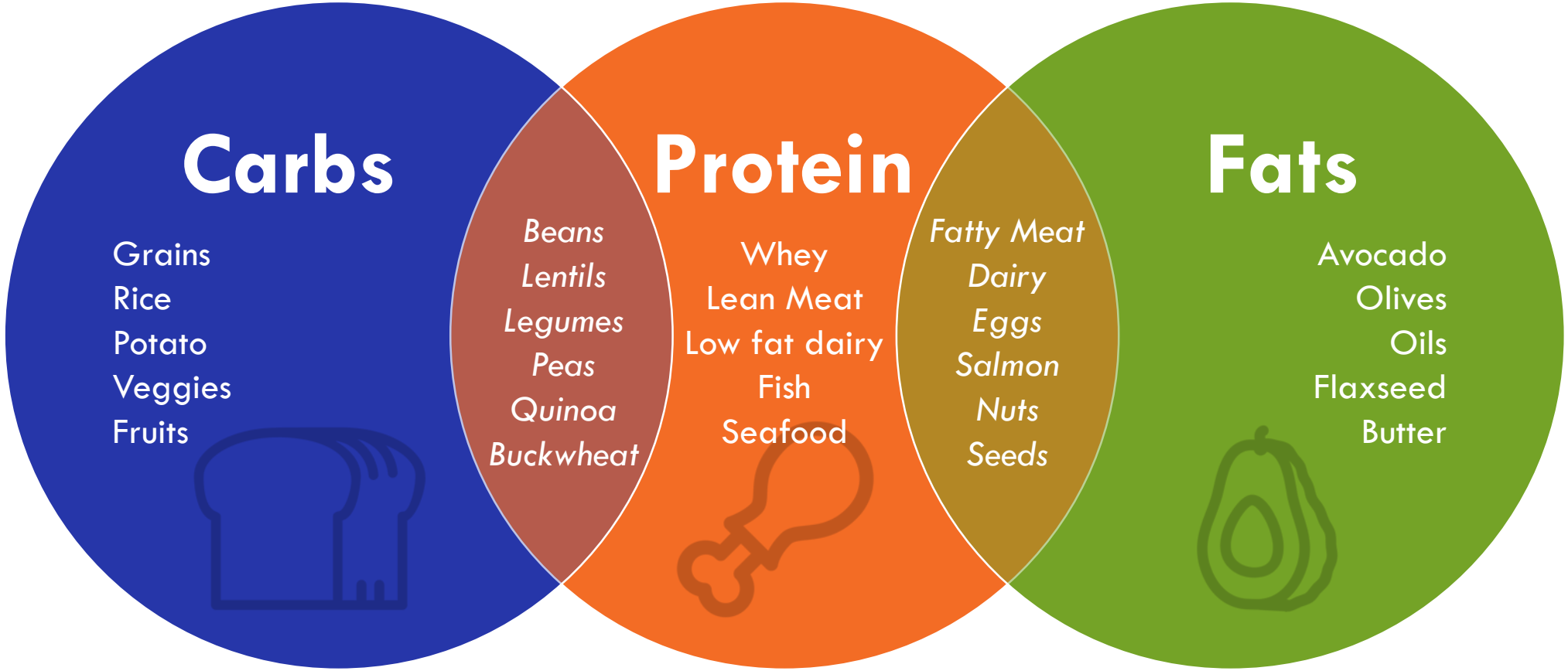
[Indexed for MEDLINE] Free PMC Article

By Contrast

Is there
a formula
for *success*?


Perhaps “optimum”
is not a number.

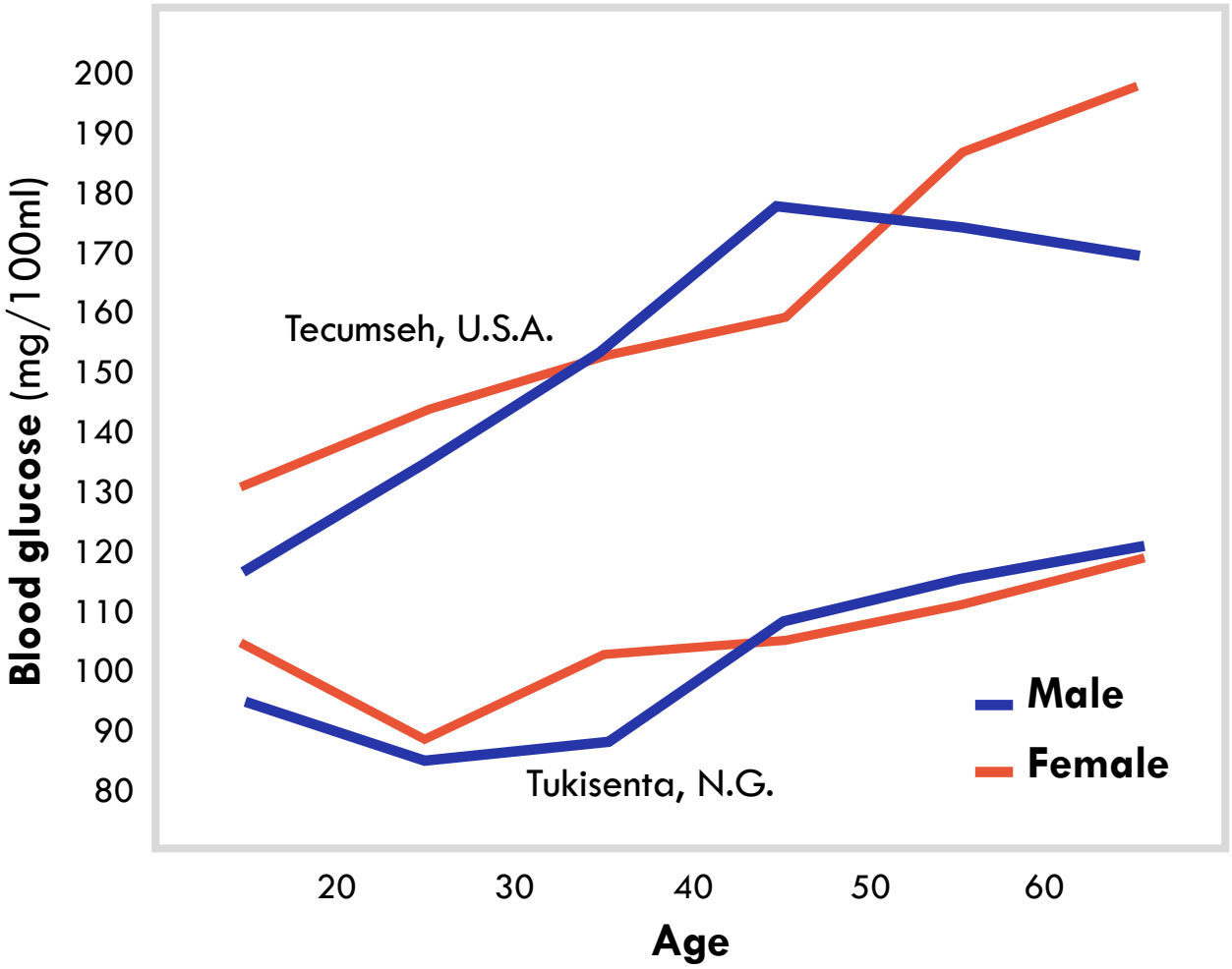
Perhaps **“optimum” is a SEAMLESS transition between a wide variety of fuels.**



Perhaps "optimum" is a relative absence of modern degenerative disease

The Added Problem

How we define *"normal"*



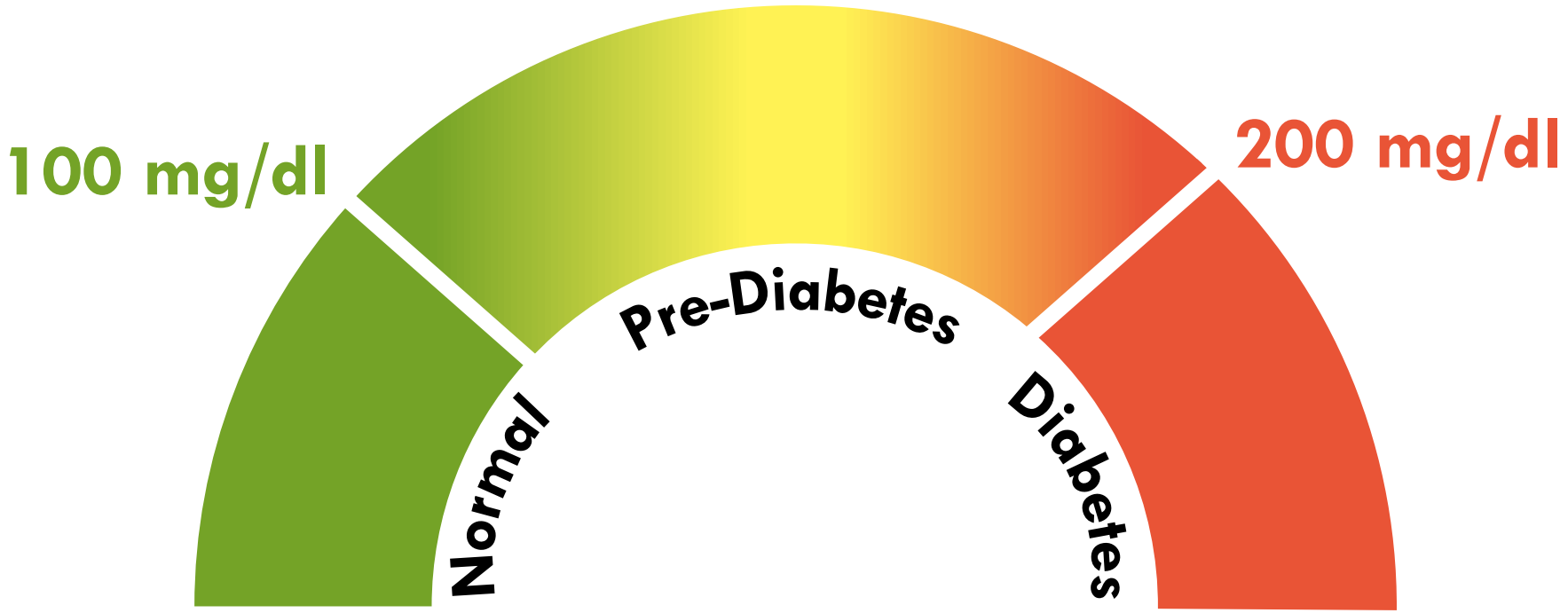
Barely above baseline and **little decline with age!**

Also: **ONE HOUR OGTT!!!**

Reference: http://wholehealthsource.blogspot.com/2010/11/glucose-tolerance-in-non-industrial.html

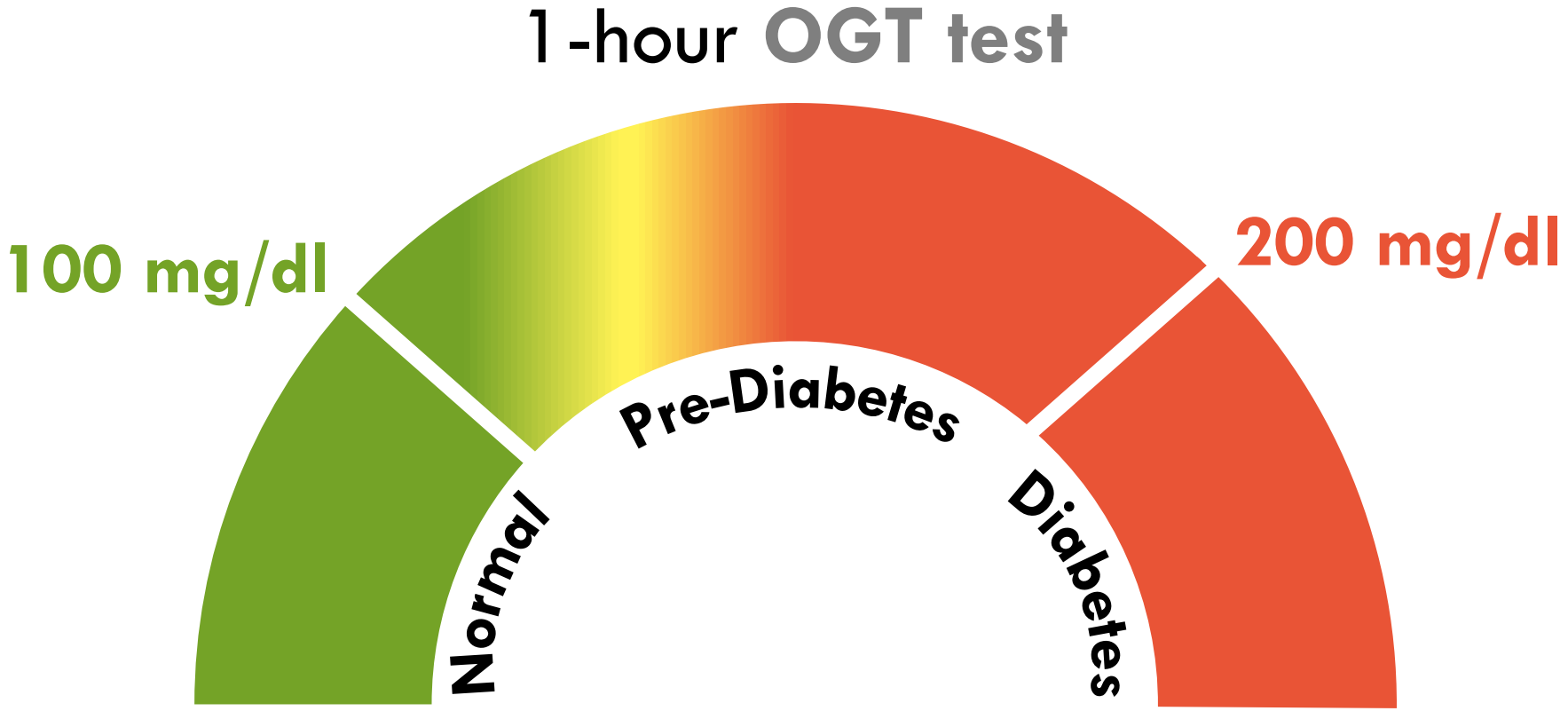
Our *"normal"*

2-hour OGT test



Normal: Not good enough

Pre-Westernized *"normal"*



Establishing Physiologic norms:
The best of Ancestral Health

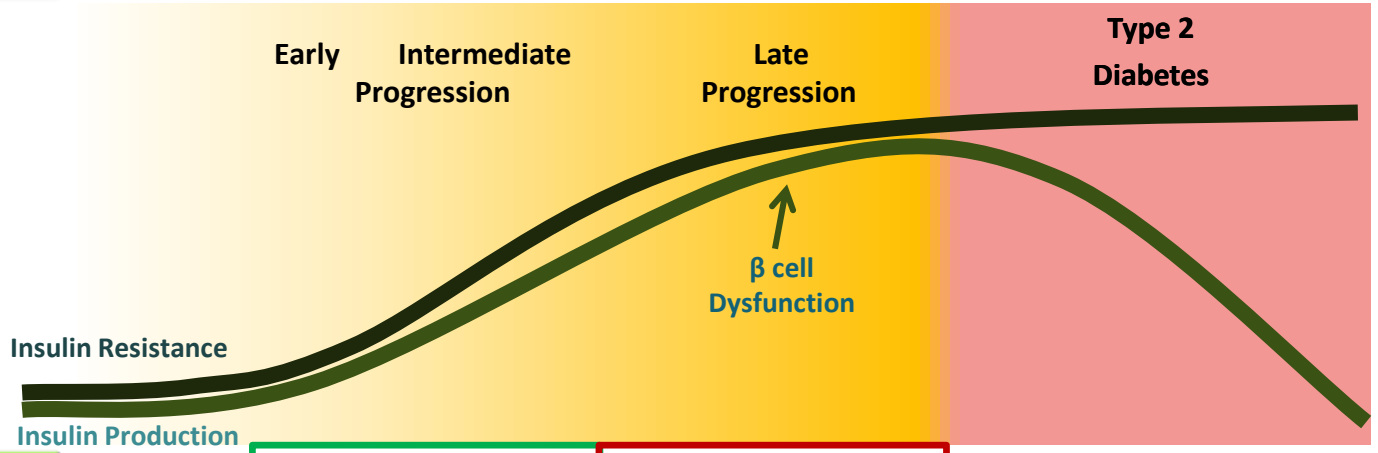
"Prediabetes" is Inadequate Marker of DM Risk

DIAGNOSTIC DILEMMA

Risk of diabetic progression is assessed by glucose measures that are the "response", not the "cause" of worsening glucose metabolism.

< 100 mg/dL		100-125 mg/dL								≥ 125 mg/dL	
"Normal" Glucose		Pre-Diabetes								Diabetes	
80	85	90	95	100	105	110	115	120	125	>125	Plasma Glucose, mg/dL
		5.5		5.7		6.0			6.4	>6.4	HbA1c %

Continuum of Type 2 Diabetes Progression



UNMET DIAGNOSTIC NEED

"Diagnostic tests should be developed to better distinguish patients who will progress to diabetes from those who will not." [1]

Many with glucose 100-110 mg/dL do not progress to T2DM/

Others with glucose 90-99 mg/dL develop diabetes in <5 years

Those with late stage progression (e.g. FPG > 110) frequently have vascular complications and increased pancreatic dysfunction with high risk of progression to Type 2 diabetes

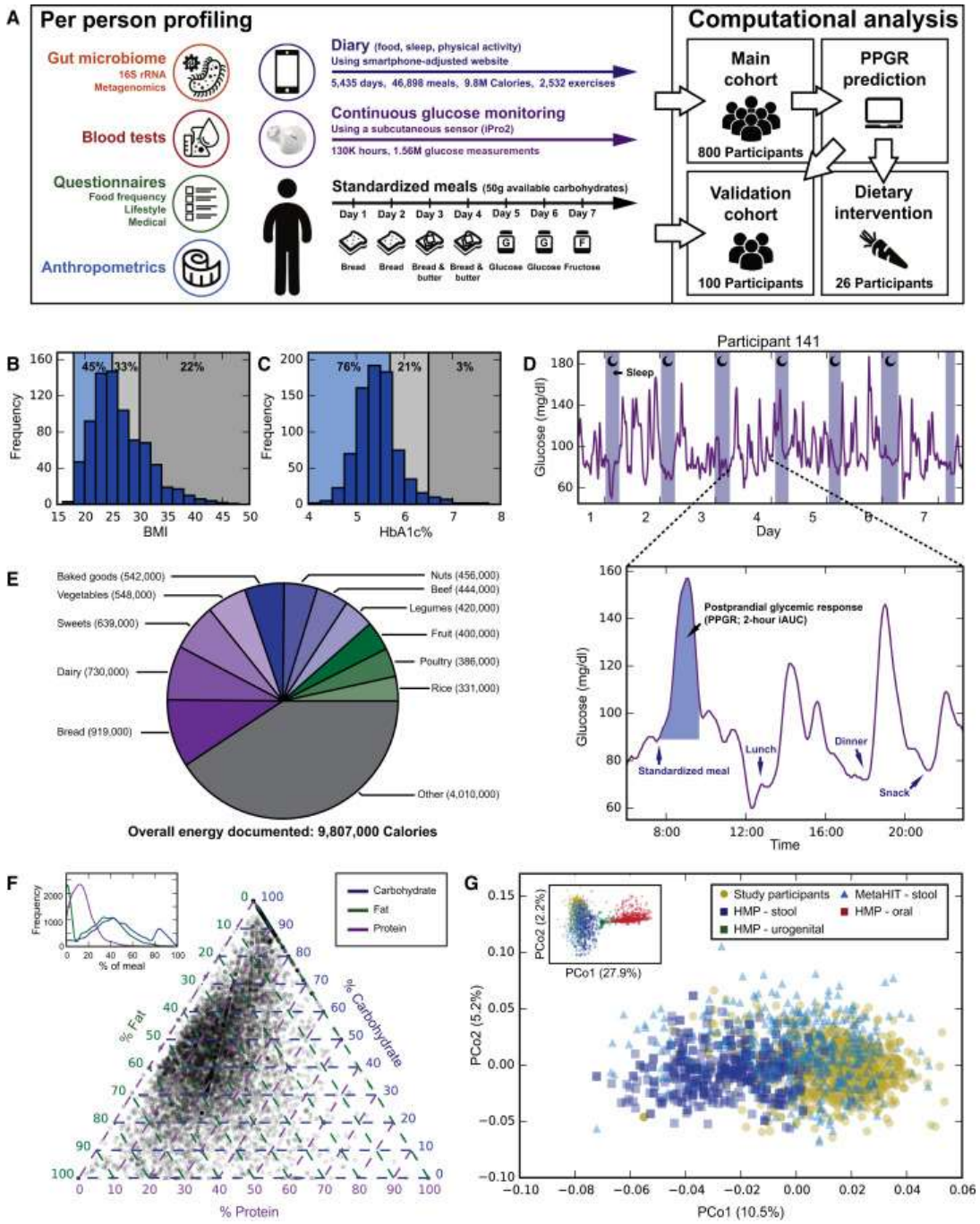
1. AACE Prediabetes Consensus Statement, Endocr Pract. 2008;14(No. 7) 941

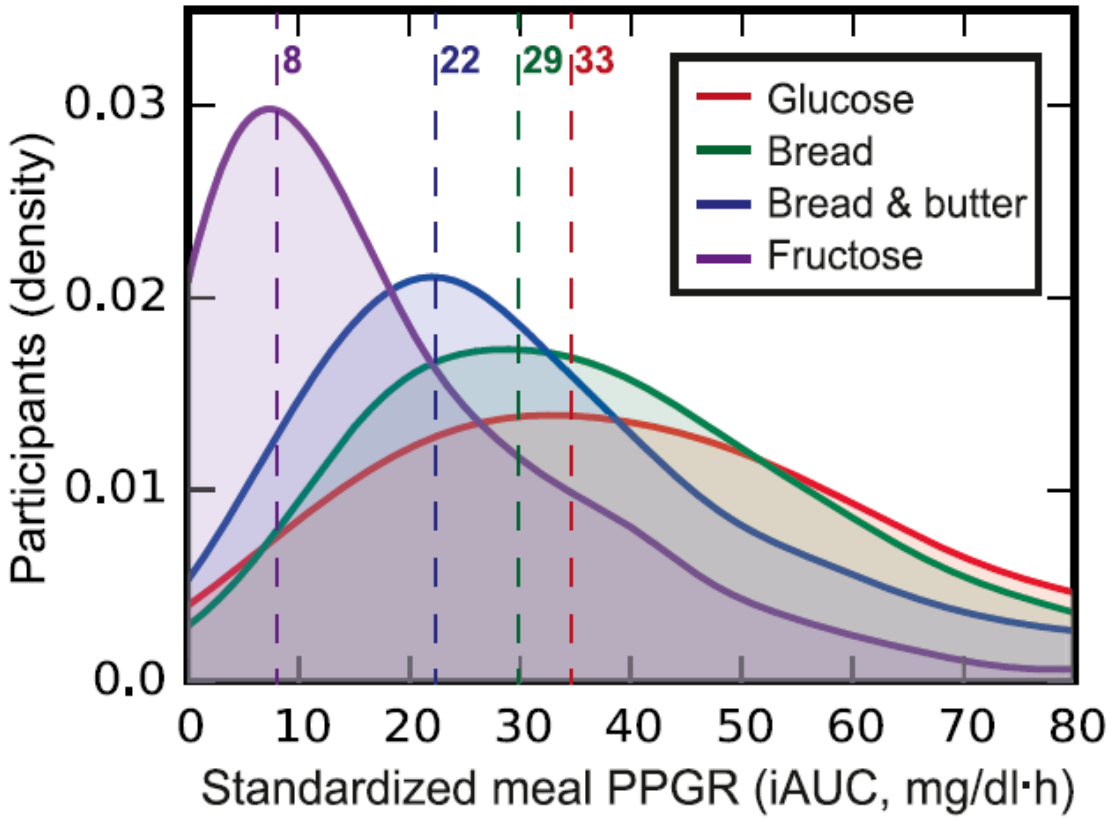
Insulin load/glycemic response important due to individual variations

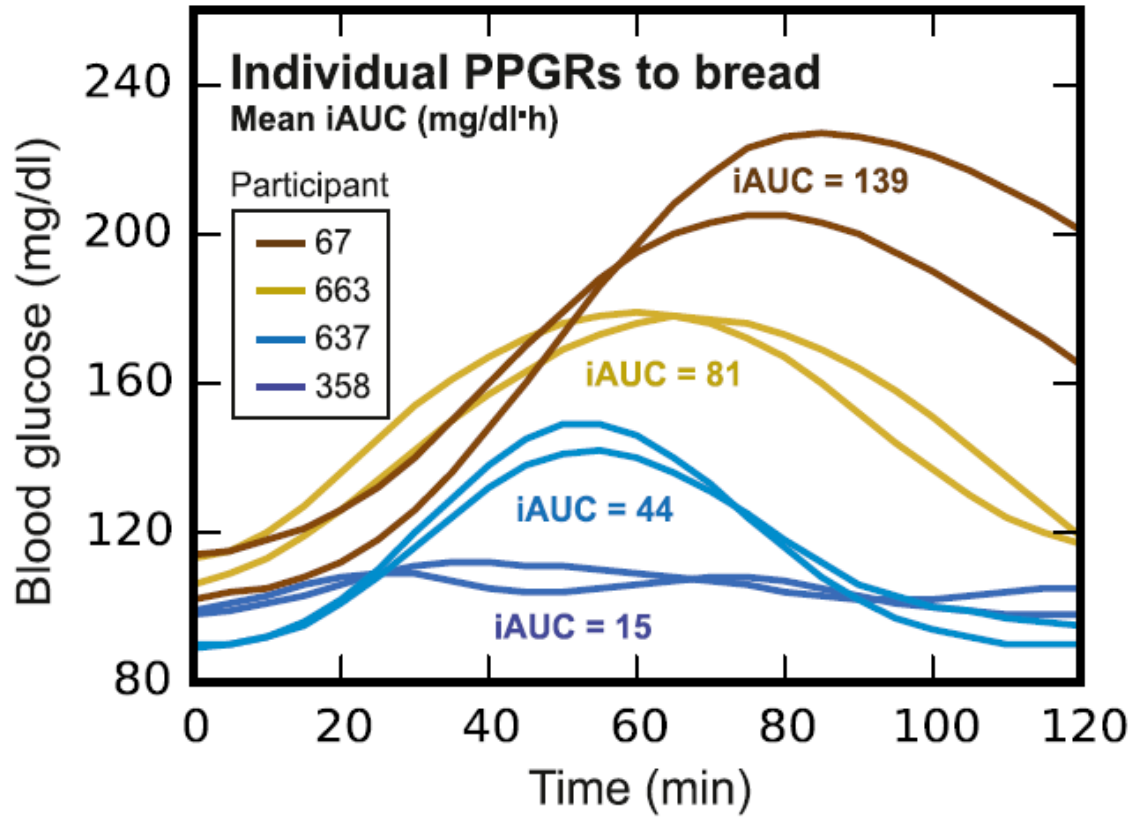
Weitzman inst.

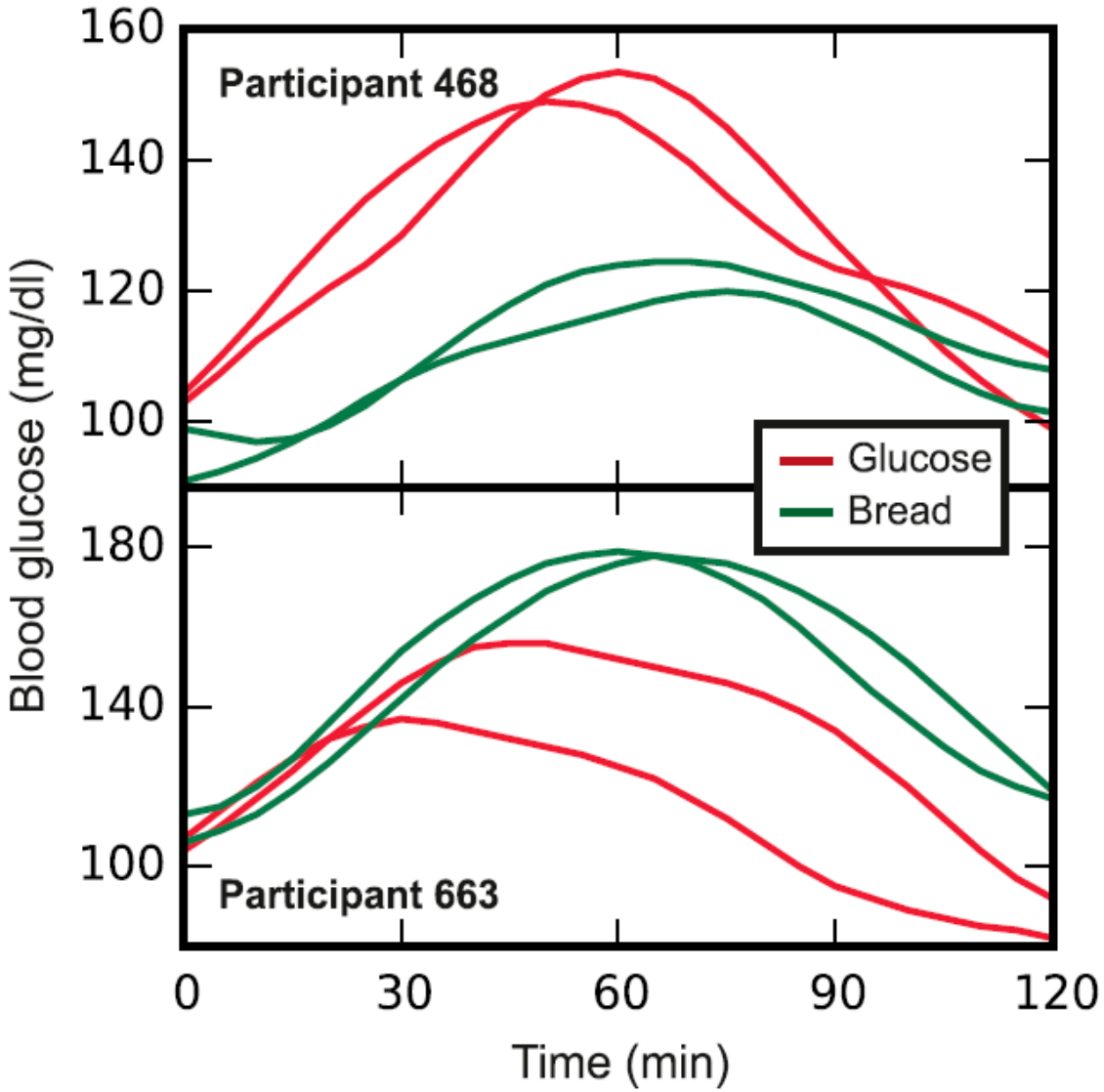
Brain hates glucose deltas *(Remember Hysteresis!!)*

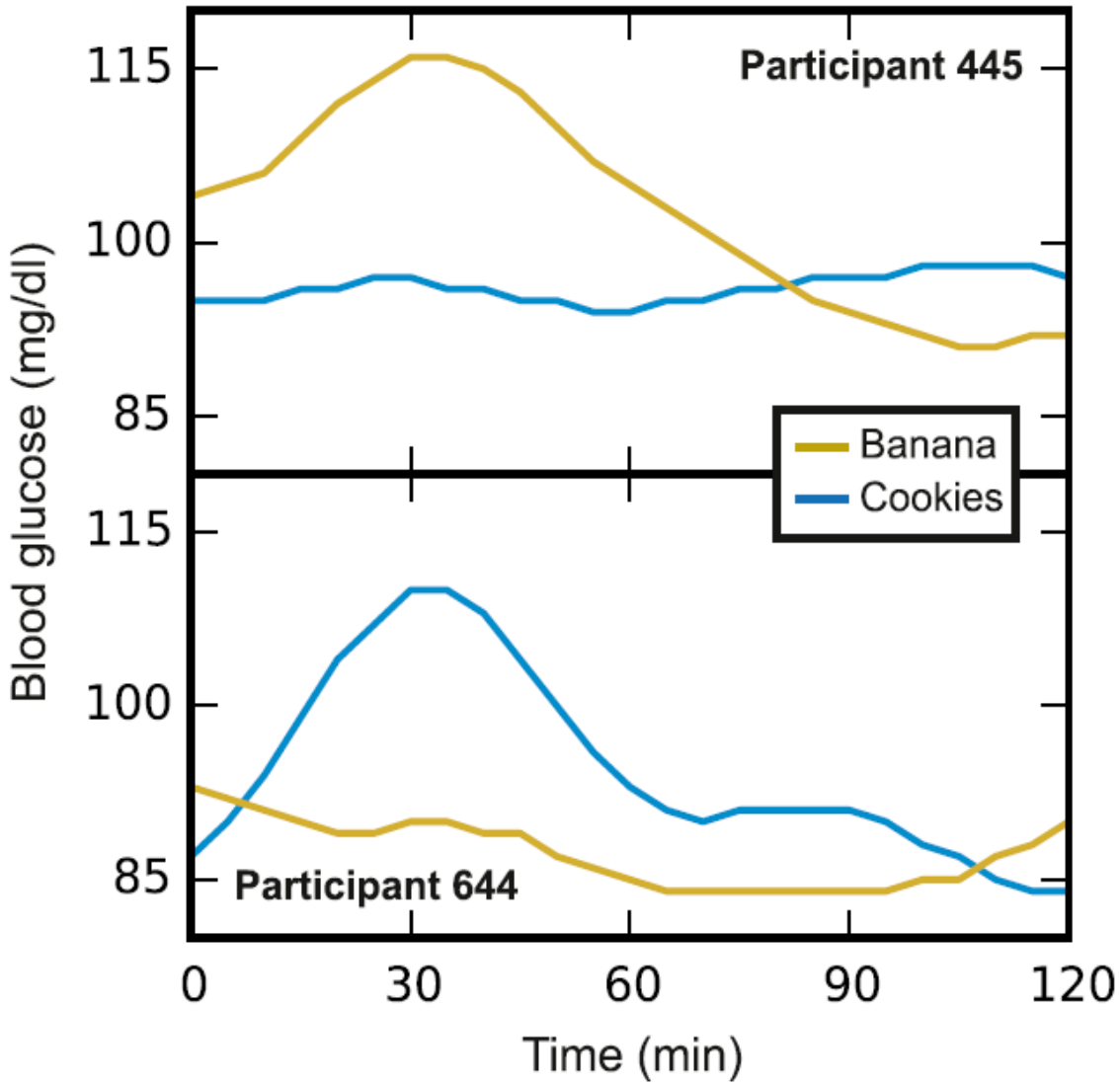
Overeating




8
22
29
33
Glucose
Bread
Bread & butter
Fructose
Participants (density)
0.03
0.02
0.01
0.0
0
10
20
30
40
50
60
70
80
Standardized meal PPGR (iAUC, mg/dl·h)


Individual PPGRs to bread
Mean iAUC (mg/dl·h)
Participant
67
663
637
358
iAUC = 139
iAUC = 81
iAUC = 44
iAUC = 15
Blood glucose (mg/dl)
240
200
160
120
80
0
20
40
60
80
100
120
Time (min)


Participant 468
Participant 663
Glucose
Bread
Blood glucose (mg/dl)
160
140
120
100
180
140
100
0
30
60
90
120
Time (min)


Participant 445
Participant 644
Banana
Cookies
Blood glucose (mg/dl)
115
100
85
115
100
85
0
30
60
90
120
Time (min)

Real World Example:

7 Day Carb Test

My
Blood Glucose

Quantified

US

Nicki's
Blood Glucose

Real World Example #2:

Is the LC flu a symptom of problems or normal?

Transition to Ketosis

OK,

why *can't* we transition seamlessly?

Sci Transl Med. Author manuscript; available in PMC 2013 Sep 3.
Published in final edited form as:
Sci Transl Med. 2013 Jul 3; 5(192): 192ra85.
doi: 10.1126/scitranslmed.3006055

PMCID: PMC3760005
NIHMSID: NIHMS509241

Bactericidal Antibiotics Induce Mitochondrial Dysfunction and Oxidative Damage in Mammalian Cells

Sameer Kalghatgi,[#1] Catherine S. Spina,[#1,2,3] James C. Costello,[1] Marc Liesa,[3] J Ruben Morones-Ramirez,[1] Shimyn Slomovic,[1] Anthony Molina,[3,4] Orian S. Shirihai,[3] and James J. Collins[1,2,3,*]

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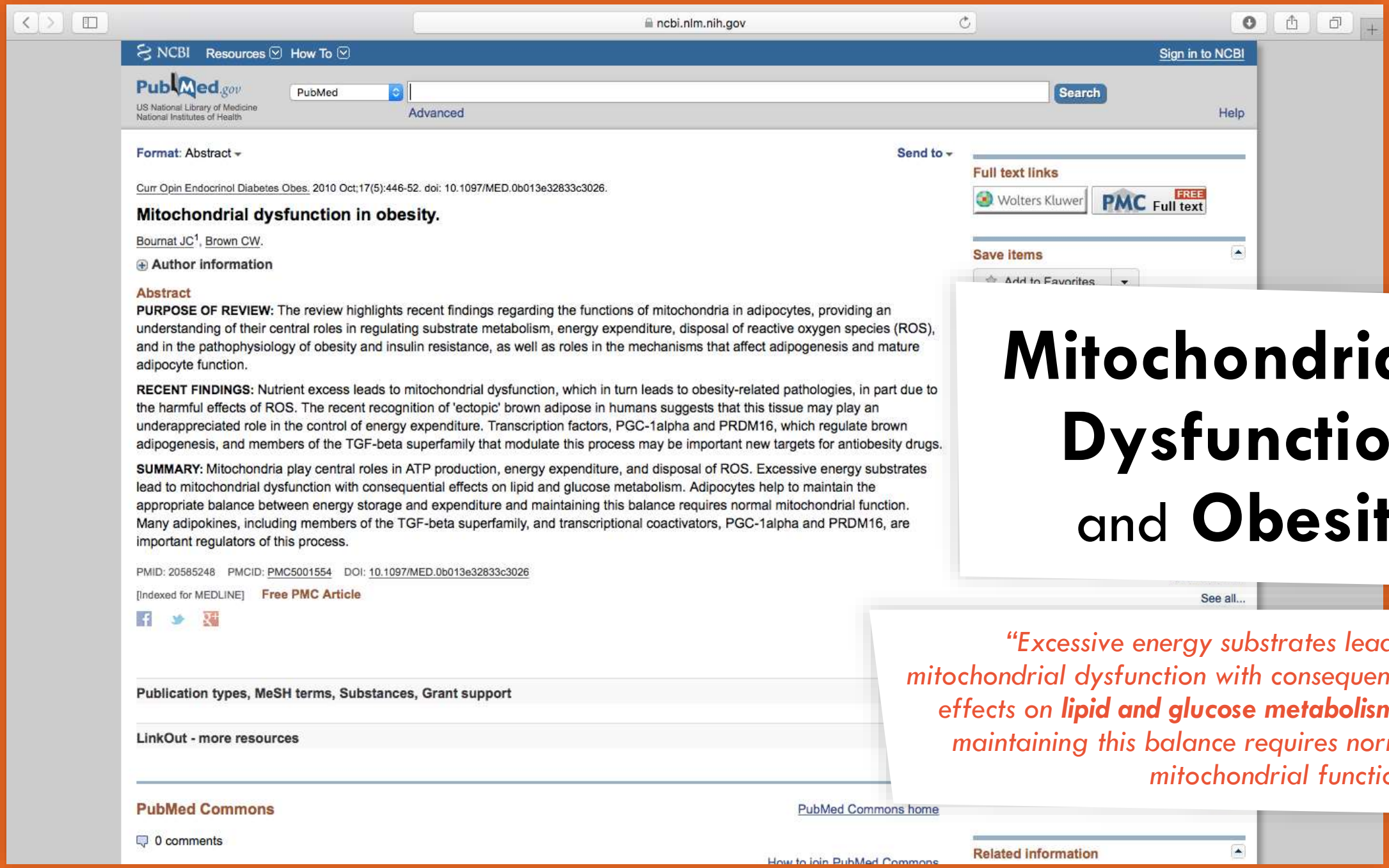
Abstract

Go to:

Prolonged antibiotic treatment can lead to detrimental side effects in patients, including ototoxicity, nephrotoxicity, and tendinopathy, yet the mechanisms underlying the effects of antibiotics in mammalian systems remain unclear. It has been suggested that bactericidal antibiotics induce the formation of toxic reactive oxygen species (ROS) in bacteria. We show that clinically relevant doses of bactericidal antibiotics—quinolones, aminoglycosides, and β-lactams—cause mitochondrial dysfunction and ROS overproduction in mammalian cells. We demonstrate that these bactericidal antibiotic–induced effects lead to oxidative damage to DNA, proteins, and membrane lipids. Mice treated with bactericidal antibiotics exhibited elevated oxidative stress markers in the blood, oxidative tissue damage, and up-regulated expression of key genes involved in antioxidant defense mechanisms, which points to the potential physiological relevance of these antibiotic effects. The deleterious effects of bactericidal antibiotics were alleviated in cell culture and in mice by the administration of the antioxidant N-acetyl-L-cysteine or prevented by preferential use of bacteriostatic antibiotics. This work highlights the role of antibiotics in the production of oxidative tissue damage in mammalian cells and presents strategies to mitigate or prevent the resulting damage, with the goal of improving the safety of antibiotic treatment in people.

Dysfunctional Mitochondria and Antibiotics



Mitochondrial Dysfunction and Obesity

*"Excessive energy substrates lead to mitochondrial dysfunction with consequential effects on **lipid and glucose metabolism…** maintaining this balance requires normal mitochondrial function."*

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Free Radic Biol Med. 2010 Aug 1;49(3):401-7. doi: 10.1016/j.freeradbiomed.2010.04.033. Epub 2010 May 5.

Mitochondrial dysfunction may explain the cardiomyopathy of chronic iron overload.

Gao X[1], Qian M, Campian JL, Marshall J, Zhou Z, Roberts AM, Kang YJ, Prabhu SD, Sun XF, Eaton JW.

Author information

Abstract

In patients with hemochromatosis, cardiac dysfunction may appear years after they have reached a state of iron overload. We hypothesized that cumulative iron-catalyzed oxidant damage to mitochondrial DNA (mtDNA) might explain the cardiomyopathy of chronic iron overload. Mice were given repetitive injections of iron dextran for a total of 4weeks after which the iron-loaded mice had elevated cardiac iron, modest cardiac hypertrophy, and cardiac dysfunction. qPCR amplification of near-full-length (approximately 16kb) mtDNA revealed >50% loss of full-length product, whereas amounts of a qPCR product of a nuclear gene (13kb region of beta globin) were unaffected. Quantitative rtPCR analyses revealed 60-70% loss of mRNA for proteins encoded by mtDNA with no change in mRNA abundance for nuclear-encoded respiratory subunits. These changes coincided with proportionate reductions in complex I and IV activities and decreased respiration of isolated cardiac mitochondria. We conclude that chronic iron overload leads to cumulative iron-mediated damage to mtDNA and impaired synthesis of mitochondrial respiratory chain subunits. The resulting respiratory dysfunction may explain the slow development of cardiomyopathy in chronic iron overload and similar accumulation of damage to mtDNA may also explain the mitochondrial dysfunction observed in slowly progressing diseases such as neurodegenerative disorders.

PMID: 20450972 PMCID: PMC2900522 DOI: 10.1016/j.freeradbiomed.2010.04.033

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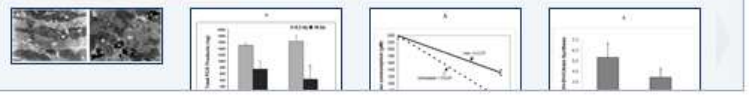

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Increased mitochondrial DNA deletions and copy number in transfusion-depende [JCI Insight. 2016]

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Sleep Disorders Associated with Primary Mitochondrial Diseases

Ryan J. Ramezani, B.S.[1]; Peter W. Stacpoole, Ph.D., M.D.[1,2]

More..

ABSTRACT

Study Objectives

Primary mitochondrial diseases are caused by heritable or spontaneous mutations in nuclear DNA or mitochondrial DNA. Such pathological mutations are relatively common in humans and may lead to neurological and neuromuscular complication that could compromise normal sleep behavior. To gain insight into the potential impact of primary mitochondrial disease and sleep pathology, we reviewed the relevant English language literature in which abnormal sleep was reported in association with a mitochondrial disease.

Design

We examined publications reported in Web of Science and PubMed from February 1976 through January 2014, and identified 54 patients with a proven or suspected primary mitochondrial disorder who were evaluated for sleep disturbances.

Measurements and Results

Both nuclear DNA and mitochondrial DNA mutations were associated with abnormal sleep patterns. Most subjects who underwent polysomnography had central sleep apnea, and only 5 patients had obstructive sleep apnea. Twenty-four patients showed decreased ventilatory drive in response to hypoxia and/or hypercapnia that was not considered due to weakness of the intrinsic

Microbial Ecology in Health and Disease

Taylor & Francis Taylor & Francis Group

Microb Ecol Health Dis. 2015; 26: 10.3402/mehd.v26.27458. PMCID: PMC4425813

Published online 2015 May 7. doi: 10.3402/mehd.v26.27458

Gastrointestinal dysfunction in autism spectrum disorder: the role of the mitochondria and the enteric microbiome

Richard E. Frye,[1,2,*] Shannon Rose,[1,2] John Slattery,[1,2] and Derrick F. MacFabe[3]

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Abstract

Go to:

Autism spectrum disorder (ASD) affects a significant number of individuals worldwide with the prevalence continuing to grow. It is becoming clear that a large subgroup of individuals with ASD demonstrate abnormalities in mitochondrial function as well as gastrointestinal (GI) symptoms. Interestingly, GI disturbances are common in individuals with mitochondrial disorders and have been reported to be highly prevalent in individuals with co-occurring ASD and mitochondrial disease. The majority of individuals with ASD and mitochondrial disorders do not manifest a primary genetic mutation, raising the possibility that their mitochondrial disorder is acquired or, at least, results from a combination of genetic susceptibility interacting with a wide range of environmental triggers. Mitochondria are very sensitive to both endogenous and exogenous environmental stressors such as toxicants, iatrogenic medications, immune activation, and metabolic disturbances. Many of these same environmental stressors have been associated with ASD, suggesting that the mitochondria could be the biological link between environmental stressors and neurometabolic abnormalities associated with ASD. This paper reviews the possible links between GI abnormalities, mitochondria, and ASD. First, we review the link between GI symptoms and abnormalities in mitochondrial function. Second, we review the evidence supporting the notion that environmental stressors linked to ASD can also adversely affect both mitochondria and GI function. Third, we review the evidence that enteric bacteria that are overrepresented in children with ASD, particularly *Clostridia* spp., produce short-chain fatty acid metabolites that are potentially toxic to the

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Mitochondrial Dysfunction and Microbiome

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The many roads to mitochondrial dysfunction in neuroimmune and neuropsychiatric disorders

Gerwyn Morris and Michael Berk

BMC Medicine 2015 **13**:68
https://doi.org/10.1186/s12916-015-0310-y
Received: 23 December 2014 | Accepted: 4 March 2015 | Published: 1 April 2015

Open Peer Review reports

Abstract

Background

Mitochondrial dysfunction and defects in oxidative metabolism are a characteristic feature of many chronic illnesses not currently classified as mitochondrial diseases. Examples of such illnesses include bipolar disorder, multiple sclerosis, Parkinson's disease, schizophrenia, depression, autism, and chronic fatigue syndrome.

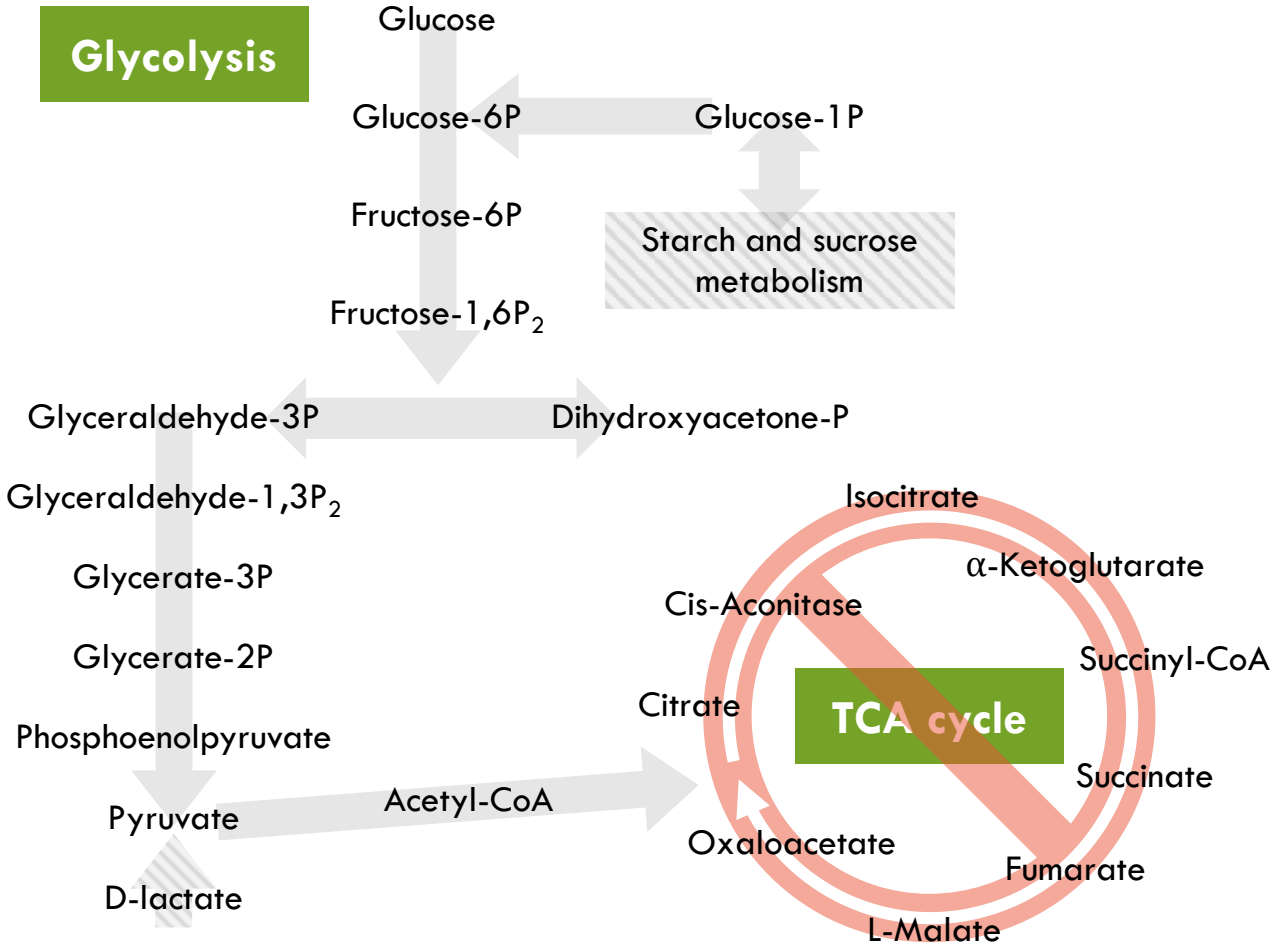
Discussion

While the majority of patients with multiple sclerosis appear to have widespread

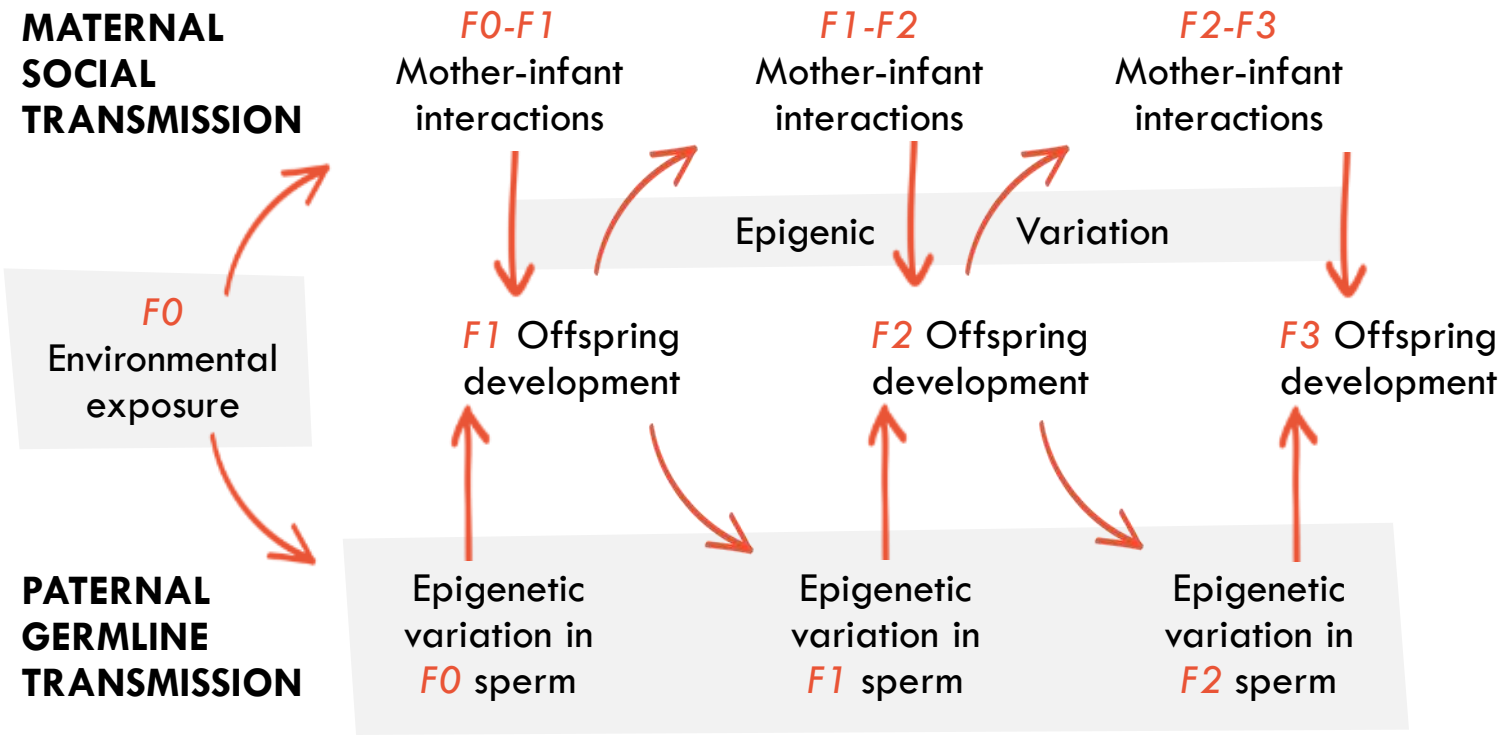
Many Roads

Your Metabolism = your Mitochondria

If your mitochondria are broken, only thing left is Glycolysis. Complexes 1, 3, 4, etc.



What about multi-generational epigenetic changes??




ENOUGH TALK!

OK Propeller Head,

that sounds great

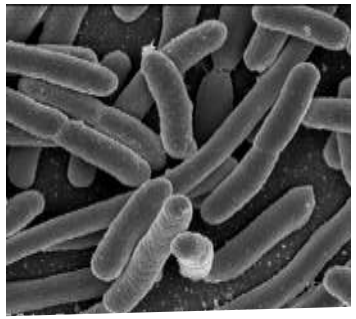
but *what* **to DO**

Reclaiming and **maintaining** **our metabolic flexibility** *(ie. mitochondrial health)*

Circadian rhythm

Sleep

Light exposure



Gut health

Meaningful relationships

Lift weights

Go fast

Go slow

Novel experiences

Regarding Food

Test and track **BG response** *(WTE-7 Day Carb Test)*

Be aware of **immunogenic foods**

Eat with the **seasons**

Get **as much variety as possible** without getting in trouble - hyperpalatability

Our consistent and fatal mistake:

The "Procrustean Bed" of the **Macronutrient Wars**

Image Source: http://canacopegdl.com/images/procrustean/procrustean-0.jpg

Make your bed fit you!

Works Cited

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