A *FAST* way to longer life…

Harvey S. Hahn, MD, FACC

Program Director, Cardiovascular Fellowship Training Program

Be Well

mind body spirit

American Heart Association®

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Fasting? Crashing?

The Big 3….

- Diet
- Exercise
- Sleep / stress
- What is the ONE thing that can tie all 3 together?
- Intermittent fasting!

Effects of intermittent fasting on body composition and clinical health markers in humans

Grant M. Tinsley and Paul M. La Bounty

2015

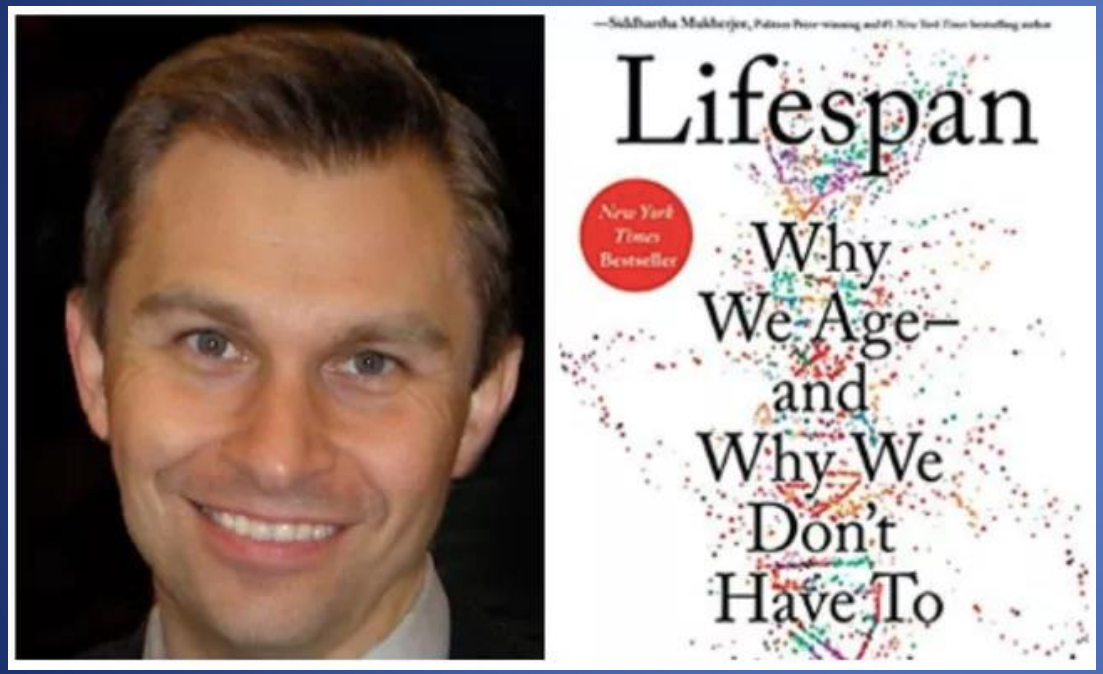
	Wt	Fat (kg)	Chole	Trig	Length
ADF	↓3-7%	↓3-5.5	↓10-21%	↓14-42%	3-12 wks
Whole day	↓3-9%	↓3-9	↓5-20%	↓17-50%	12-24 wks
Time-restricted	?	?	?	?	?

Nutrition Reviews® Vol. 73(10):661–674

Things always happen in 3s…

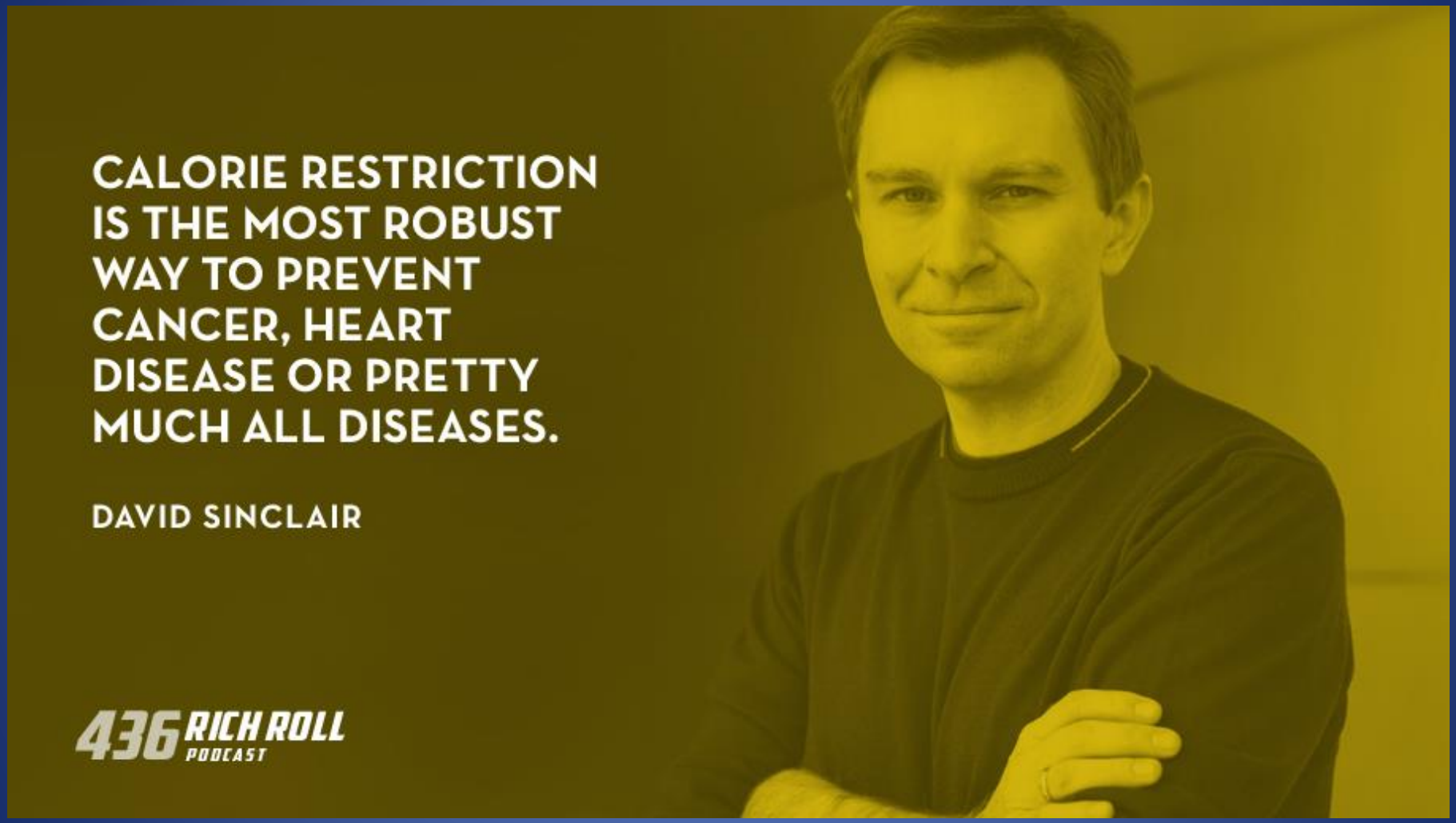
- David Sinclair publishes “Lifespan” Sept 2019.
- The NEJM publishes a *review article* on intermittent fasting Dec 2019.
- Wilkinson et al publishes the easiest and best outcomes data on IF in Jan 2020.

Lifespan…



- Information theory of aging.
- Low level stress activates your survival pathways and tunes up your epigenetic makeup.
- Hunger, exercise, cold exposure all help you live longer!

Calorie restriction (CR)



Fasting is a subgroup of CR.

“I’ll just work it off…”

CALERIE Study

- Comprehensive Assessment of Long-term Effects of Reducing Intake of Energy
- Reduced caloric intake by 25%.
- Average caloric reduction was ~12%.
- Resulted in 10% wt loss.
- BP dropped by 4%, total cholesterol 6%, CRP 47%.

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

Dan L. Longo, M.D., *Editor*

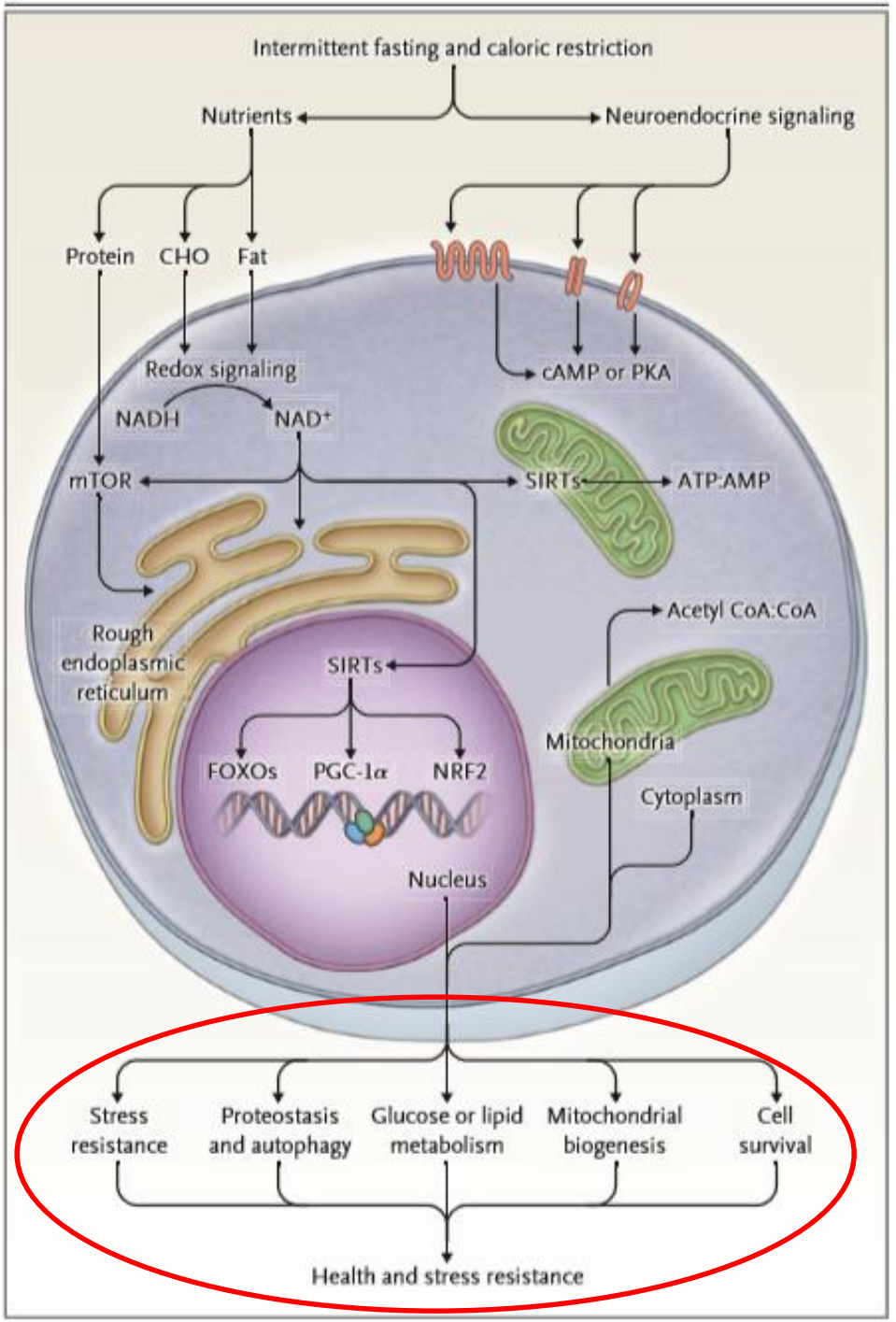
Effects of Intermittent Fasting on Health, Aging, and Disease

Rafael de Cabo, Ph.D., and Mark P. Mattson, Ph.D.

From the Translational Gerontology Branch (R.C.) and the Laboratory of Neurosciences (M.P.M.), Intramural Research Program, National Institute on Aging, National Institutes of Health, and the Department of Neuroscience, Johns Hopkins University School of Medicine (M.P.M.) — both in Baltimore. Address reprint requests to Dr. Mattson at the Department of Neuroscience, Johns Hopkins University School of Medicine, 725 N. Wolfe St., Baltimore, MD 21205, or at mmattso2@jhmi.edu.

This article was updated on December 26, 2019, at NEJM.org.

N Engl J Med 2019;381:2541-51.
DOI: 10.1056/NEJMra1905136


Intermittent fasting and caloric restriction
Nutrients
Neuroendocrine signaling
Protein
CHO
Fat
Redox signaling
NADH
NAD+
cAMP or PKA
mTOR
SIRTs
ATP:AMP
Rough endoplasmic reticulum
Acetyl CoA:CoA
SIRTs
Mitochondria
FOXOs
PGC-1α
NRF2
Cytoplasm
Nucleus
Stress resistance
Proteostasis and autophagy
Glucose or lipid metabolism
Mitochondrial biogenesis
Cell survival
Health and stress resistance

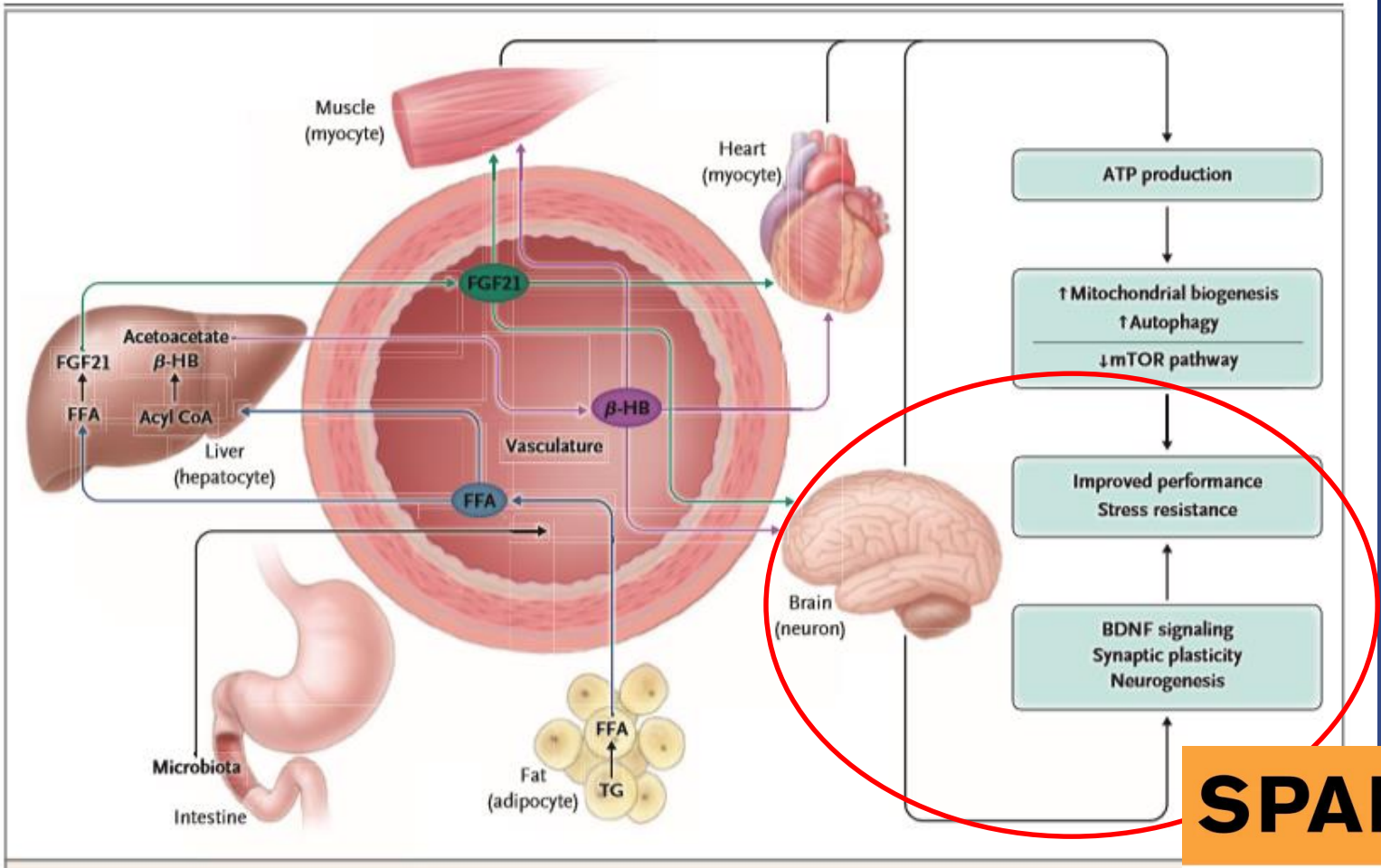
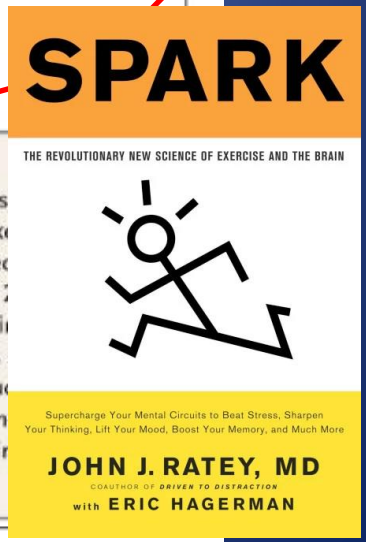


Figure 2. Metabolic Adaptations to Intermittent Fasting.

Energy restriction for 10 to 14 hours or more results in depletion of liver glycogen stores and hydrolysis of triglycerides (TGs
acids (FFAs) in adipocytes. FFAs released into the circulation are transported into hepatocytes, where they produce the k
acetoacetate and β-hydroxybutyrate (β-HB). FFAs also activate the transcription factors peroxisome proliferator–activate
(PPAR-α) and activating transcription factor 4 (ATF4), resulting in the production and release of fibroblast growth factor
protein with widespread effects on cells throughout the body and brain. β-HB and acetoacetate are actively transported i
they can be metabolized to acetyl CoA, which enters the tricarboxylic acid (TCA) cycle and generates ATP. β-HB also has
tions, including the activation of transcription factors such as cyclic AMP response element–binding protein (CREB) and nu
(NF-κB) and the expression of brain-derived neurotrophic factor (BDNF) in neurons. Reduced levels of glucose and amin
fasting result in reduced activity of the mTOR pathway and up-regulation of autophagy. In addition, energy restriction sti
chondrial biogenesis and mitochondrial uncoupling.



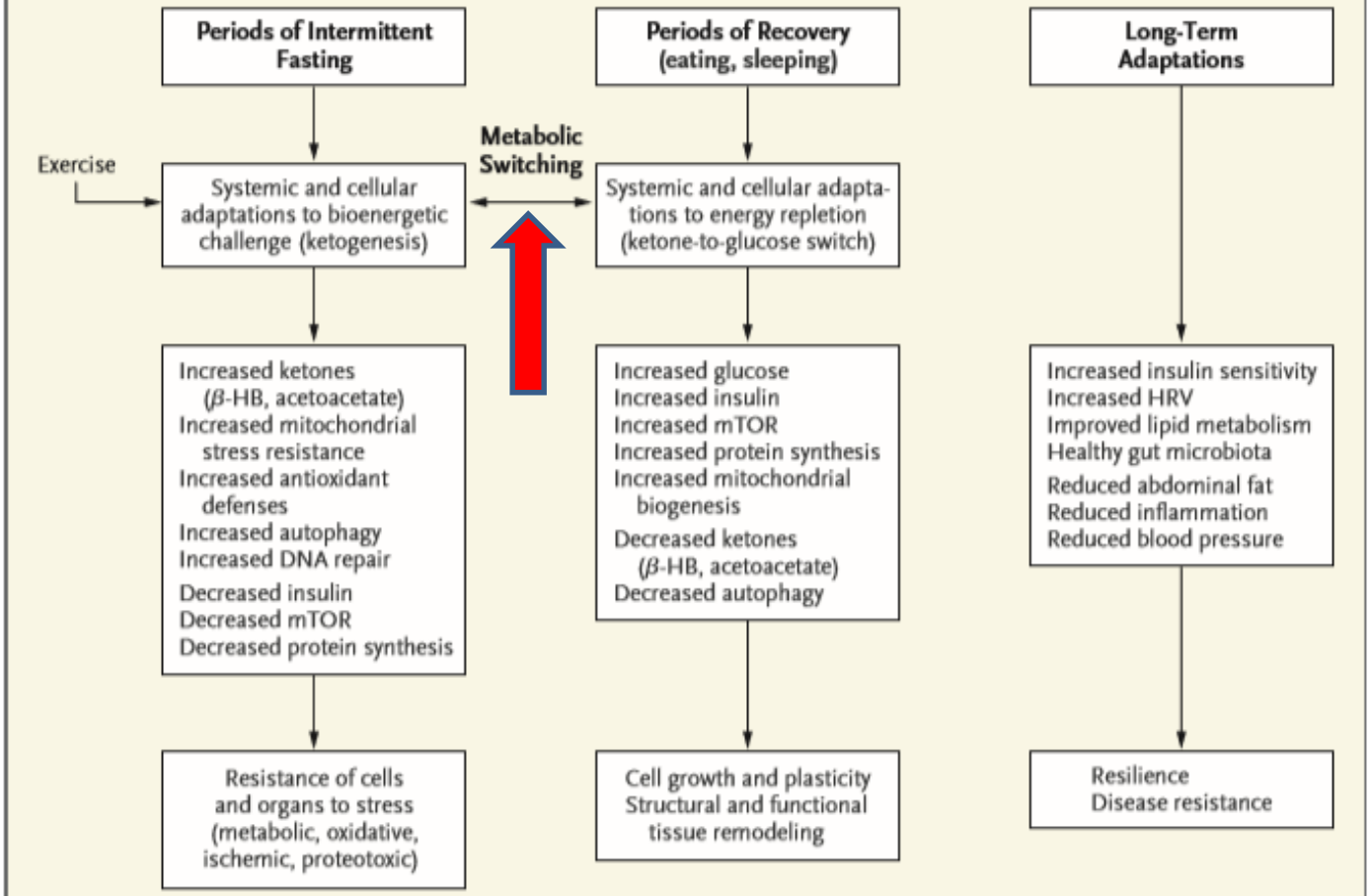


Figure 3. Cellular and Molecular Mechanisms Underlying Improved Organ Function and Resistance to Stress and Disease with Intermittent Metabolic Switching.

Periods of dietary energy restriction sufficient to cause depletion of liver glycogen stores trigger a metabolic switch toward use of fatty acids and ketones. Cells and organ systems adapt to this bioenergetic challenge by activating signaling pathways that bolster mitochondrial function, stress resistance, and antioxidant defenses while up-regulating autophagy to remove damaged molecules and recycle their components. During the period of energy restriction, cells adopt a stress-resistance mode through reduction in insulin signaling and overall protein synthesis. Exercise enhances these effects of fasting. On recovery from fasting (eating and sleeping), glucose levels increase, ketone levels plummet, and cells increase protein synthesis, undergoing growth and repair. Maintenance of an intermittent-fasting regimen, particularly when combined with regular exercise, results in many long-term adaptations that improve mental and physical performance and increase disease resistance. HRV denotes heart-rate variability.

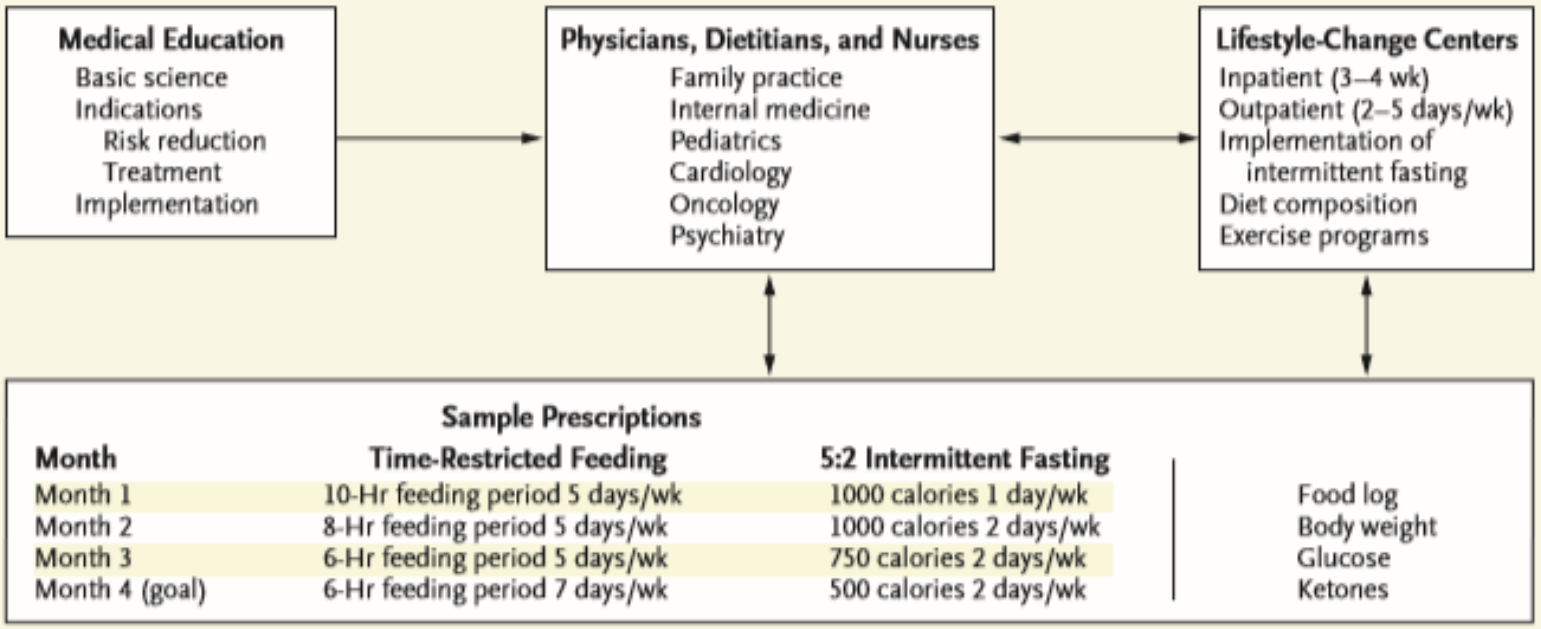


Figure 4. Incorporation of Intermittent-Fasting Patterns into Health Care Practice and Lifestyles.

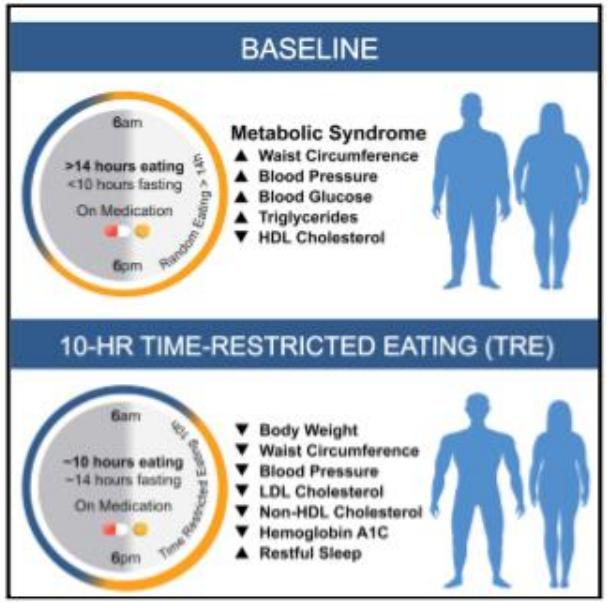
As a component of medical school training in disease prevention, students could learn the basics of how intermittent fasting affects metabolism and how cells and organs respond adaptively to intermittent fasting, the major indications for intermittent fasting (obesity, diabetes, cardiovascular disease, and cancers), and how to implement intermittent-fasting prescriptions to maximize long-term benefits. Physicians can incorporate intermittent-fasting prescriptions for early intervention in patients with a range of chronic conditions or at risk for such conditions, particularly those conditions associated with overeating and a sedentary lifestyle. One can envision inpatient and outpatient facilities staffed by experts in diet, nutrition, exercise, and psychology that will help patients make the transition to sustainable intermittent-fasting and exercise regimens (covered by basic health insurance policies). As an example of a specific prescription, the patient could choose either a daily time-restricted feeding regimen (an 18-hour fasting period and a 6-hour eating period) or the 5:2 intermittent-fasting regimen (fasting [i.e., an intake of 500 calories] 2 days per week), with a 4-month transition period to accomplish the goal. To facilitate adherence to the prescription, the physician's staff should be in frequent contact with the patient during the 4-month period and should closely monitor the patient's body weight and glucose and ketone levels.

Clinical and Translational Report

Cell Metabolism

Ten-Hour Time-Restricted Eating Reduces Weight, Blood Pressure, and Atherogenic Lipids in Patients with Metabolic Syndrome

Graphical Abstract



Highlights

- 10 h time-restricted eating (TRE) in metabolic syndrome (MetS) promotes weight loss
- TRE in MetS reduces waist circumference, percent body fat, and visceral fat
- TRE in MetS lowers blood pressure, atherogenic lipids, and glycated hemoglobin
- Benefits of TRE are "add-ons" to statin and anti-hypertensive medications

Authors

Michael J. Wilkinson, Emily N.C. Manoogian, Adena Zadourian, ..., Saket Navlakha, Satchidananda Panda, Pam R. Taub

Correspondence

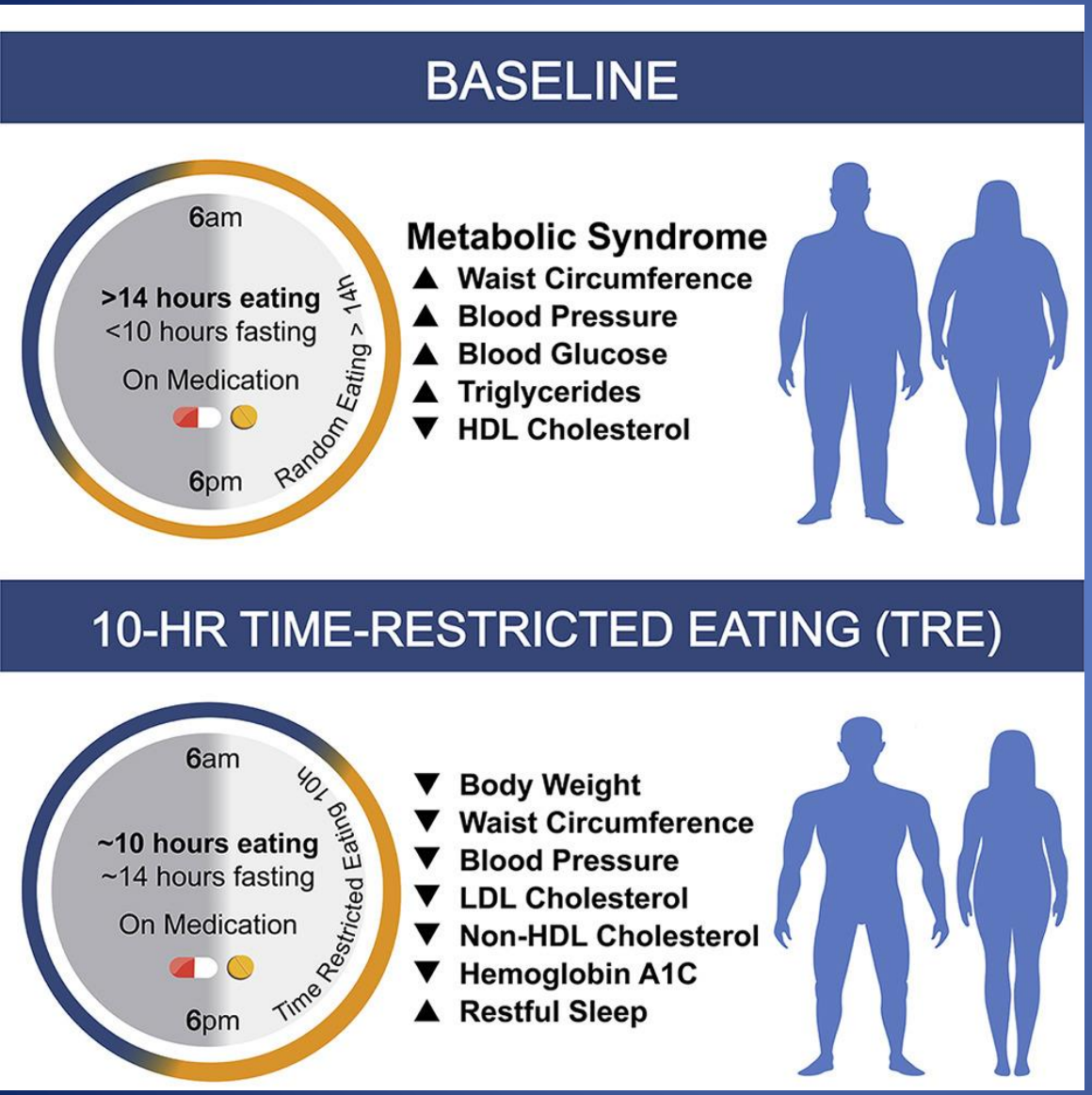
satchin@salk.edu (S.P.), ptaub@ucsd.edu (P.R.T.)

In Brief

Wilkinson and Manoogian et al. studied the impact of time-restricted eating in metabolic syndrome by reducing participant's daily eating window from ≥14 h to a self-selected 10 h window for 12 weeks. Time-restricted eating led to weight loss, healthier body composition, lower blood pressure, and decreased levels of cardiovascular disease-promoting lipids.

Wilkinson et al., 2020, Cell Metabolism *31*, 92–104
January 7, 2020
https://doi.org/10.1016/j.cmet.2019.11.004

CellPress

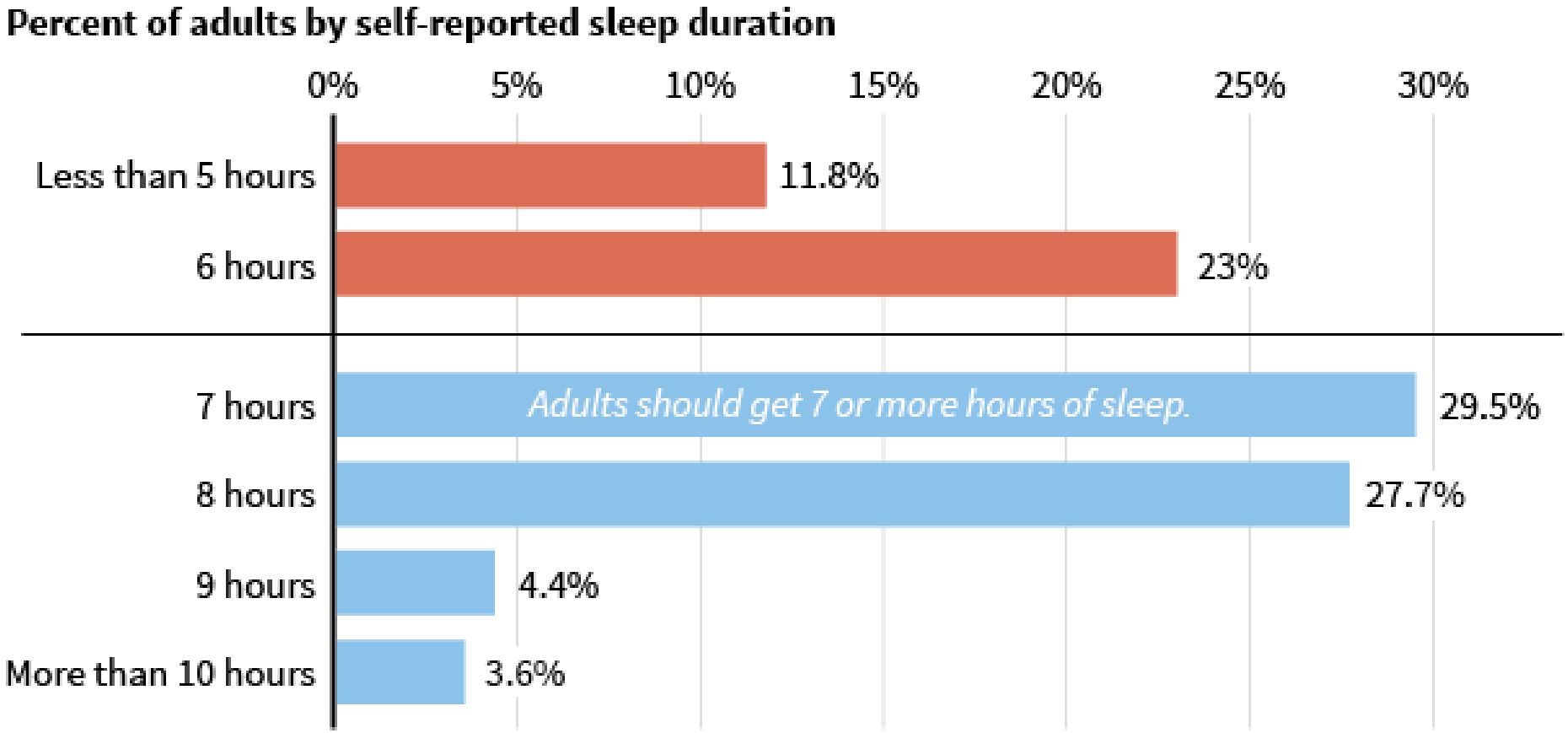


- Small study.
- 10 hr feeding, not 8.
- No change in diet quality.
- No exercise component.
- Pt already on BP meds and statins!
- CR of 8.6%.
- Lost 3.3 kg (7.3 lbs).
- 12 wks long.

So what's the easiest way to fast?

RMR persists even when you sleep

More Than A Third Of U.S. Adults Don't Get Enough Sleep



Source: CDC

The Huffington Post

ORIGINAL RESEARCH

American Heart Association.

Interplay of Objective Sleep Duration and Cardiovascular and Cerebrovascular Diseases on Cause-Specific Mortality

Julio Fernandez-Mendoza, PhD, CBSM, DBSM; Fan He, MS; Alexandros N. Vgontzas, MD, ScD; Duanping Liao, MD, PhD; Edward O. Bixler, PhD

(*J Am Heart Assoc*. 2019;8:e013043. DOI: 10.1161/JAHA.119.013043.)

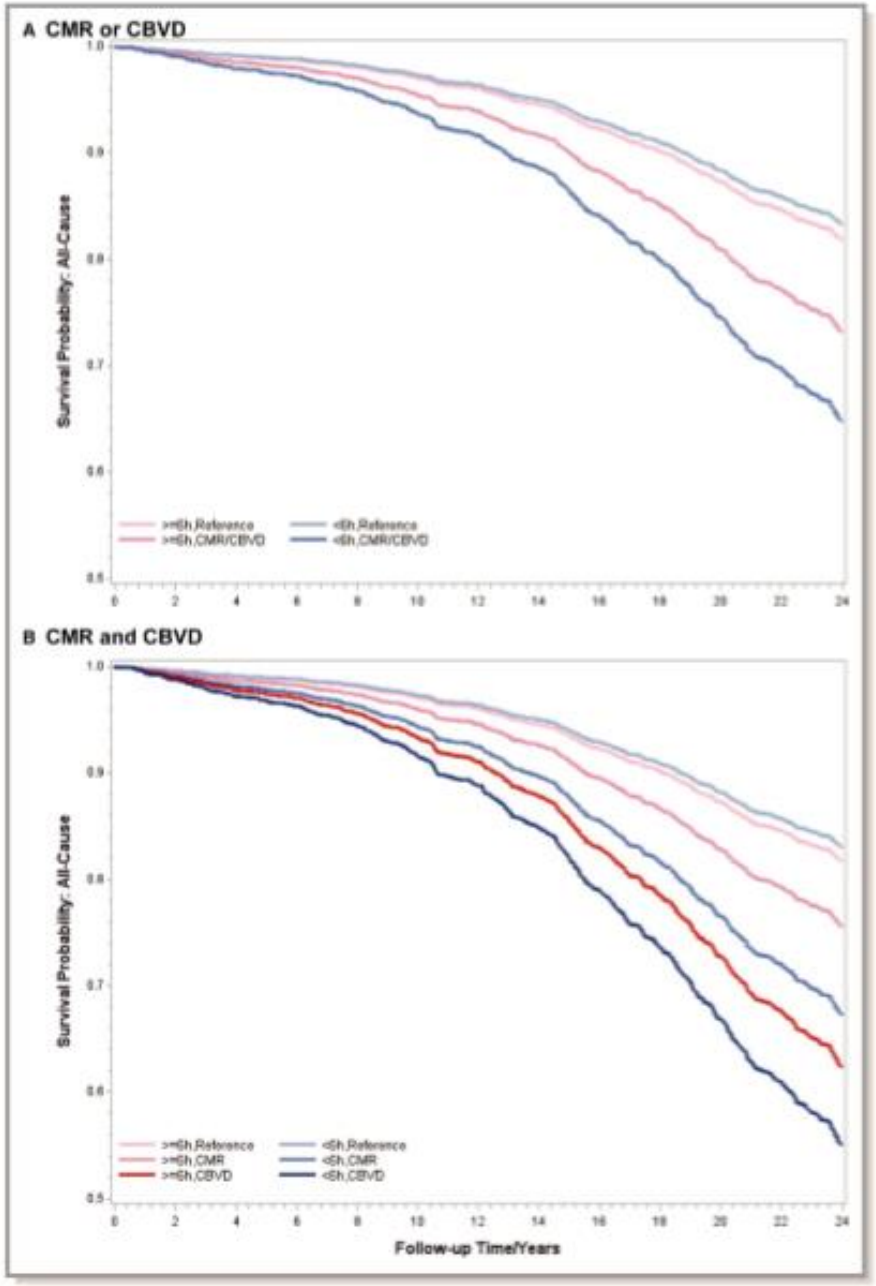


Figure 1. Multivariable-adjusted survival curves for all-cause mortality associated with cardiometabolic risk factors (CMRs) and cardiovascular and cerebrovascular disease (CBVD) at baseline. **A,** Survival curves for all-cause mortality associated with CMRs or CBVD. **B,** Survival curves for all-cause mortality associated with CMRs and CBVD. All data adjusted for age, race, sex, education, body mass index, smoking, alcohol use, apnea-hypopnea index, other physical health conditions, and mental health conditions.

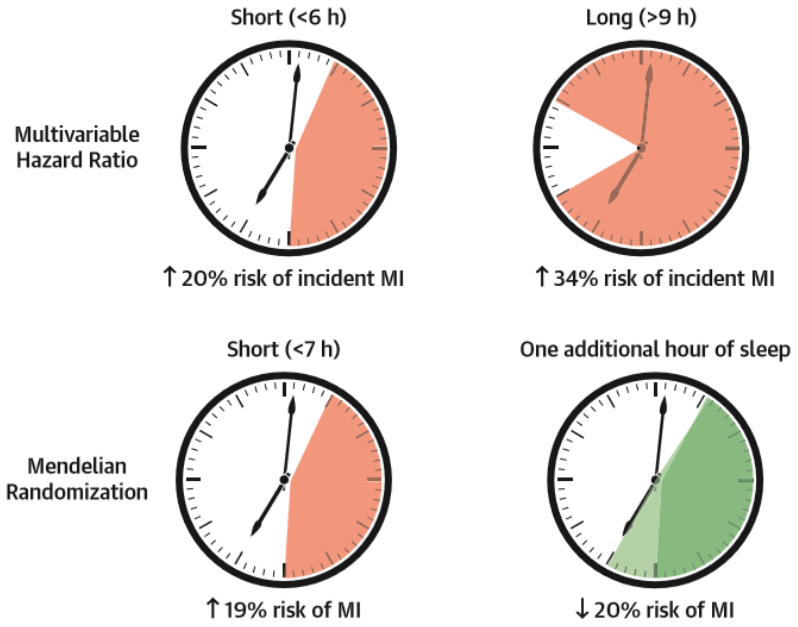
JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY
VOL. 74, NO. 10, 2019

PUBLISHED BY ELSEVIER

Sleep Duration and Myocardial Infarction

Iyas Daghlas, BS,[a,b] Hassan S. Dashti, PhD, RD,[a,b] Jacqueline Lane, PhD,[a,b,c] Krishna G. Aragam, MD, MS,[a,b,d] Martin K. Rutter, MD,[e,f] Richa Saxena, PhD,[a,b,c] Céline Vetter, PhD[a,g]

CENTRAL ILLUSTRATION **Associations of Sleep Duration With Coronary Disease: Observational and 2-Sample Mendelian Randomization**



Daghlas, I. et al. J Am Coll Cardiol. 2019;74(10):1304–14.

Magnitude of effects observed for effects of phenotypic and genetically varying sleep duration. Note, that although the clocks show variable sleep timing for short and long sleep, the observed effects are independent of self-reported sleep timing preference. MI = myocardial infarction; MR = Mendelian randomization.

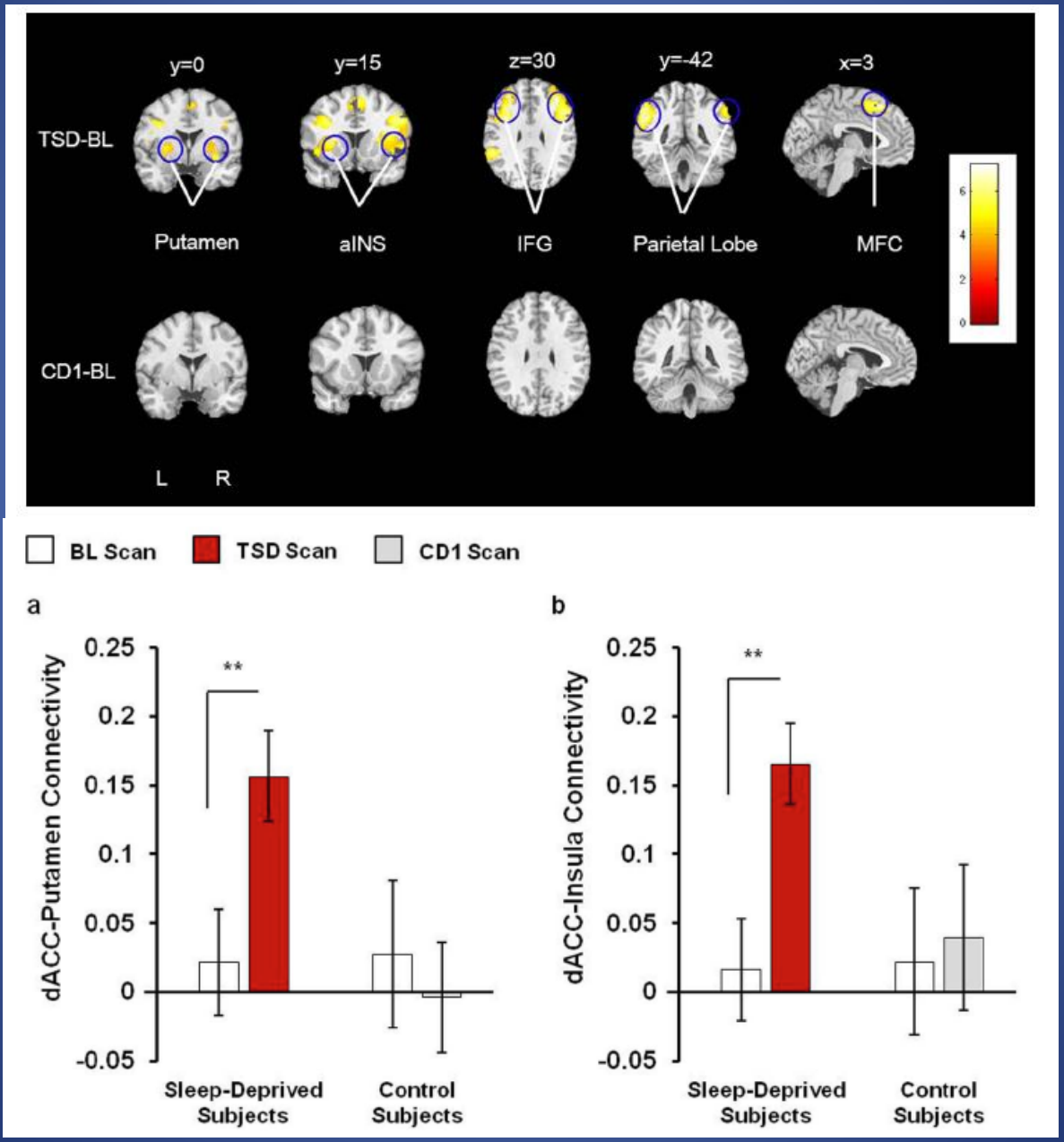
Altered salience network connectivity predicts macronutrient intake after sleep deprivation

Zhuo Fang[1]*, Andrea M. Spaeth[2]*, Ning Ma[1], Senhua Zhu[1], Siyuan Hu[1], Namni Goel[3], John A. Detre[1], David F. Dinges[3] & Hengyi Rao[1,3]

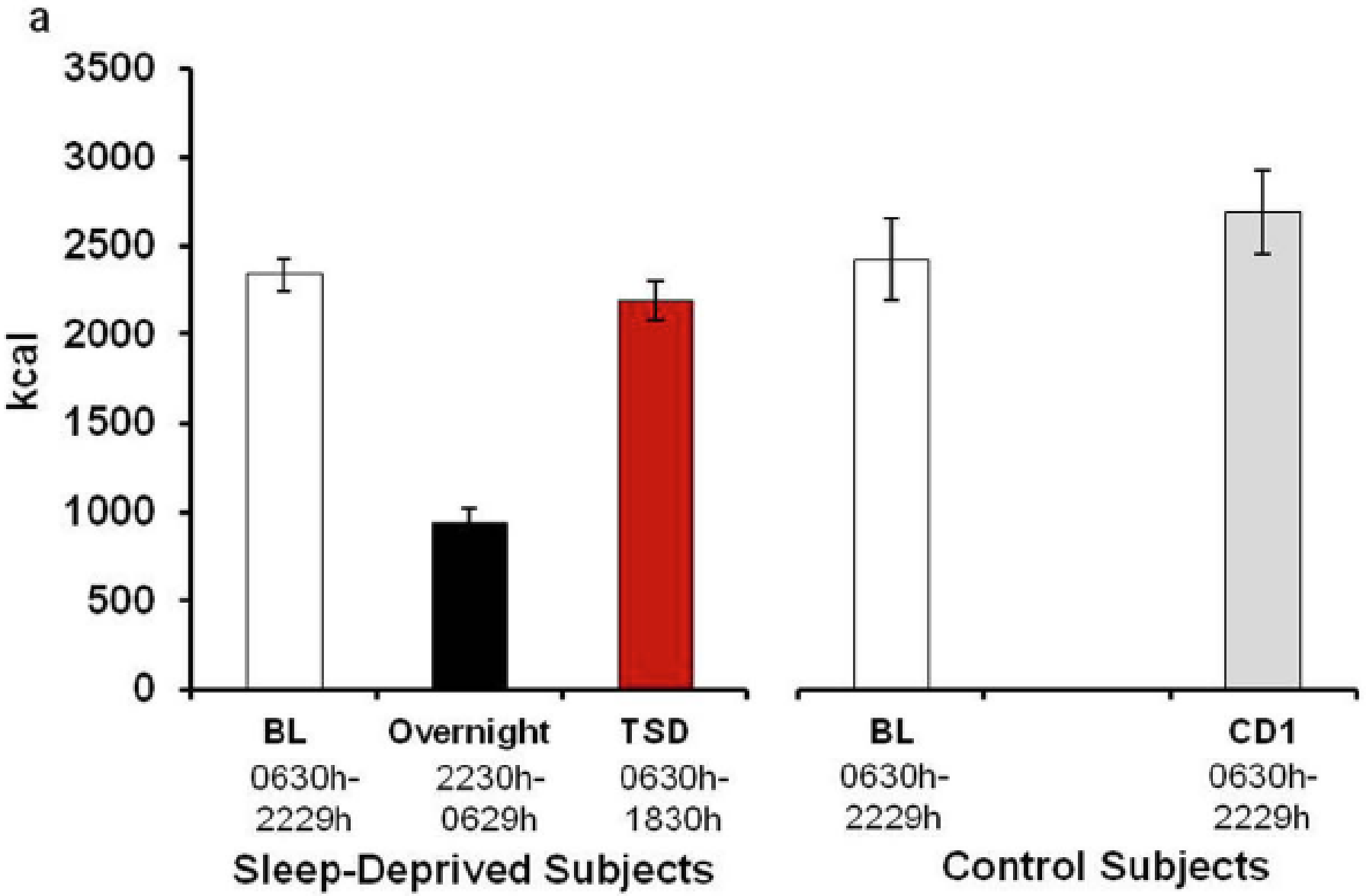
[1]Center for Functional Neuroimaging, Department of Neurology, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, [2]Center for Sleep and Circadian Neurobiology, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, [3]Division of Sleep and Chronobiology, Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA.

- Poor sleep lowers will power.
- It also increases caloric consumption and fat intake the next day.
- How / why?

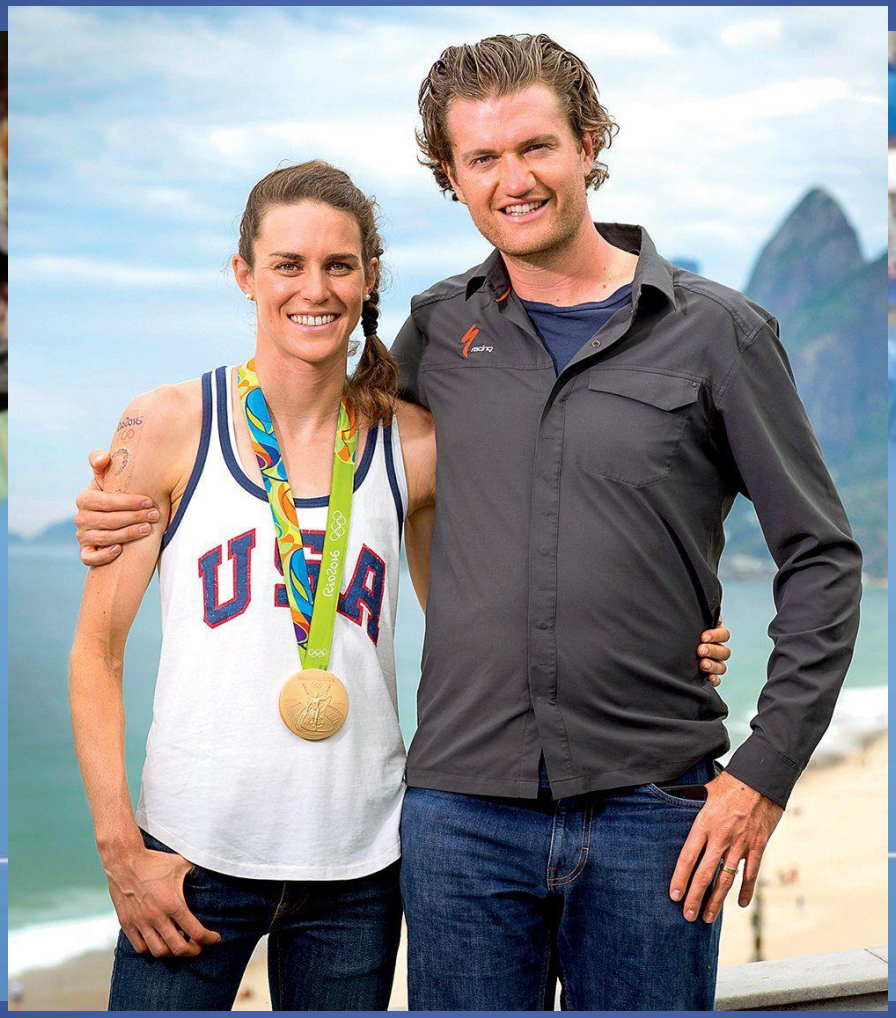
“I deserve this…”



An extra ~1000 calories…

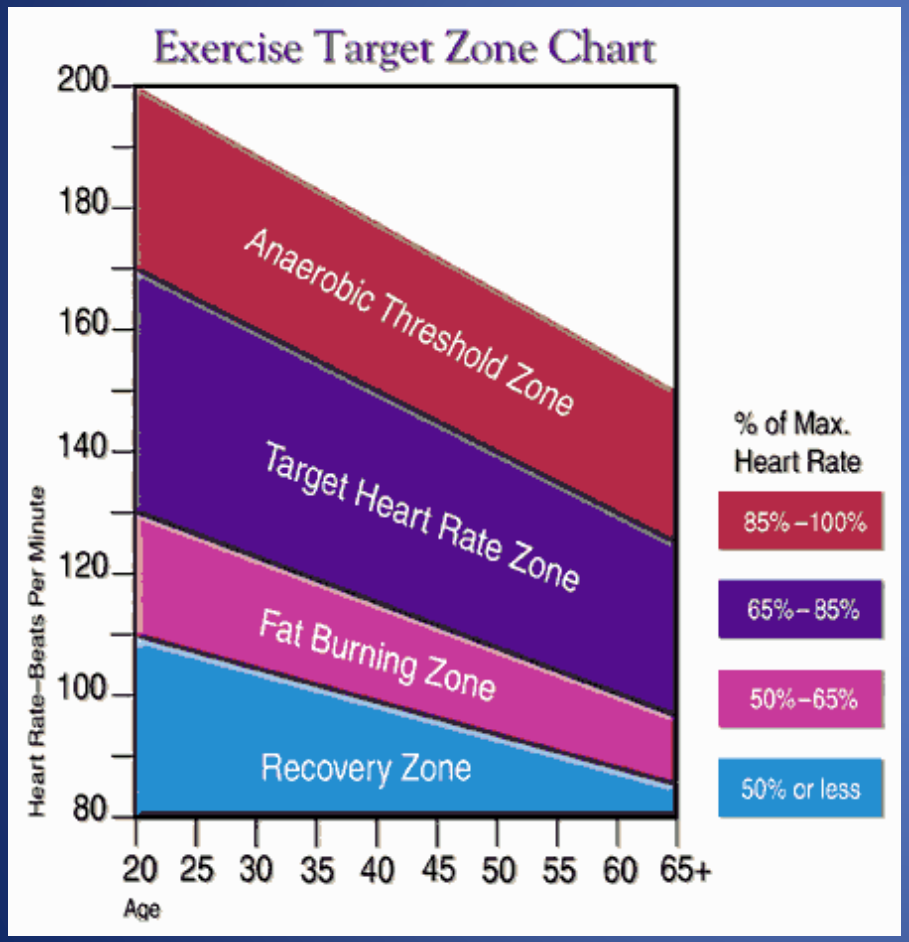


Gwen Jorgenson



Sleeps 40-50% of her LIFE!

Myth: Cardio is the best way to drop weight…



- What do you call doing cardio 5 days a wk?
- *The road to nowhere!*
- *Why?*

The Law of Diminishing Returns…

Constrained Total Energy Expenditure and Metabolic Adaptation to Physical Activity in Adult Humans

Herman Pontzer,[1,2,*] Ramon Durazo-Arvizu,[3] Lara R. Dugas,[3] Jacob Plange-Rhule,[4] Pascal Bovet,[5,6] Terrence E. Forrester,[7] Estelle V. Lambert,[8] Richard S. Cooper,[3] Dale A. Schoeller,[9] and Amy Luke[3]
[1]Department of Anthropology, Hunter College, City University of New York, 695 Park Avenue, New York, NY 10065, USA
[2]New York Consortium for Evolutionary Primatology, New York, NY 10065, USA
[3]Public Health Sciences, Stritch School of Medicine, Loyola University Chicago, 2160 South First Avenue, Maywood, IL 60153, USA
[4]Kwame Nkrumah University of Science and Technology, Kumasi, Ghana
[5]Institute of Social & Preventive Medicine, Lausanne University Hospital, Rue de la Corniche 10, 1010 Lausanne, Switzerland
[6]Ministry of Health, PO Box 52, Victoria, Mahé, Seychelles
[7]UWI Solutions for Developing Countries, The University of the West Indies, 25 West Road, UWI Mona Campus, Kingston 7, Jamaica
[8]Research Unit for Exercise Science and Sports Medicine, University of Cape Town, PO Box 115, Newlands 7725, Cape Town, South Africa
[9]Nutritional Sciences, Biotechnology Center, University of Wisconsin–Madison, 425 Henry Mall, Madison, WI 53705, USA
*Correspondence: herman.pontzer@hunter.cuny.edu
http://dx.doi.org/10.1016/j.cub.2015.12.046

Two theories…

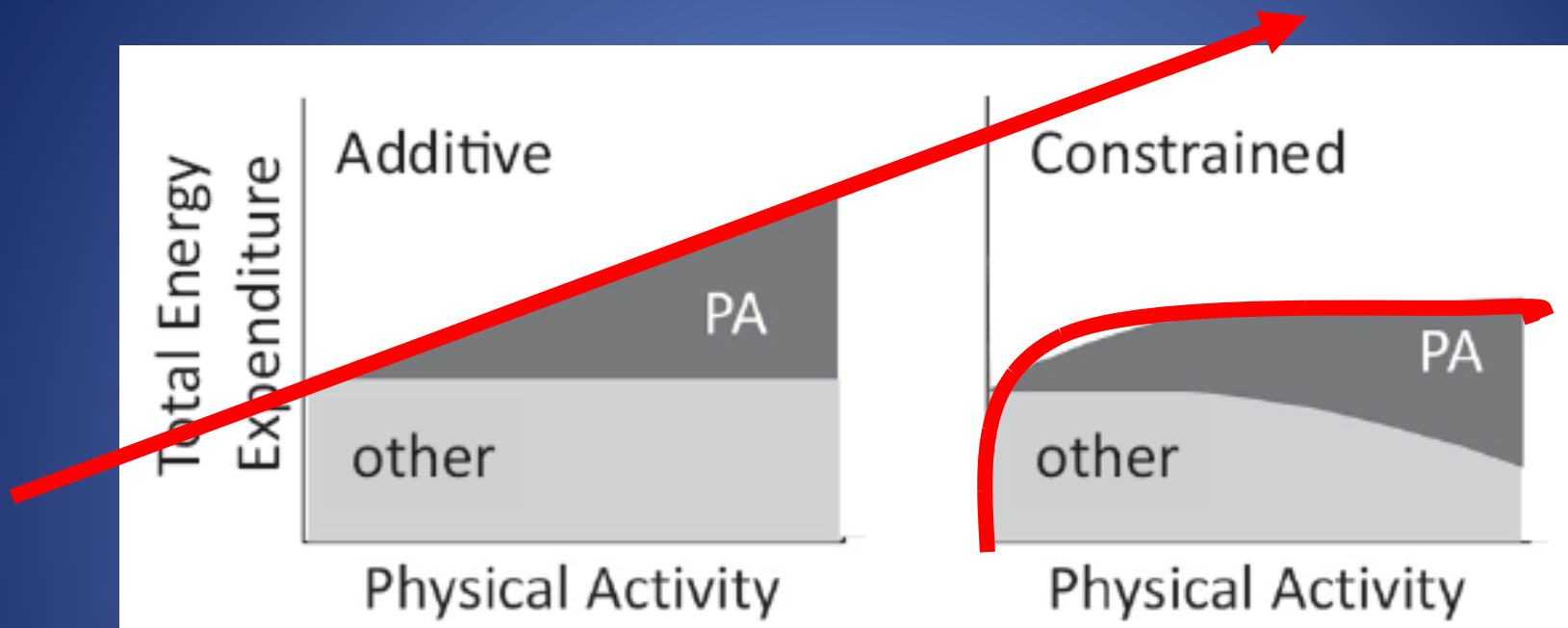
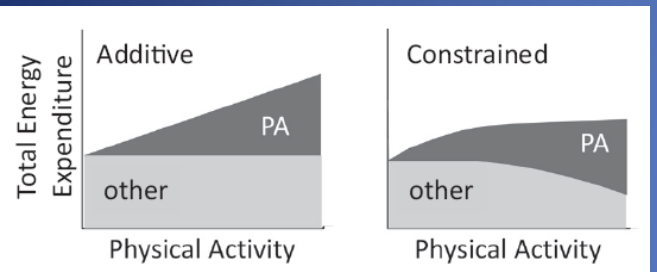


Figure 1. Schematic of Additive Total Energy Expenditure and Constrained Total Energy Expenditure Models

In Additive total energy expenditure models, total energy expenditure is a simple linear function of physical activity, and variation in physical activity energy expenditure (PA) determines variation in total energy expenditure. In Constrained total energy expenditure models, the body adapts to increased physical activity by reducing energy spent on other physiological activity, maintaining total energy expenditure within a narrow range.

The winner is…



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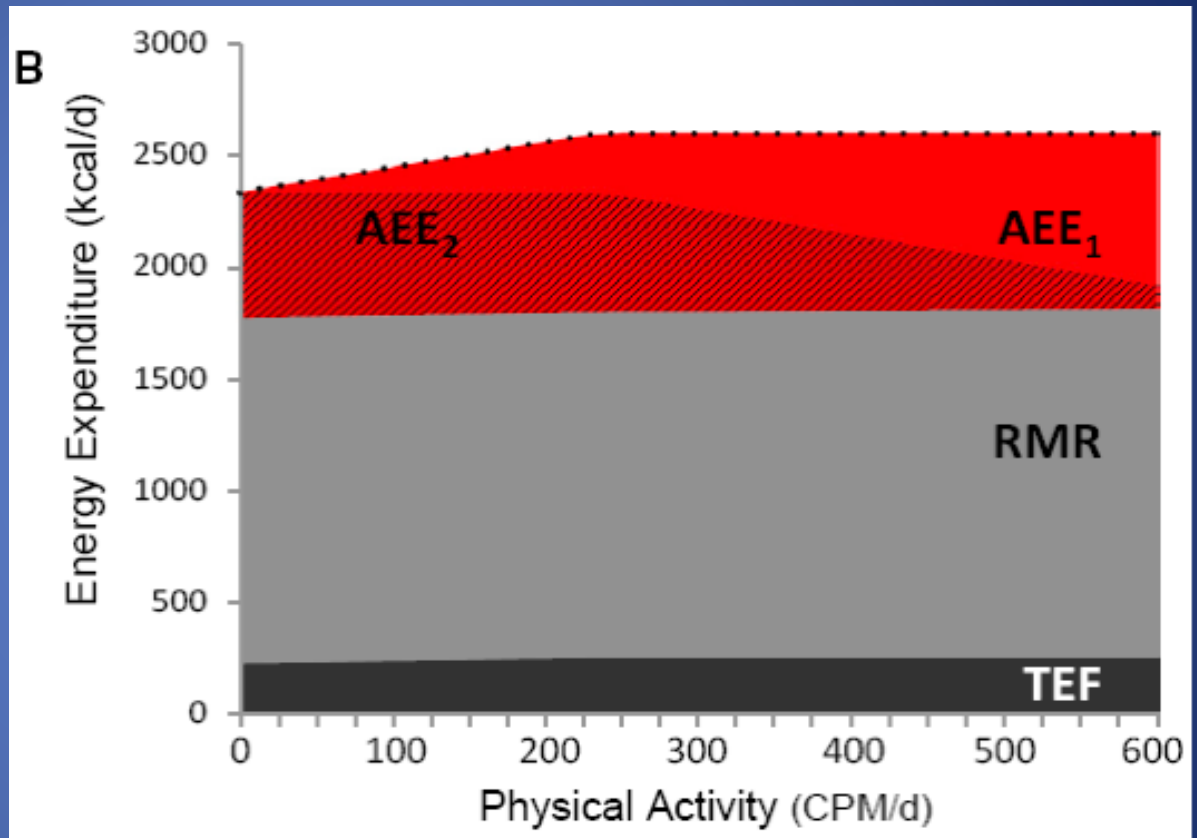
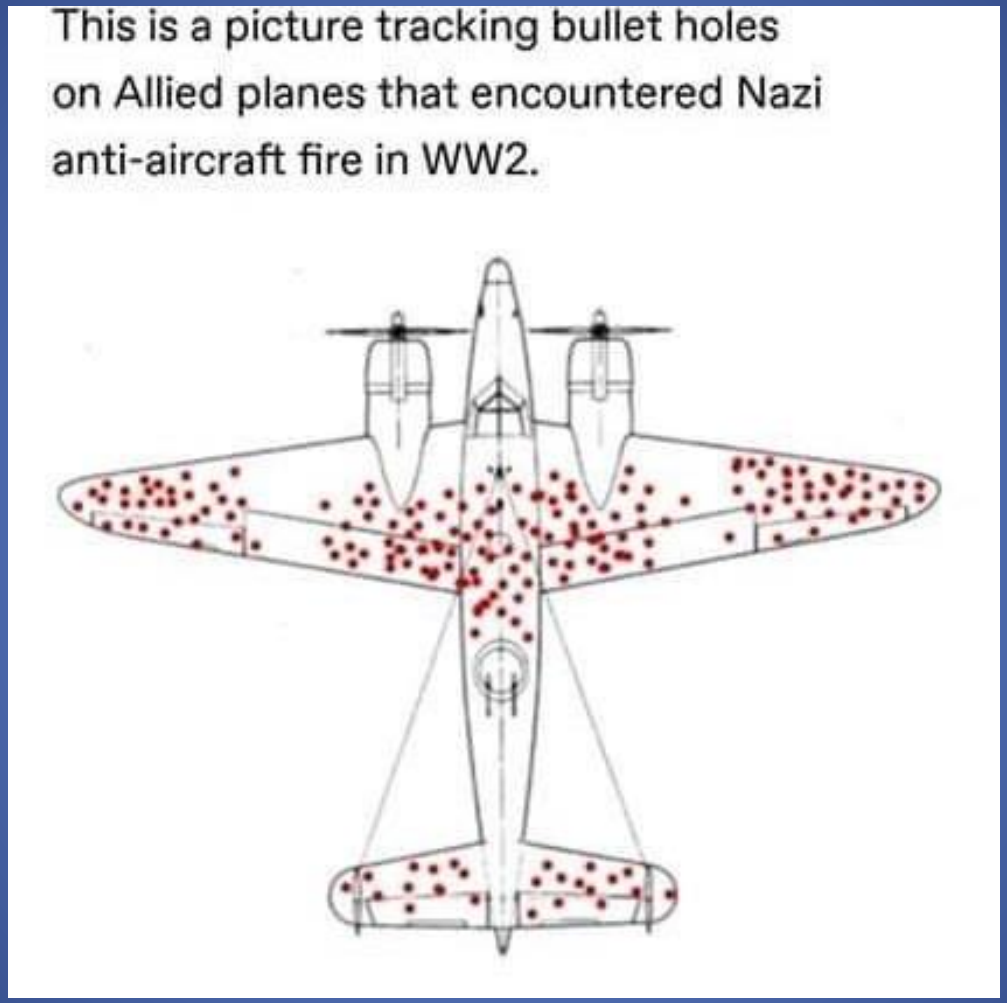


Figure 3. The Effect of Physical Activity on Total Energy Expenditure and Its Components

IF vs keto in Exercise

- What are the 2 main questions about fasted and ketogenic exercise?
- #1-will it increase performance?
- #2-will it lead to more fat burn?

Keto-off target?



What was the logic error?

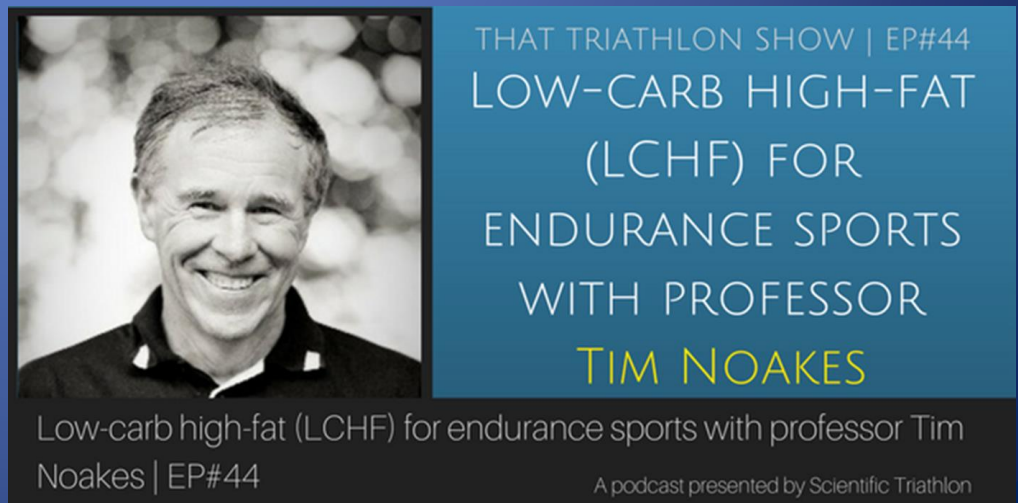
IF vs keto

Keto

- Early wt loss is due to water loss.
- Biggest change is eliminating garbage processed carbs (50-60% of the SAD).
- If increased bad fats then lipids levels can rise.
- ***Most never really achieve ketosis.***
- ***Most don't even try to measure ketosis.***

IF

- Shrinks stomach.
- Increases leptin which reduces hunger in the long run.
- Associated with a survival benefit.
- ***8-12 hours of fasting typically induces a state of ketosis.***



The data

- Carbs are best for performance.
- Fat is not.
- Protein is best for muscle gains.
- Keto is trendy and cool. Exclusive diets sell better. Hard to make the case that your program is better if it's like other programs.
- The internet and social media are terrible.
- Pubmed hits as of 1.23.2020

	Performance	Fat loss
Keto (2880)	13	17
IF (118,980)	526	1908

Sage Canaday
@SageCanaday

Follow

Why do people lose weight on a #keto diet? Um, because they end up eating less calories (than their former diet): Laws of Thermodynamics. But remember weight loss does" not always equal "better health". That should not always be the goal. One can have a low BMI and be unhealthy.

6:57 AM - 9 Mar 2019

21 Retweets **260** Likes

28 21 260

Sage Canaday @SageCanaday · 9 Mar 2019

yes of course, but high protein and high fat meals are more filling/satiating....so they eat smaller portion size and many into keto are also into "intermittent fasting"... so they maybe only eat two meals a day (instead of 3). Hence, less calories.

1 1

Sage Canaday
@SageCanaday

Follow

Replying to @NegativeSplits

Give me a list of low carb high animal fat "keto cultures" doing well health and longevity-wise long term...there simply arent any. Thats not my opinion. You obviously havent looked into this much.

7:12 AM - 8 Feb 2019

Sage uses carbs during races!

Sage also does his long runs fasted.

Proceedings of the Nutrition Society (2019), **78**, 110–117
doi:10.1017/S0029665118002574
 First published online 18 October 2018

The Nutrition Society Scottish Section Meeting was held at the Royal College of Physicians and Surgeons, Glasgow on 26–27 March 2018

Conference on 'Nutrient–nutrient interaction'
Symposium 3: The mechanisms of nutrient interactions

Is exercise best served on an empty stomach?

Gareth A. Wallis[1]* and Javier T. Gonzalez[2]
[1]*School of Sport, Exercise and Rehabilitation Sciences, University of Birmingham, Birmingham B15 2TT, UK*
[2]*Department for Health, University of Bath, Bath BA2 7AY, UK*

- Currently we don't know!
- Mixed data for both keto and IF on exercise performance.
- Everyone is different.
- Recommend experimenting on yourself.

Exercise and timing of food intake

Acute response + Chronic adaptation

Energy balance behaviours

↓ daily energy intake[27,31]
daily energy expenditure?

??

Blood

Glycaemic control – variable [23,25,26]
↓ TAG[21,22]

??

Liver

↔Liver fat[15]

??

Muscle

↓ glycogen use[12]
↑ IMTAG use[14]
↑ *GLUT4*[45]
↑ *FAT/CD36*[44]
↑ *CPT1*[44]
↑ *PDK4*[44]
↑ *AMPK*[44]

↑glycogen (basal)[35]
↑GLUT4[40]
↑ FAT/CD36[40]
↑ FABP[47]

Adipose

↑ NEFA use[13]
↑ *GLUT4*[43]
↑ *FAT/CD36*[43]
↑ *PDK4*[43]
↑ *IRS2*[43]

??

British Journal of Nutrition (2016), **116**, 1153–1164
doi:10.1017/S0007114516003160

Effects of aerobic exercise performed in fasted *v.* fed state on fat and carbohydrate metabolism in adults: a systematic review and meta-analysis

Alexandra Ferreira Vieira[2*], Rochelle Rocha Costa[1,2], Rodrigo Cauduro Oliveira Macedo[1,3], Leandro Coconcelli[1,2] and Luiz Fernando Martins Kruel[1,2]

[1]*Physical Education, Physiotherapy and Dance School, Federal University of Rio Grande do Sul, 750, Felizardo Street, 90690-200 Porto Alegre, Brazil*
[2]*Research Group on Water and Land Activities, Federal University of Rio Grande do Sul, 750, Felizardo Street, 90690-200 Porto Alegre, Brazil*
[3]*Research Group on Exercise Physiology and Biochemistry, Federal University of Rio Grande do Sul, 750, Felizardo Street, 90690-200 Porto Alegre, Brazil*

(Submitted 7 March 2016 – Final revision received 29 July 2016 – Accepted 3 August 2016 – First published online 9 September 2016)

- Decreased insulin production.
- Decreased glucose levels.
- And…

More fat oxidation!

Study name	Difference in means	SE	Variance	Lower limit	Upper limit	*Z*-value	*P*
			Statistics for each study				
Bergman & Brooks, 1999a	–7·87	7·65	58·57	–22·87	7·13	–1·03	0·304
Bergman & Brooks, 1999b	–4·49	5·24	27·46	–14·76	5·78	–0·86	0·391
Farah & Gill, 2013	–5·70	1·84	3·38	–9·30	–2·10	–3·10	0·002
Gonzalez *et al.*, 2013	–6·00	4·46	19·86	–14·74	2·74	–1·35	0·178
Isacco *et al.*, 2012a	–4·59	1·86	3·48	–8·24	–0·94	–2·46	0·014
Isacco *et al.*, 2012b	–1·74	1·20	1·45	–4·10	0·62	–1·45	0·148
Little *et al.*, 2010	–5·15	3·43	11·74	–11·87	1·57	–1·50	0·133
Massicotte *et al.*, 1990	–5·00	4·99	24·99	–14·80	4·80	–1·00	0·317
Maughan & Gleeson, 1988	–0·90	7·63	58·27	–15·86	14·06	–0·12	0·906
Paul *et al.*, 1996	–41·40	17·54	307·60	–75·78	–7·03	–2·36	0·018
Whitley *et al.*, 1998	–14·38	9·67	93·52	–33·33	4·57	–1·49	0·137
Willcutts *et al.*, 1988	–2·62	1·02	1·04	–4·62	–0·62	–2·57	0·010
Wu *et al.*, 2003	–18·10	5·16	26·65	–28·22	–7·98	–3.51	0·000
	–3·53	0·63	0·40	–4·76	–2·30	–5·62	0·000

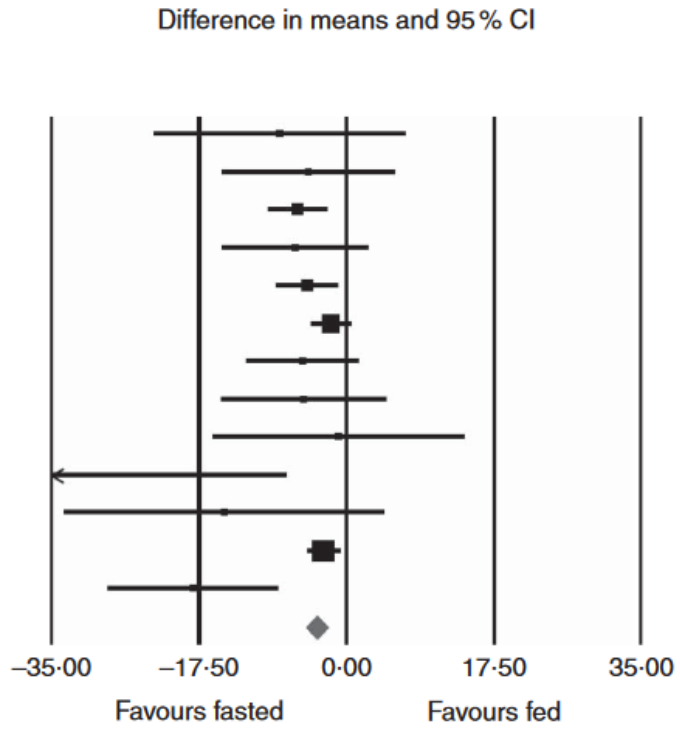


Fig. 4. Fat oxidation (g) during exercise performed in the fasted state *v.* fed state. ■, Study-specific estimates: ◆, pooled estimates of fixed-effects meta-analyses.

Alternate Day Fasting and Endurance Exercise Combine to Reduce Body Weight and Favorably Alter Plasma Lipids in Obese Humans

Surabhi Bhutani, Monica C. Klempel, Cynthia M. Kroeger, John F. Trepanowski and Krista A. Varady

Obesity (2013) **21**, 1370-1379. doi:10.1002/oby.20353

TABLE 3 Body weight and body composition during the 12-week trial

	Intervention	Week 1	Week 12	P-value[a]	P-value[b]	Change[c]	P-value[d]
Body weight (kg)	Combination	91 ± 6	85 ± 6	<0.001	0.393	−6 ± 4[a]	<0.001
	ADF	94 ± 3	91 ± 3	<0.001		−3 ± 1[b]	
	Exercise	93 ± 2	92 ± 2	0.027		−1 ± 0[b]	
	Control	93 ± 5	93 ± 5	0.577		0 ± 0[c]	
Body mass index (kg/m^2)	Combination	35 ± 1	33 ± 1	<0.001	0.334	−2 ± 0[a]	<0.001
	ADF	35 ± 1	34 ± 1	<0.001		−1 ± 0[b]	
	Exercise	35 ± 1	34 ± 1	0.030		−1 ± 0[b]	
	Control	35 ± 1	35 ± 1	0.707		0 ± 0[c]	
Fat mass (kg)	Combination	45 ± 2	40 ± 2	<0.001	0.054	−5 ± 1[a]	<0.001
	ADF	43 ± 2	41 ± 2	0.008		−2 ± 1[b]	
	Exercise	46 ± 2	45 ± 2	0.182		−1 ± 0[b]	
	Control	43 ± 4	43 ± 4	0.570		0 ± 1[b]	
Fat free mass (kg)	Combination	46 ± 2	46 ± 2	0.221	0.299	0 ± 1	0.527
	ADF	51 ± 2	50 ± 2	0.031		−1 ± 1	
	Exercise	48 ± 1	47 ± 1	0.321		−1 ± 0	
	Control	50 ± 2	49 ± 2	0.693		−1 ± 1	
Waist circumference (cm)	Combination	96 ± 2	88 ± 1	<0.001	0.310	−8 ± 1[a]	<0.001
	ADF	100 ± 2	95 ± 2	<0.001		−5 ± 1[b]	
	Exercise	98 ± 2	95 ± 2	<0.001		−3 ± 1[b]	
	Control	98 ± 3	97 ± 2	0.640		−1 ± 1[b]	

Digging deeper…

TABLE 5 LDL particle size during the 12-week trial

	Intervention	Week 1	Week 12	P-value[a]	P-value[b]	Change[c]	P-value[d]
LDL particle size (Å)	Combination	260 ± 1	264 ± 2 [a]	<0.001	0.031	4 ± 1 [a]	0.010
	ADF	261 ± 1	266 ± 1 [a]	<0.001		5 ± 1 [a]	
	Exercise	261 ± 2	262 ± 2 [b]	0.426		1 ± 1 [b]	
	Control	259 ± 1	260 ± 2 [b]	0.884		0 ± 1 [b]	
Large LDL particles (%)	Combination	38 ± 4	45 ± 5 [a]	0.142	0.014	7 ± 5	0.064
	ADF	36 ± 3	51 ± 4[a]	<0.001		15 ± 3	
	Exercise	39 ± 3	40 ± 4[b]	0.792		1 ± 5	
	Control	30 ± 3	31 ± 4[b]	0.883		1 ± 4	
Medium LDL particles (%)	Combination	37 ± 2	38 ± 2	0.845	0.301	1 ± 3	0.817
	ADF	37 ± 1	35 ± 1	0.288		−2 ± 2	
	Exercise	41 ± 3	40 ± 3	0.453		−1 ± 2	
	Control	41 ± 2	40 ± 2	0.717		−1 ± 2	
Small LDL particles (%)	Combination	25 ± 3	18 ± 3[a]	0.010		−7 ± 2[a]	0.007
	ADF	27 ± 3	15 ± 3[a]	<0.001	0.023	−12 ± 3[a]	
	Exercise	21 ± 3	20 ± 4[b]	0.972		−1 ± 4[b]	
	Control	29 ± 3	30 ± 3[b]	0.776		1 ± 3[b]	

International Journal of
EXERCISE SCIENCE

Original Research

Effects of Prior Fasting on Fat Oxidation during Resistance Exercise

KENDALL FRAWLEY*, GABRIELLE GREENWALD*, REBECCA R. ROGERS‡, JOHN K. PETRELLA‡, and MALLORY R. MARSHALL‡

Department of Kinesiology, Samford University, Birmingham, AL, USA

- NCAA women
- 10 hours fasted vs fed
- Respiratory exchange ratio was lower in the fasted group implying more fat metabolism than carb metabolism.

Int J Exerc Sci 11(2): 827-833, 2018

EPOC, not

- Excess post-exercise oxygen consumption-after burn!
- This is the Holy Grail of weight loss.
- Can it be done?
- HIIT
- Resistance training

Relation of Muscle Mass and Fat Mass to Cardiovascular Disease Mortality

Preethi Srikanthan, MD, MS[a,*], Tamara B. Horwich, MD, MS[b], and Chi Hong Tseng, PhD[c]

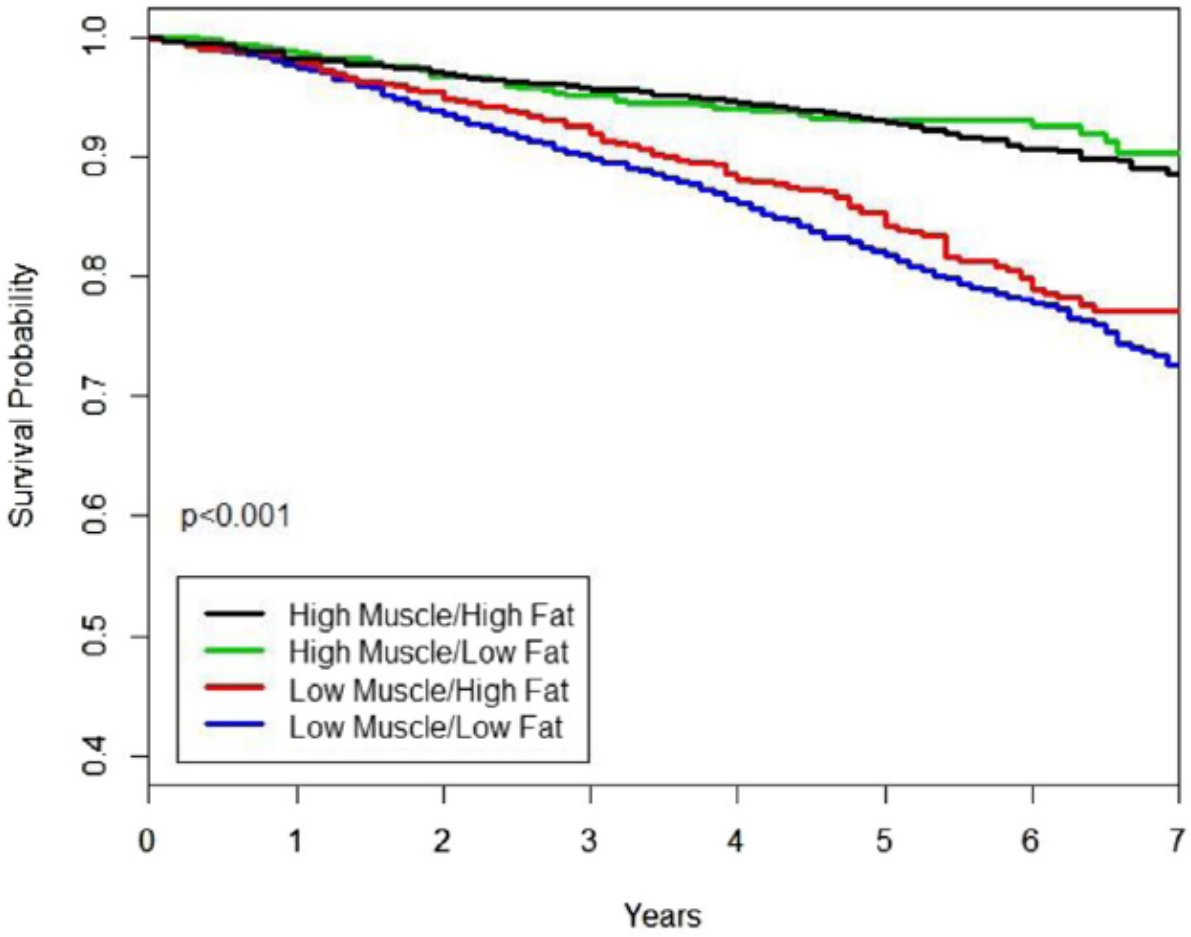


Figure 2. Kaplan-Meier plot of all-cause mortality for the 4 body composition types based on AMMI and TRFI.

(Am J Cardiol 2016;117:1355–1360)

Brown fat vs white fat…

It’s not just burning fat, but changing it. You can brown your white fat!

The Hunger Games…

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Long-Term Persistence of Hormonal Adaptations to Weight Loss

Priya Sumithran, M.B., B.S., Luke A. Prendergast, Ph.D.,
Elizabeth Delbridge, Ph.D., Katrina Purcell, B.Sc., Arthur Shulkes, Sc.D.,
Adamandia Kriketos, Ph.D., and Joseph Proietto, M.B., B.S., Ph.D.

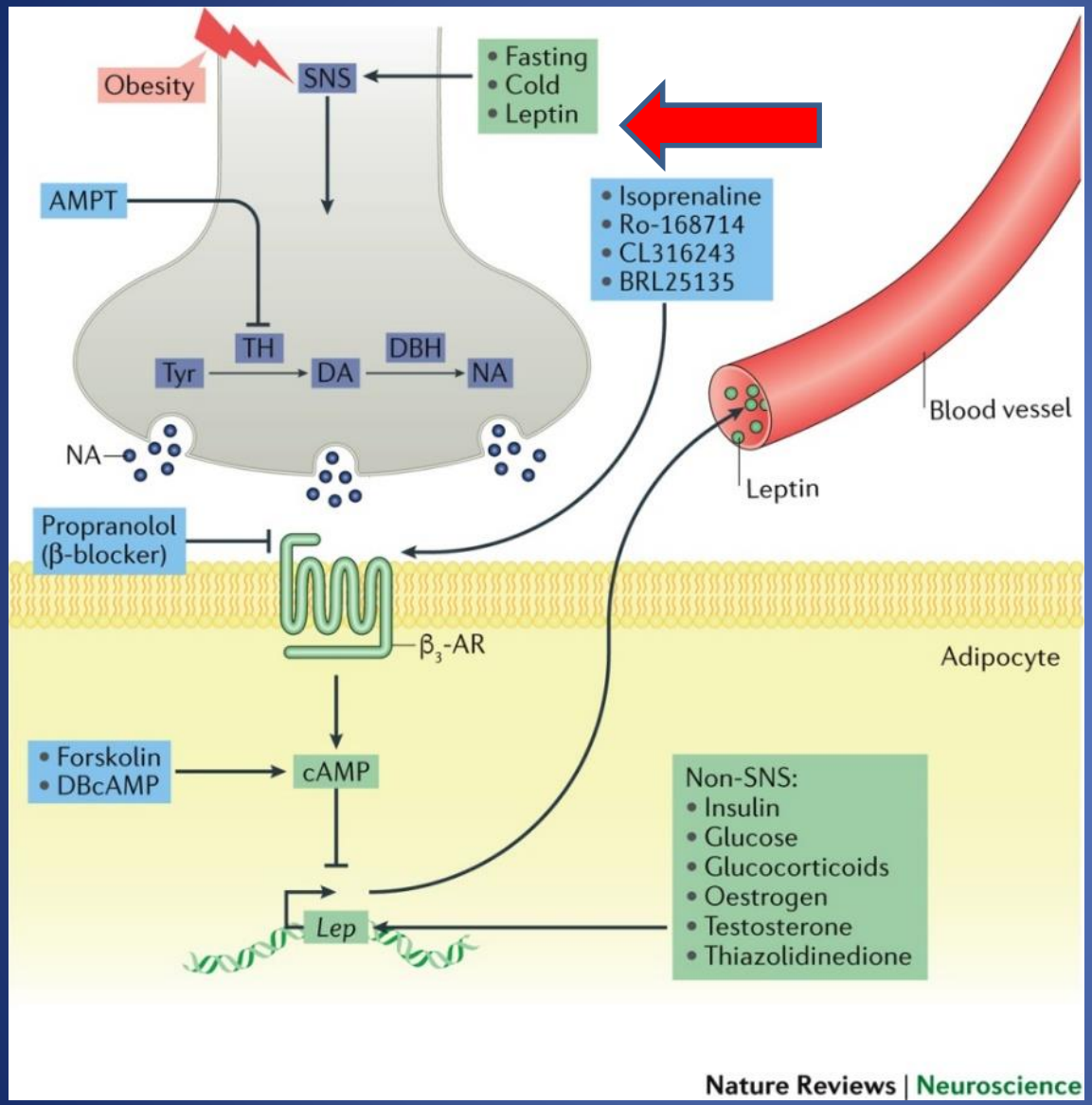
Leptin and brain–adipose crosstalks

Alexandre Caron, Syann Lee, Joel K. Elmquist and Laurent Gautron**

Abstract | Interactions between the brain and distinct adipose depots have a key role in maintaining energy balance, thereby promoting survival in response to metabolic challenges such as cold exposure and starvation. Recently, there has been renewed interest in the specific central neuronal circuits that regulate adipose depots. Here, we review anatomical, genetic and pharmacological studies on the neural regulation of adipose function, including lipolysis, non-shivering thermogenesis, browning and leptin secretion. In particular, we emphasize the role of leptin-sensitive neurons and the sympathetic nervous system in modulating the activity of brown, white and beige adipose tissues. We provide an overview of advances in the understanding of the heterogeneity of the brain regulation of adipose tissues and offer a perspective on the challenges and paradoxes that the community is facing regarding the actions of leptin on this system.

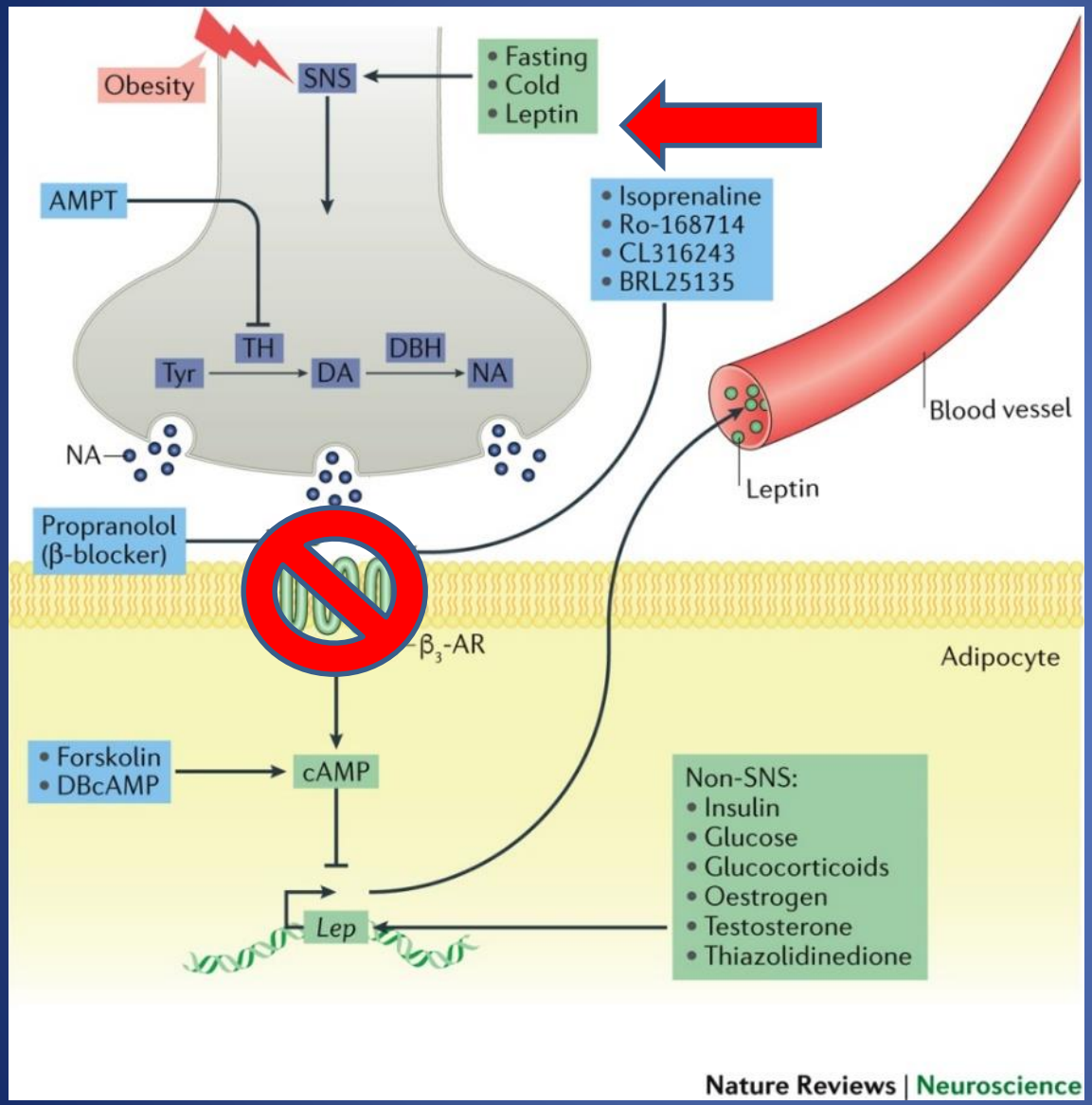
Division of Hypothalamic Research and Department of Internal Medicine, The University of Texas Southwestern Medical Center, Dallas, TX, USA.
**e-mail: laurent.gautron@utsouthwestern.edu; joel.elmquist@utsouthwestern.edu*

doi:10.1038/nrn.2018.7
Published online 16 Feb 2018



Leptin

- Anti-hunger hormone.
- NOT an acute reactant.
- Works on a ***chronic*** basis.
- You get less hungry over time!



Leptin

- Why do b-blockers impede weight loss?
- Decreased HR
- Blocks a signaling pathway for leptin production.

MECHANISMS IN ENDOCRINOLOGY

Brown adipose tissue in humans: regulation and metabolic significance

Moe Thuzar[1,2] and **Ken K Y Ho**[1,2]

[1]Department of Endocrinology and Diabetes, Princess Alexandra Hospital, Brisbane, Queensland, Australia and
[2]School of Medicine, University of Queensland, Brisbane, Queensland 4102, Australia

Correspondence should be addressed to K K Y Ho
Email
k.ho@uq.edu.au

Table 1 Summary of studies reporting detected amount of BAT and associated CIT after acute cold stimulation in adult humans.

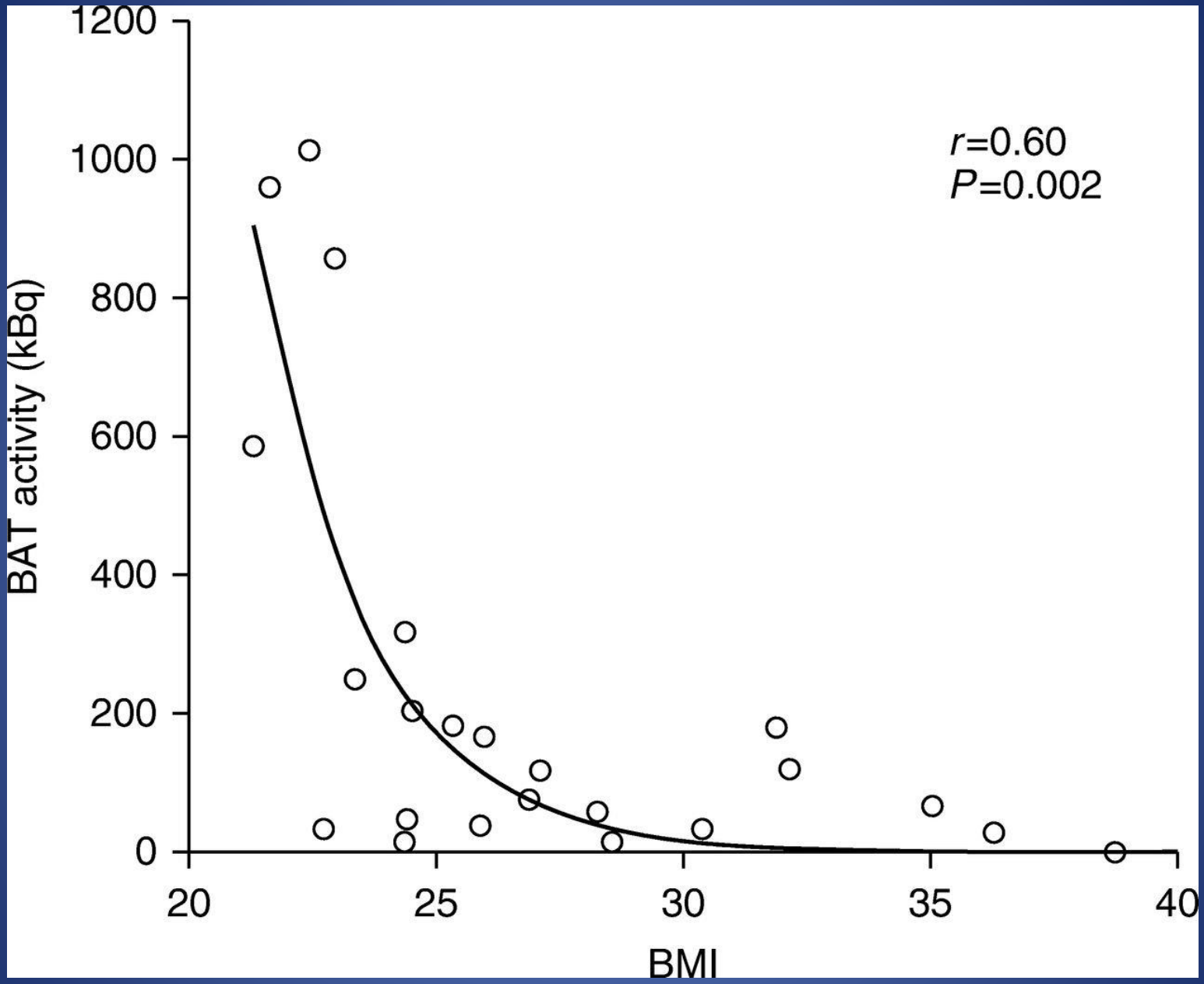
References	Number of subjects	Cooling temperature (°C)	BAT amount	CIT (kcal/day)
(33)	6	18	Mean 168 ml	Mean 2000
(34)	9 with high BAT	15	Mean 59.1 g	Mean 237
	15 with low BAT		Mean 2.2 g	Mean 39
(35)	19 BAT positive	17 ± 1	Mean 34 g	Mean 287[a]
	8 BAT negative			Mean 167[a]
(38)	24	19	Mean 63 ml	Mean 88
(39)	10	14	Median 15 ml	Mean 79
(153)	6 BAT positive	19	No data	Mean 410
	7 BAT negative			Mean 42

[a]Converted from Megajoule using 1 MJ = 239 kcal, normalised for fat free mass.

Table 2 Summary of cold acclimatisation studies addressing the role of BAT in adiposity and substrate metabolism in humans.

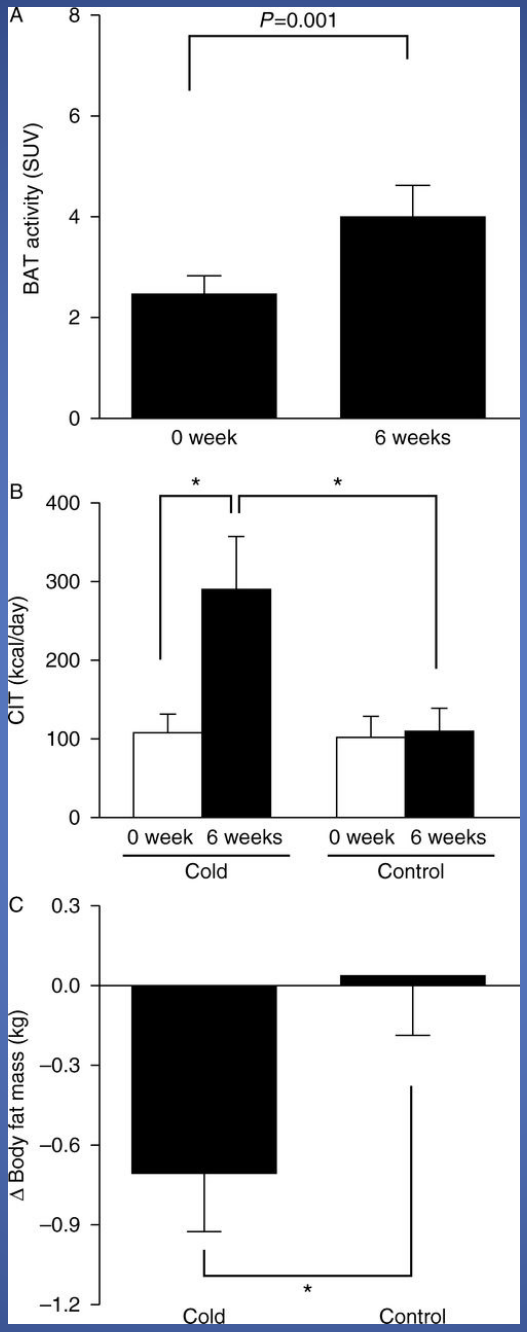
References	Number of subjects	Acclimatisation protocol	ΔBAT activity from baseline	ΔMetabolism from baseline
(24)	22 randomised to cold intervention or usual living (control)	17 °C, 2 h/day for 6 weeks	58% increase (no significant change in the control group)	5.2% or 0.7 kg decrease in body fat mass (no significant change in the control group)
(21)	6	10 °C, 2 h/day for 4 weeks	45% increase	6.2% decrease in plasma glucose
(22)	5	19 °C overnight for 1 month	54% increase	>50% increase in insulin sensitivity
(37)	10	14–15 °C for 10 days	~50% increase	43% increase in insulin sensitivity

Figure 1



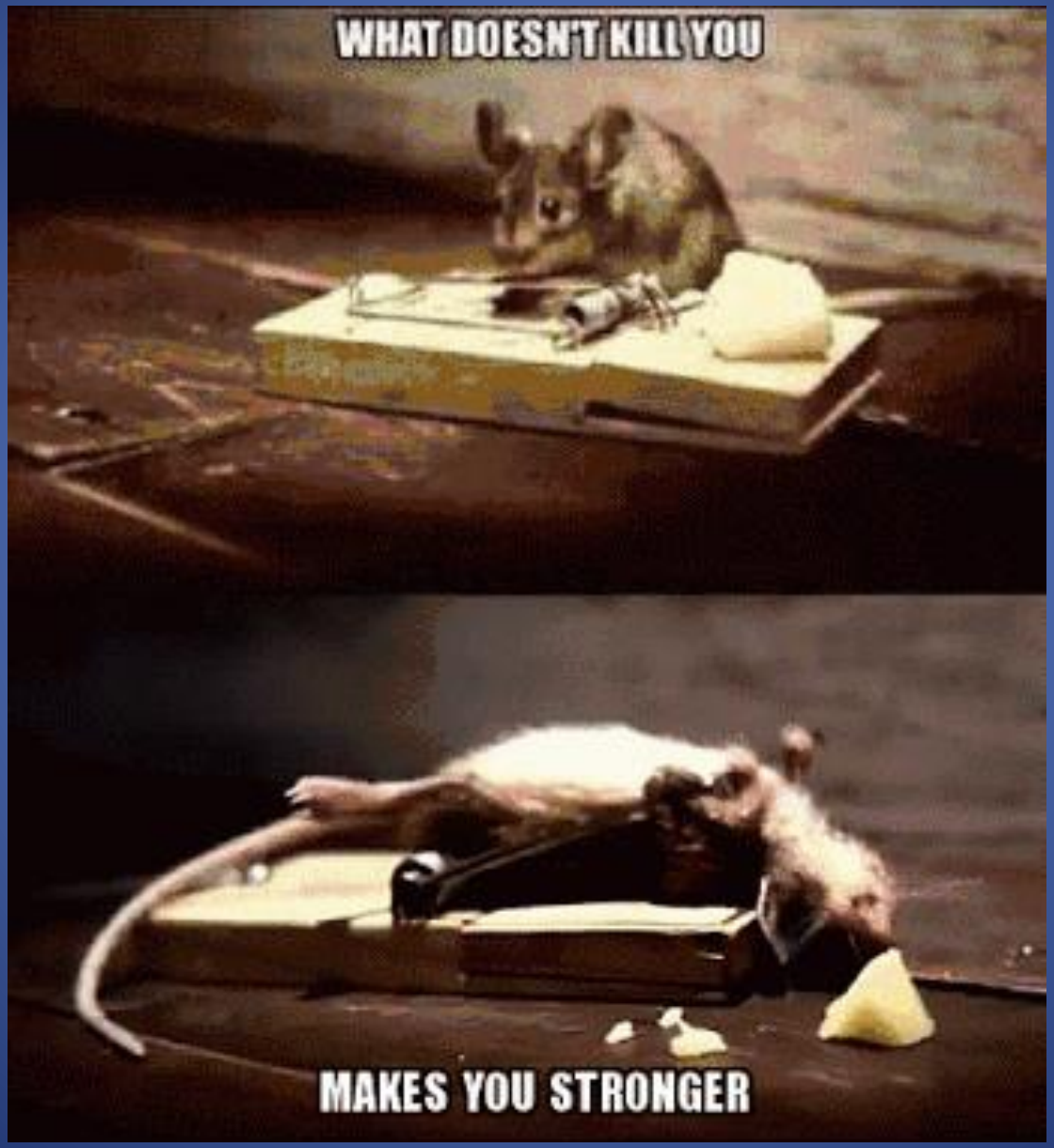
Relationship between BMI and BAT activity measured on FDG–PET–CT scans after acute cold exposure (Reproduced with permission from van Marken Lichtenbelt WD, Vanhommerig JW, Smulders NM, Drossaerts JM, Kemerink GJ, Bouvy ND, Schrauwen P & Teule GJ. Cold-activated brown adipose tissue in healthy men. New England Journal of Medicine 2009 360 1500–1508).

Figure 2



Effect of chronic cold exposure on BAT activity, CIT and body fat mass in humans. (A) BAT activity plotted in standardised uptake value (SUV), measured on FDG–PET–CT scans before and after 6 weeks of daily cold exposure. (B) CIT expressed in kcal/day in subjects who underwent the cold intervention (cold group) and those who continued their usual daily living (control group). (C) Change in body fat mass from baseline after 6 weeks in the cold group and control group. Expressed in mean±s.e.m.; *P<0.05 (Adapted with permission from (24)).

Stress leads to growth…



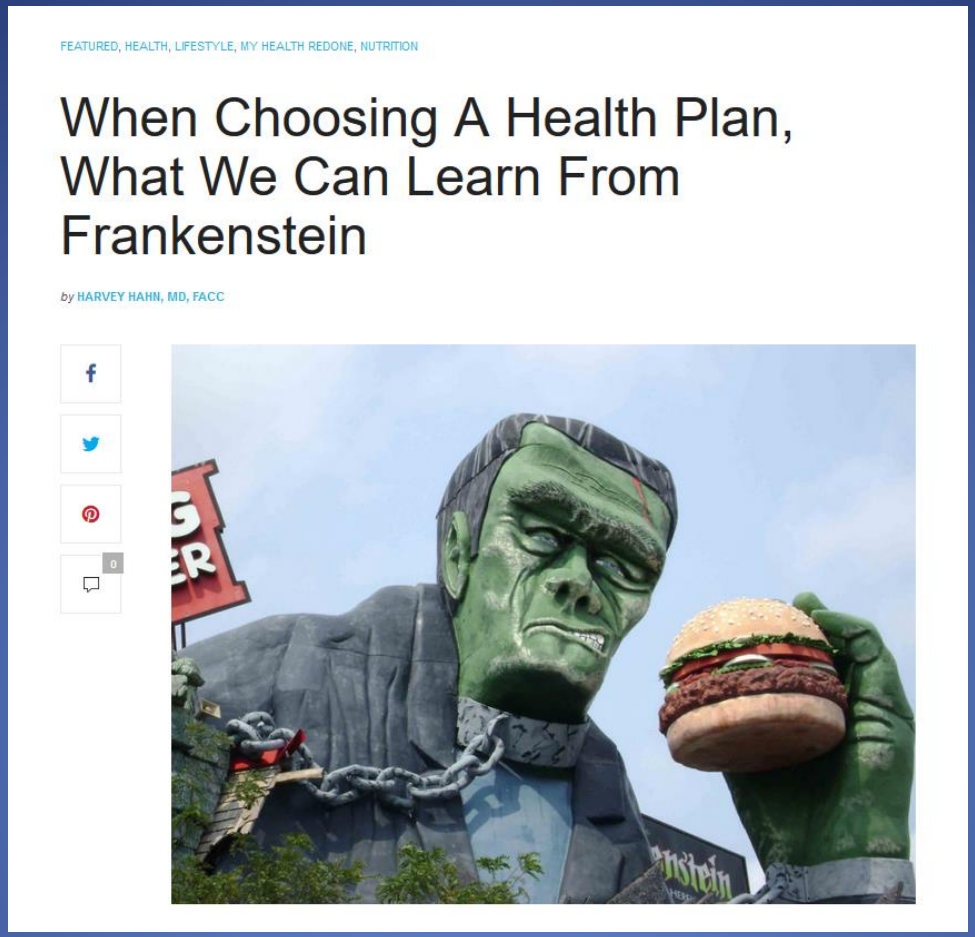
Stimulus response chart

Stimulus	Sign / symptom	Minimal dose	Overdose
Calorie restriction	?	10%	Starvation
Intermittent fasting	Hunger	8 hour fast, 5 days/month	Starvation
Exercise	Tachy, DOE, sweat, uncomfortable	10-15 mins continuous	Overtraining syndrome, cortisol, injury
Cold exposure	Shiver, goosebumps	10 min	Frostbite
Sleep	None, should be asleep.	6 hrs continuous	Coma or death…

Live long and prosper!

and *FAST!*

Back up slides



- LifeandHealth.org
- https://lifeandhealth.org/slider/when-choosing-a-health-plan-what-we-can-learn-from-frankenstein/2317658.html

Summary

	Low-carbohydrate	Low-fat/ vegetarian/vegan	Low-glycemic	Mediterranean	Mixed/balanced	Paleolithic
Health benefits relate to:	Emphasis on restriction of refined starches and added sugars in particular.	Emphasis on plant foods direct from nature; avoidance of harmful fats.	Restriction of starches, added sugars; high fiber intake.	Foods direct from nature; mostly plants; emphasis on healthful oils, notably monounsaturates.	Minimization of highly processed, energy-dense foods; emphasis on wholesome foods in moderate quantities.	Minimization of processed foods. Emphasis on natural plant foods and lean meats.
Compatible elements:	Limited refined starches, added sugars, processed foods; limited intake of certain fats; emphasis on whole plant foods, with or without lean meats, fish, poultry, seafood.					
And all potentially consistent with:	**Food, not too much, mostly plants[a,b,c].**					

[a]From Reference 135.
[b]Portion control may be facilitated by choosing better-quality foods which have the tendency to promote satiety with fewer calories.
[c]While neither the low-carbohydrate nor Paleolithic diet need be "mostly plants," both can be.

Katz DL, Meller S. 2014.
Annu. Rev. Public Health. 35:83–103

- Everyone is different.
- Sustainability is key, not a diet…
- Enjoy life, don't torture yourself,
- or others for that matter…

Higher compared with lower dietary protein during an energy deficit combined with intense exercise promotes greater lean mass gain and fat mass loss: a randomized trial[1,2]

*Thomas M Longland, Sara Y Oikawa, Cameron J Mitchell, Michaela C Devries, and Stuart M Phillips**

Department of Kinesiology, Exercise Metabolism Research Group, McMaster University, Hamilton, Canada

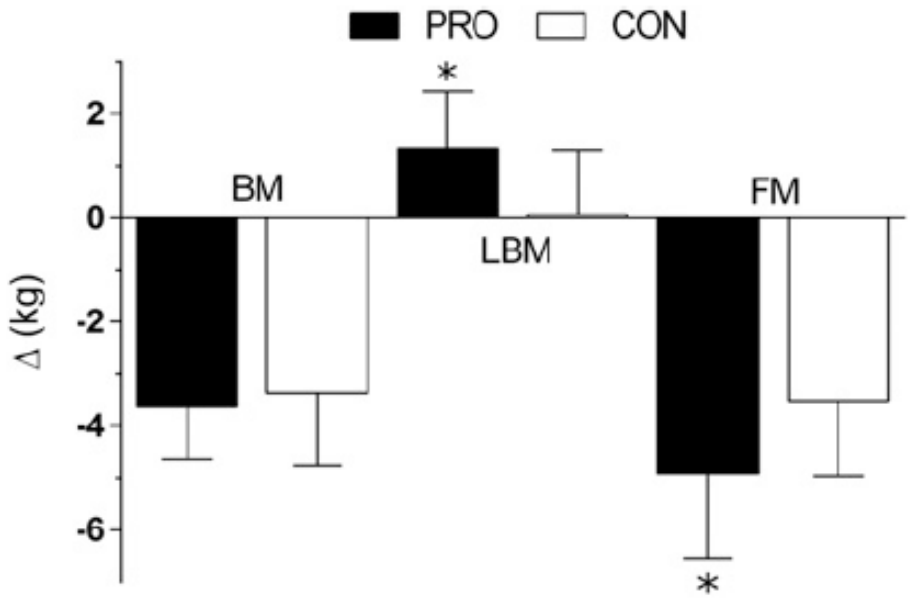


FIGURE 2 Four-compartment model-derived changes in BM, LBM, and FM during the intervention in both PRO and CON groups; data were analyzed with the use of an unpaired *t* test. Values are means ± SDs; $n = 40$ (20/group). *Significantly different from CON ($P < 0.05$). BM, body mass; CON, lower-protein (1.2 g · kg^{-1} · d^{-1}) control diet; FM, fat mass; LBM, lean body mass; PRO, higher-protein (2.4 g · kg^{-1} · d^{-1}) diet.

Am J Clin Nutr doi: 10.3945/ajcn.115.119339.

The Importance of Breakfast in Atherosclerosis Disease

Insights From the PESA Study

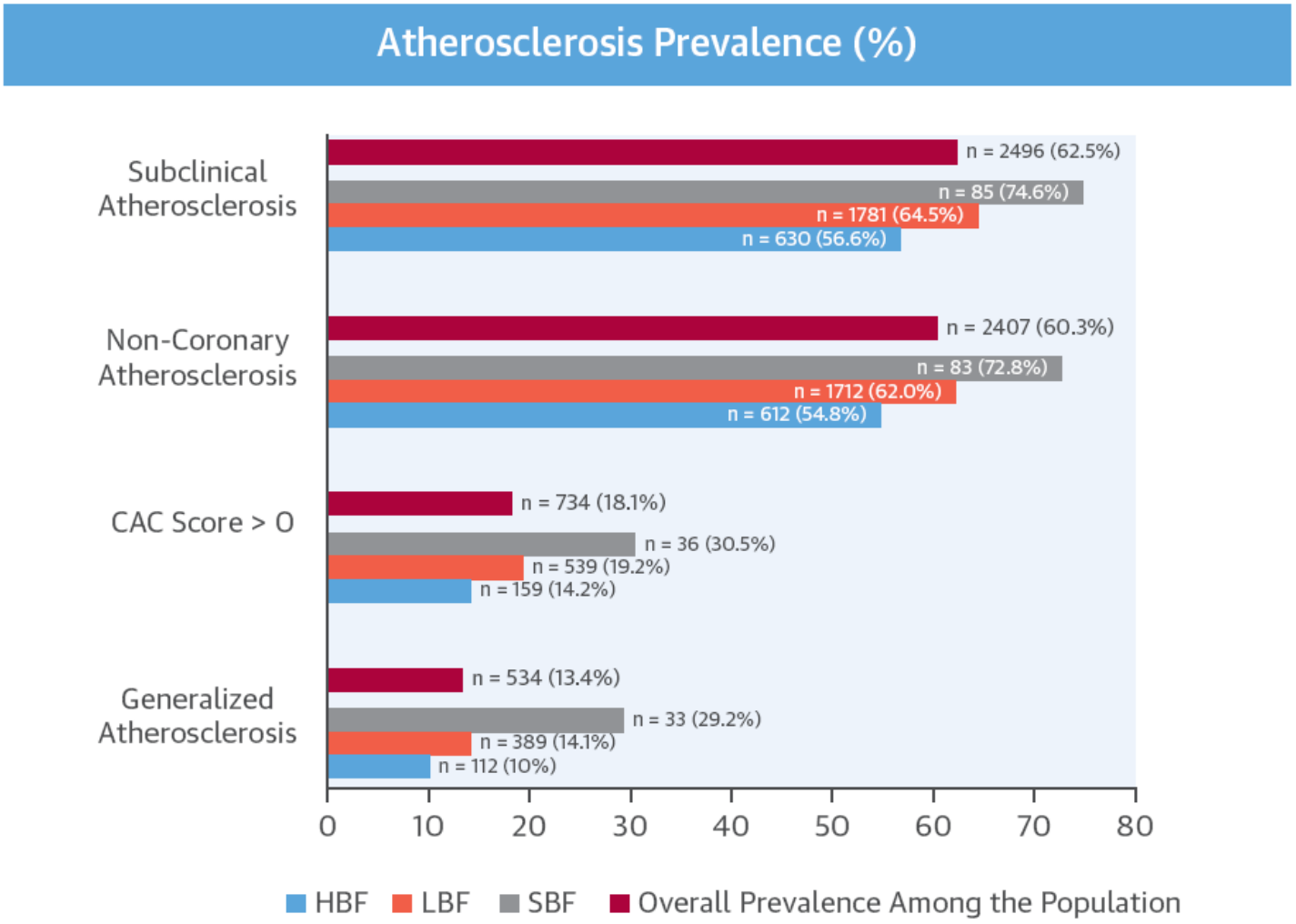
Irina Uzhova, MSc,[a] Valentín Fuster, MD, PhD,[a,b] Antonio Fernández-Ortiz, MD, PhD,[a,c,d,e] José M. Ordovás, PhD,[a,f,g] Javier Sanz, MD,[a,b] Leticia Fernández-Friera, MD, PhD,[a,c,h] Beatriz López-Melgar, MD, PhD,[a,h] José M. Mendiguren, MD,[i] Borja Ibáñez, MD, PhD,[a,c,j] Héctor Bueno, MD, PhD,[a,d,k] José L. Peñalvo, PhD[l]

JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY

PUBLISHED BY ELSEVIER

VOL. 70, NO. 15, 2017
ISSN 0735-1097/$36.00
http://dx.doi.org/10.1016/j.jacc.2017.08.027

CENTRAL ILLUSTRATION **Overall Prevalence of an Atherosclerosis Among PESA Study Participants and According to the Type of Breakfast Consumed**



Uzhova, I. et al. J Am Coll Cardiol. 2017;70(15):1833–42.

The prevalence of an atherosclerosis is presented for total population, as well as by breakfast habits categories. The SBF group presents the highest proportion of individuals with subclinical, noncoronary, generalized atherosclerosis and increased coronary artery calcium score. CACs = coronary artery calcium score; HBF = high-energy breakfast; LBF = low-energy breakfast; PESA = Progression of Early Subclinical Atherosclerosis; SBF = skipping breakfast.

TABLE 2 Overall Dietary Profile of PESA Study Participants According to Breakfast Pattern

	HBF (n = 1,122)	LBF (n = 2,812)	SBF (n = 118)
Macronutrients, g/day or mg/day			
Energy intake, kcal	2,234 ± 450*†	2,345 ± 467‡	2,358 ± 562‡
Total protein	94.3 ± 18.0*†	102.4 ± 20.0‡	105.7 ± 24.0‡
Animal protein	64.8 ± 15.0*†	72.1 ± 17.1†‡	76.6 ± 20.7*‡
Vegetable protein	29.08 ± 8.23*	29.84 ± 8.39‡	28.69 ± 9.83
Total fat	103.1 ± 22.9*†	108.3 ± 24.2‡	113.6 ± 30.6‡
Cholesterol	334.4 ± 98.2*†	361.6 ± 94.8†‡	385.7 ± 111.0*‡
MUFA	47.0 ± 11.6*†	49.3 ± 11.5†‡	52.4 ± 13.8*‡
PUFA	16.62 ± 5.09*†	17.81 ± 5.48†‡	19.05 ± 7.06*‡
SFA	29.98 ± 8.62*†	32.05 ± 9.00‡	32.84 ± 10.90‡
Carbohydrates	218.5 ± 58.1†	220.0 ± 58.8†	197.0 ± 63.8*‡
Sugar	94.0 ± 31.8*†	90.9 ± 30.6†‡	75.5 ± 34.4*‡
Polysaccharides	119.7 ± 40.6*	125.7 ± 43.2‡	119.2 ± 46.0
Fiber	21.08 ± 6.48†	20.90 ± 5.99†	18.99 ± 6.19*‡
Food group, g/day			
Fruits and vegetables	474 ± 210*†	435 ± 202†‡	369 ± 182*‡
Dried fruits	7.30 ± 10.76	7.94 ± 12.26	9.65 ± 16.64
Legumes	25.2 ± 21.2	26.0 ± 22.9	27.4 ± 23.3
Potatoes	20.0 ± 17.1	21.1 ± 17.7	19.3 ± 16.7
Refined grains	216.0 ± 92.8*	234.0 ± 98.7‡	231.0 ± 101.5
Whole grains	14.3 ± 31.9*†	9.1 ± 21.6†‡	2.5 ± 10.6*‡
Nuts	5.03 ± 5.92	5.41 ± 5.68	5.16 ± 4.91
Olives	4.05 ± 6.30*†	4.65 ± 6.52†‡	7.26 ± 15.13*‡
Red meat	93.0 ± 42.2*†	112.9 ± 50.1†‡	145.1 ± 68.6*‡
Lean meat	63.3 ± 30.7*	66.9 ± 33.5‡	67.7 ± 32.6
Seafood (fish, shellfish)	75.8 ± 36.2*	79.1 ± 38.9‡	78.1 ± 39.9
Dairy	207 ± 151†	196 ± 137†	141 ± 116*‡
Low-fat dairy	88.3 ± 125.8	90.0 ± 121.8†	61.4 ± 112.7*
Vegetable oil and fat	5.02 ± 5.92	5.40 ± 5.68	5.15 ± 4.91
Butter	5.89 ± 6.72*†	4.26 ± 4.39†‡	2.30 ± 2.03*‡
Olive oil	31.7 ± 14.1*	29.9 ± 12.2‡	31.0 ± 11.8
Precooked meals, fast food	55.6 ± 34.0*†	66.9 ± 42.1‡	68.6 ± 35.6‡
Chips and snacks	5.02 ± 7.06*†	6.49 ± 9.12†‡	8.69 ± 11.73*‡
Commercial bakery	71.4 ± 50.0†	69.6 ± 47.8†	54.3 ± 47.5*‡
Alcohol (distilled spirits, wine, beer)	122 ± 144*†	190 ± 227†‡	299 ± 328*‡
SSB	132 ± 184*†	157 ± 204†‡	256 ± 439*‡
Tea, coffee	167 ± 131†	174 ± 128	202 ± 193‡
Dietary quality			
Mediterranean cluster	533 (47.5)*†	1,052 (37.4)†‡	30 (25.4)*‡
Western cluster	485 (43.2)†	1,148 (40.8)†	35 (29.7)*‡
Social business cluster	104 (9.3)*†	612 (21.8)†‡	53 (44.9)*‡

Values are mean ± SD or n (%). Bonferroni correction was applied for categorical variables ($p < 0.017$). *$p < 0.05$ vs. LBF. †$p < 0.05$ vs. SBF. ‡$p < 0.05$ vs. HBF.

MUFA = monounsaturated fatty acids; PESA = Progression of Early Subclinical Atherosclerosis; PUFA = polyunsaturated fatty acids; SFA = saturated fatty acids; SSB = sugar-sweetened beverages; other abbreviations as in Table 1.

TABLE 2 Overall Dietary Profile of PESA Study Participants According to Breakfast Pattern

	HBF (n = 1,122)	LBF (n = 2,812)	SBF (n = 118)
Dietary quality			
Mediterranean cluster	533 (47.5)*†	1,052 (37.4)†‡	30 (25.4)*‡
Western cluster	485 (43.2)†	1,148 (40.8)†	35 (29.7)*‡
Social business cluster	104 (9.3)*†	612 (21.8)†‡	53 (44.9)*‡

Dose–Response Relation Between Work Hours and Cardiovascular Disease Risk

Findings From the Panel Study of Income Dynamics

Sadie H. Conway, PhD, Lisa A. Pompeii, PhD, Robert E. Roberts, PhD, Jack L. Follis, PhD, and David Gimeno, PhD

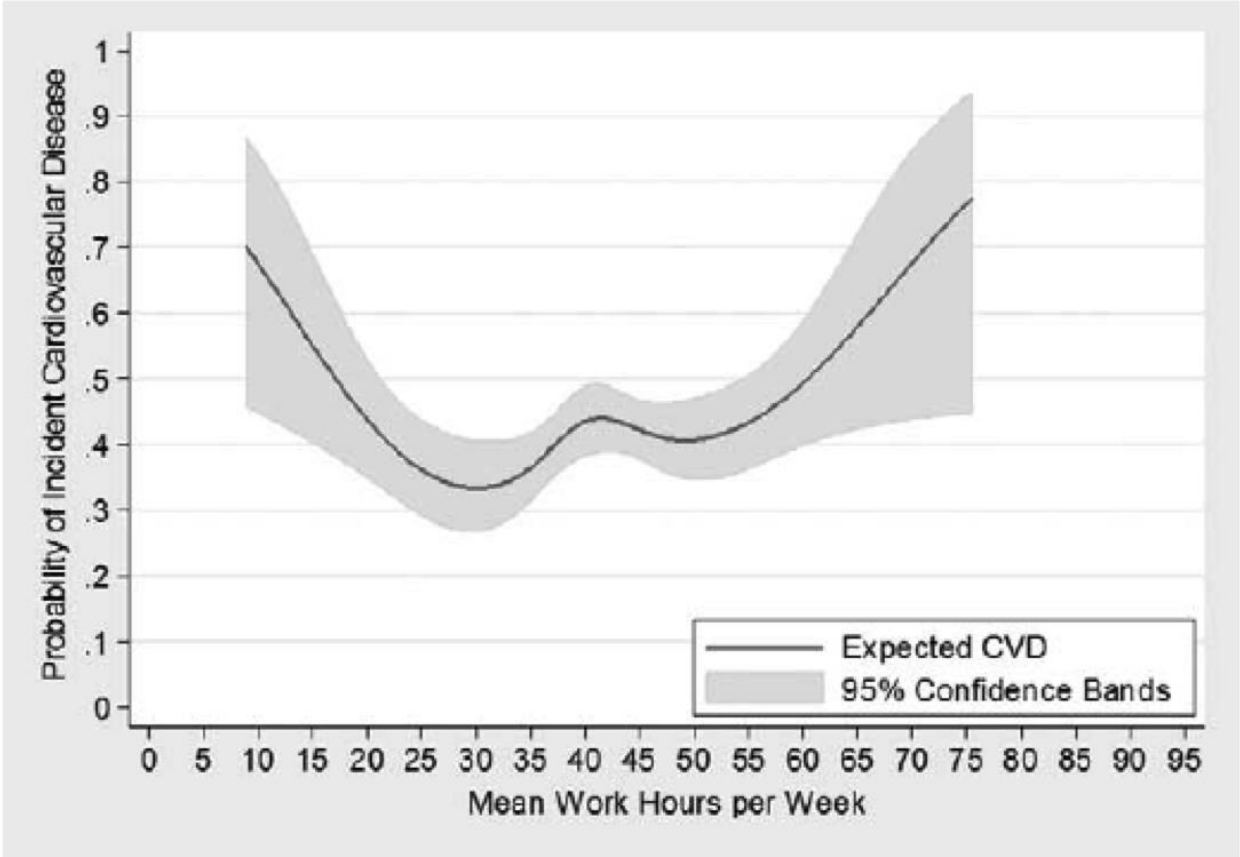
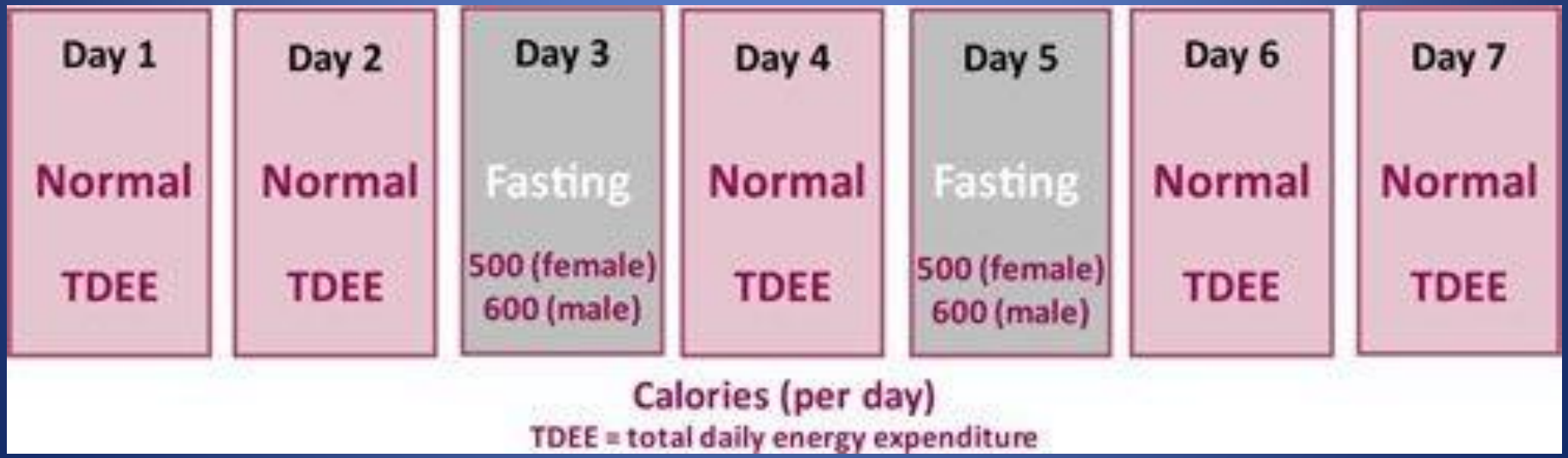


FIGURE 1. Restricted cubic spline model for the relationship between long work hours and incident cardiovascular disease: Panel Study of Income Dynamics, 1986 to 2011.

A.D.F. or 5:2 plan

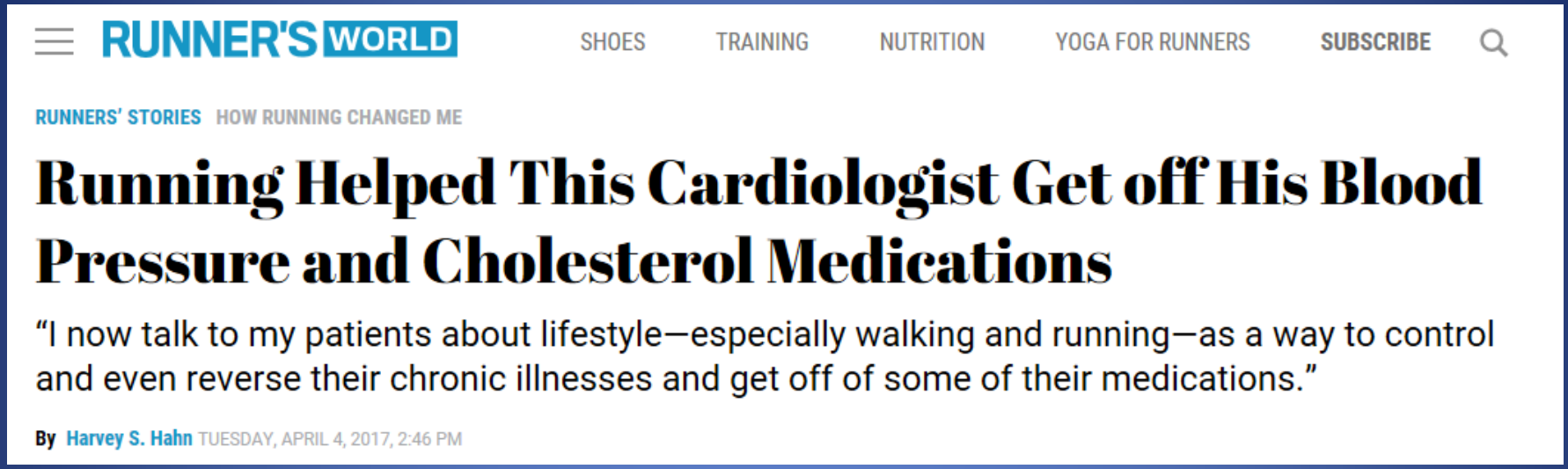
- Fasting may help prevent dementia by causing a low level stress that stimulates brain stem cell activation!



Case study:

- L-knee pain. MRI-torn meniscus. Scope.
- Gained 40 lb since HS.
- Wife, 2 kids, busy job…
- “95%” vegan, portion control, exercise...
- Lost 45 lbs. 5 inches off of waist. % body fat went from 28% to 16%. LDL 143 to 71 (off of lipitor). Got off BP med. Feeling great!
- This is me. Lifestyle works!

My personal choice!



RUNNER'S WORLD

SHOES TRAINING NUTRITION YOGA FOR RUNNERS SUBSCRIBE

RUNNERS' STORIES HOW RUNNING CHANGED ME

Running Helped This Cardiologist Get off His Blood Pressure and Cholesterol Medications

"I now talk to my patients about lifestyle—especially walking and running—as a way to control and even reverse their chronic illnesses and get off of some of their medications."

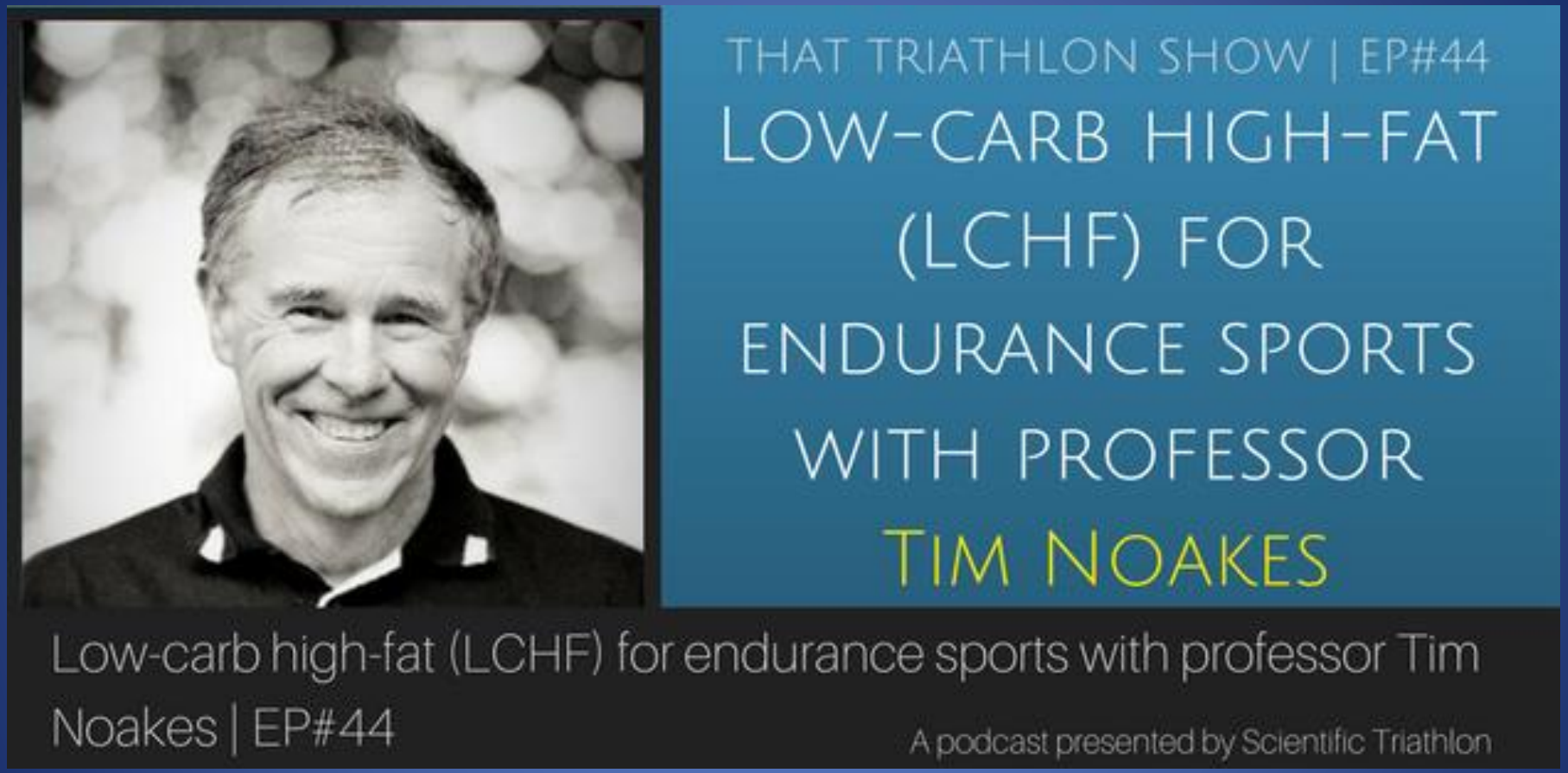
By Harvey S. Hahn TUESDAY, APRIL 4, 2017, 2:46 PM

My personal choice!

Summary for long life…

- Restrict calorie intake.
- Consider IF.
- Exercise.
- Sleep 6+ hours a night.
- Get some cold exposure.
- Live long and prosper!

Ketogenics



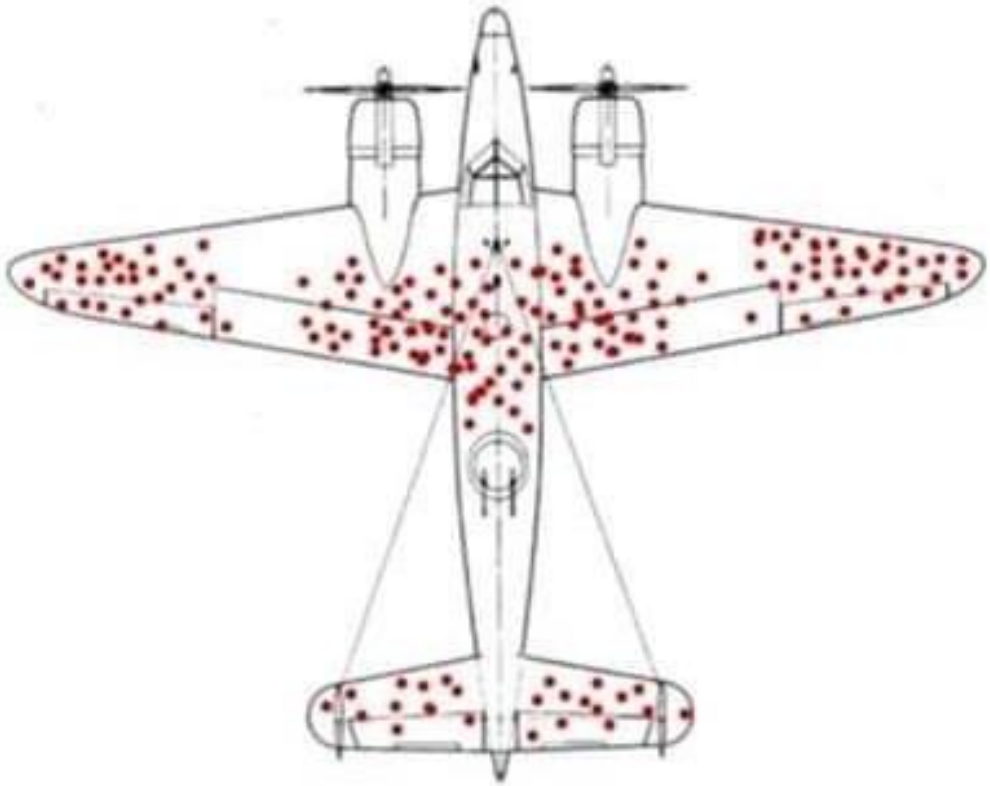
https://www.medscape.com/viewarticle/874707_1

Low carb, < 50 gm/day.

Keto-off target?

Abraham Wald

This is a picture tracking bullet holes on Allied planes that encountered Nazi anti-aircraft fire in WW2.



At first, the military wanted to reinforce those areas, because obviously that's where the ground crews observed the most damage on returning planes. Until Hungarian-born Jewish mathematician Abraham Wald pointed out that this was the damage on the planes that *made it home*, and the Allies should armor the areas where there are no dots at all, because those are the places where the planes won't survive when hit. This phenomenon is called survivorship bias, a logic error where you focus on things that survived when you should really be looking at things that didn't.

We have higher rates of mental illness now? Maybe that's because we've stopped killing people for being "possessed" or "witches." Higher rate of allergies? Anaphylaxis kills, and does so *really fast* if you don't know what's happening. Higher claims of rape? Maybe victims are less afraid of coming forward. These problems were all happening before, but now we've reinforced the medical and social structures needed to help these people survive. And we still have a long way to go.

Source: marzipanandminutiae

Med Sci Sports Exerc. 2019 Oct;51(10):2135-2146. doi: 10.1249/MSS.0000000000002008.

Effect of a Ketogenic Diet on Submaximal Exercise Capacity and Efficiency in Runners.

Shaw DM[1], Merien F[2], Braakhuis A[3], Maunder ED[1], Dulson DK[1].

Author information

Abstract

PURPOSE: We investigated the effect of a 31-d ketogenic diet (KD) on submaximal exercise capacity and efficiency.

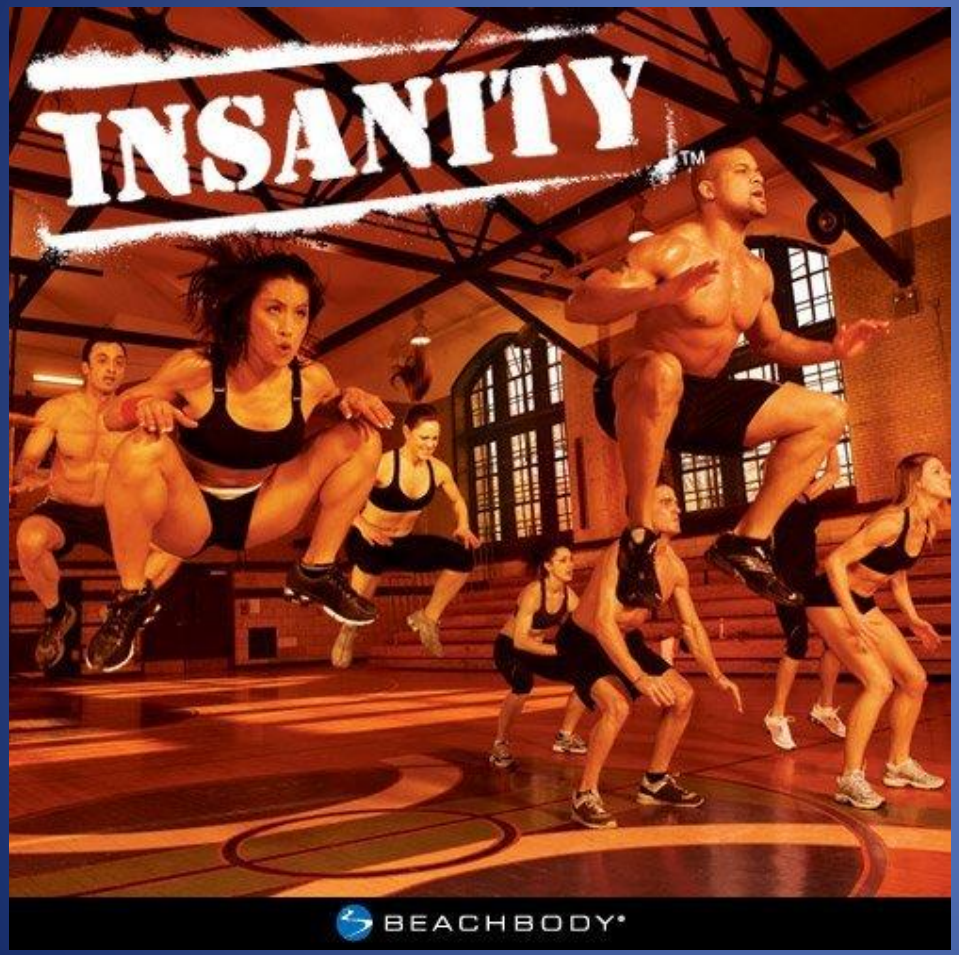
METHODS: A randomized, repeated-measures, crossover study was conducted in eight trained male endurance athletes (V˙O2max, 59.4 ± 5.2 mL·kg·min). Participants ingested their habitual diet (HD) (13.1 MJ, 43% [4.6 g·kg·d] carbohydrate and 38% [1.8 g·kg·d] fat) or an isoenergetic KD (13.7 MJ, 4% [0.5 g·kg·d] carbohydrate and 78% [4 g·kg·d] fat) from days 0 to 31 ($P < 0.001$). Participants performed a fasted metabolic test on days -2 and 29 (~25 min) and a run-to-exhaustion trial at 70% V˙O2max on days 0 and 31 following the ingestion of a high-carbohydrate meal (2 g·kg) or an isoenergetic low-carbohydrate, high-fat meal (<10 g CHO), with carbohydrate (~55 g·h) or isoenergetic fat (0 g CHO·h) supplementation during exercise.

RESULTS: Training loads were similar between trials and V˙O2max was unchanged (all, $P > 0.05$). The KD impaired exercise efficiency, particularly at >70% V˙O2max, as evidenced by increased energy expenditure and oxygen uptake that could not be explained by shifts in respiratory exchange ratio (RER) (all, $P < 0.05$). However, exercise efficiency was maintained on a KD when exercising at <60% V˙O2max (all, $P > 0.05$). Time-to-exhaustion (TTE) was similar for each dietary adaptation (pre-HD, 237 ± 44 vs post-HD, 231 ± 35 min; $P = 0.44$ and pre-KD, 239 ± 27 vs post-KD, 219 ± 53 min; $P = 0.36$). Following keto-adaptation, RER >1.0 vs <1.0 at V˙O2max coincided with the preservation and reduction in TTE, respectively.

CONCLUSION: A 31-d KD preserved mean submaximal exercise capacity in trained endurance athletes without necessitating acute carbohydrate fuelling strategies. However, there was a greater risk of an endurance decrement at an individual level.

PMID: 31033901 DOI: 10.1249/MSS.0000000000002008

Insanity…

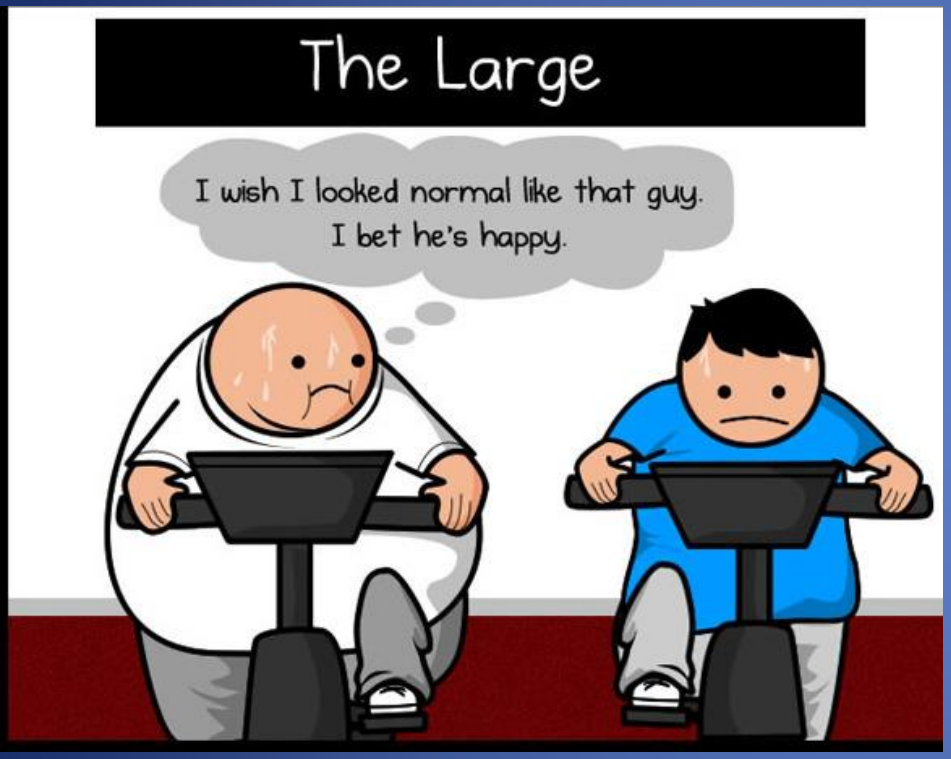


- Doing the same thing, BUT expecting different results…

Post-run recovery…

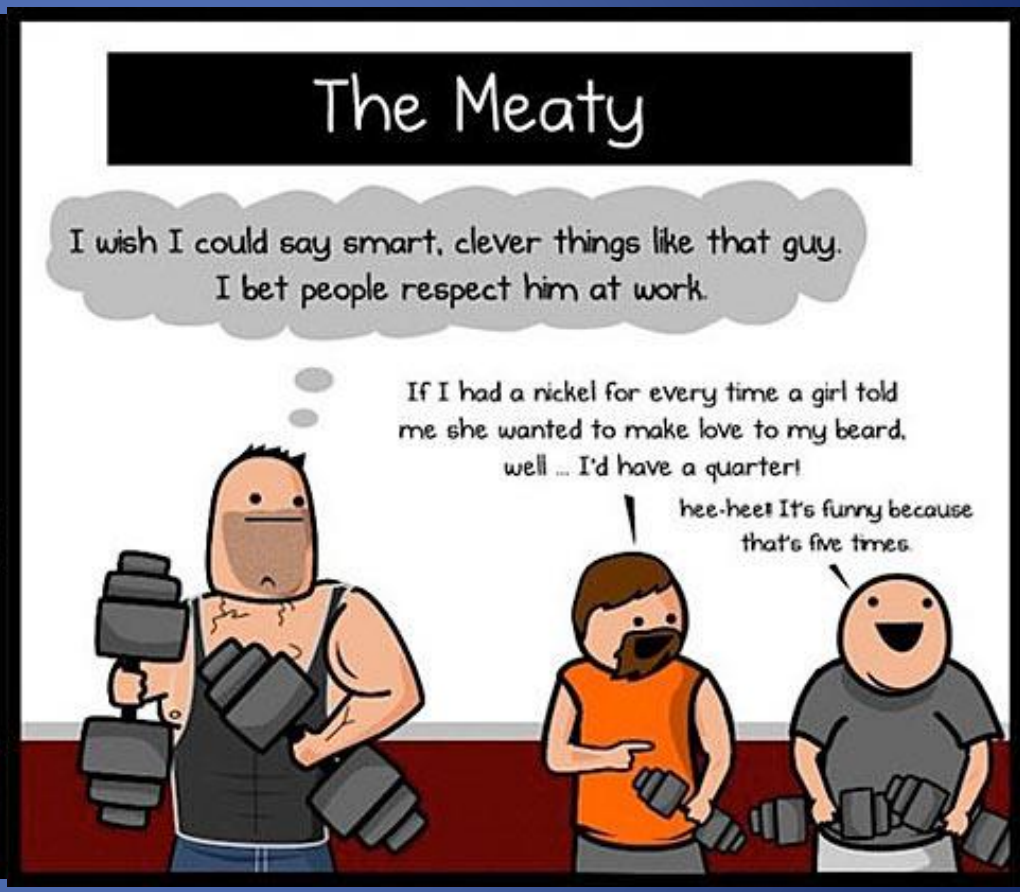
Pick your morphology.

No one is happy…

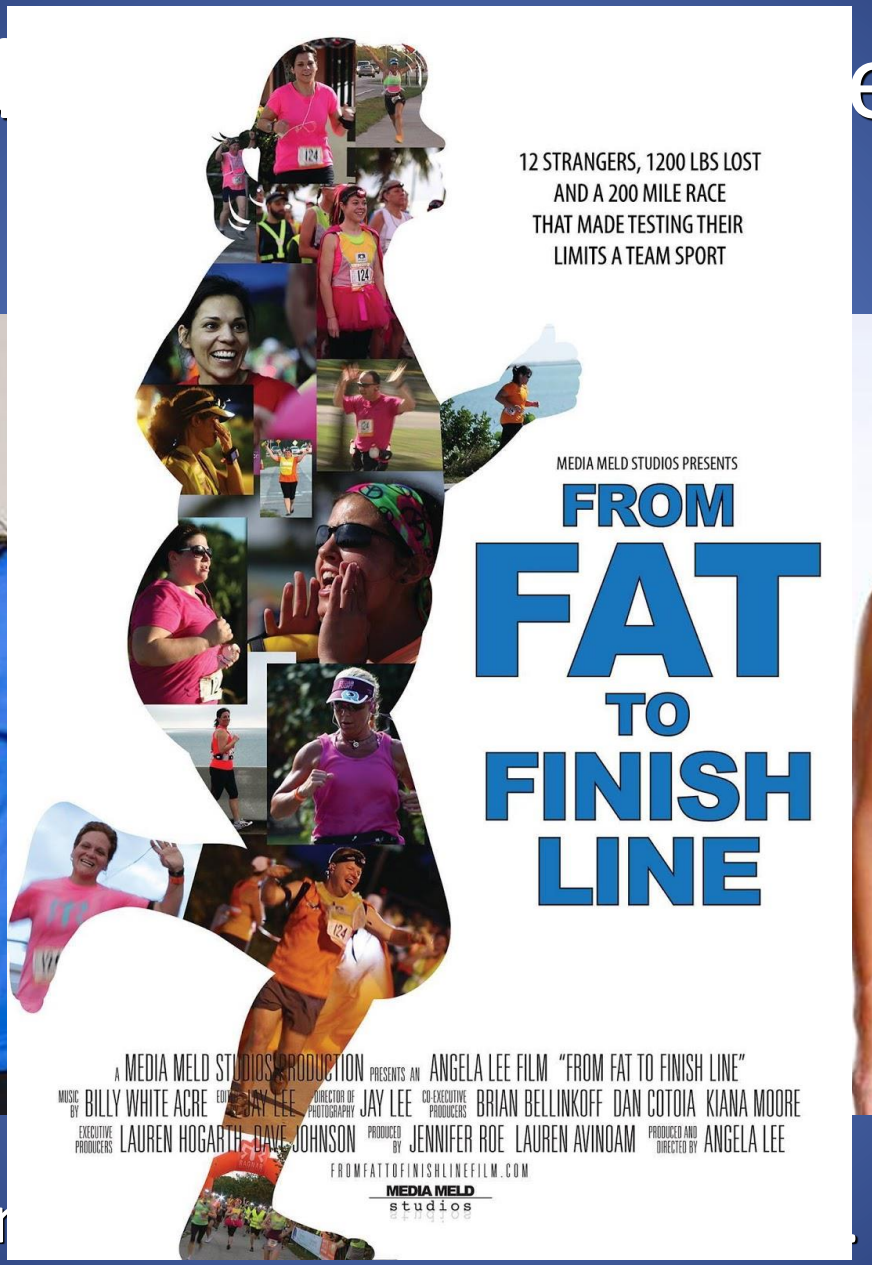


NO ONE!


The Lean
I wish I had gimungus meaty muscles like that guy.
I bet girls line up around the block


The Meaty
I wish I could say smart, clever things like that guy.
I bet people respect him at work.
If I had a nickel for every time a girl told me she wanted to make love to my beard, well ... I'd have a quarter!
hee-hee! It's funny because that's five times.

Ad er!



- Runner up or

And the loser is…

Original Article
OBESITY BIOLOGY AND INTEGRATED PHYSIOLOGY

Obesity

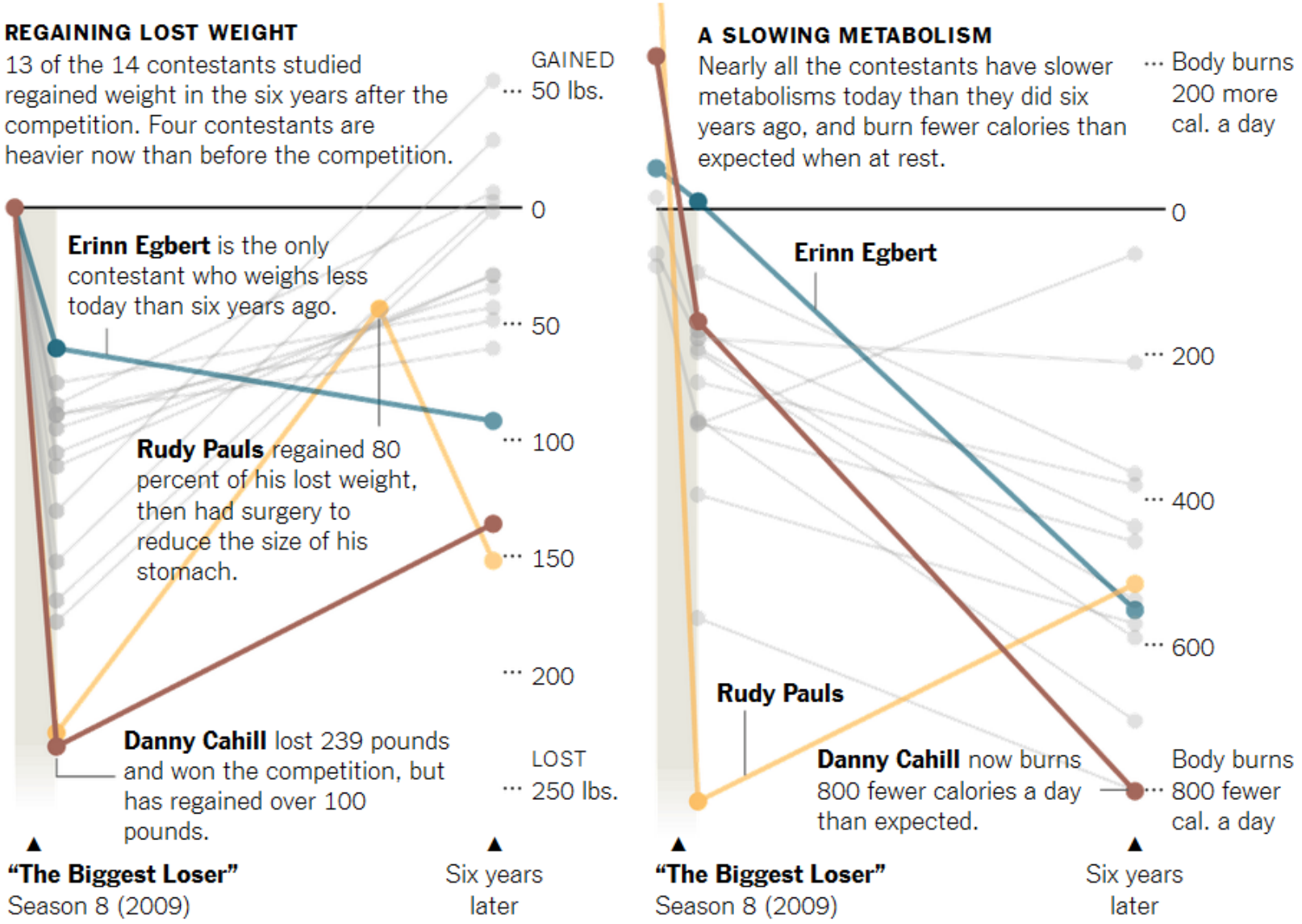
Persistent Metabolic Adaptation 6 Years After "The Biggest Loser" Competition

Erin Fothergill[1], Juen Guo[1], Lilian Howard[1], Jennifer C. Kerns[2], Nicolas D. Knuth[3], Robert Brychta[1], Kong Y. Chen[1], Monica C. Skarulis[1], Mary Walter[1], Peter J. Walter[1], and Kevin D. Hall[1]

Obesity (2016) **00**, 00-00. doi:10.1002/oby.21538

Biggest Losers Fight a Slower Metabolism

A study of contestants from "The Biggest Loser" found their metabolisms slowed during and after the competition, making it difficult to maintain weight loss.



Sources: Obesity; individual contestants

By The New York Times

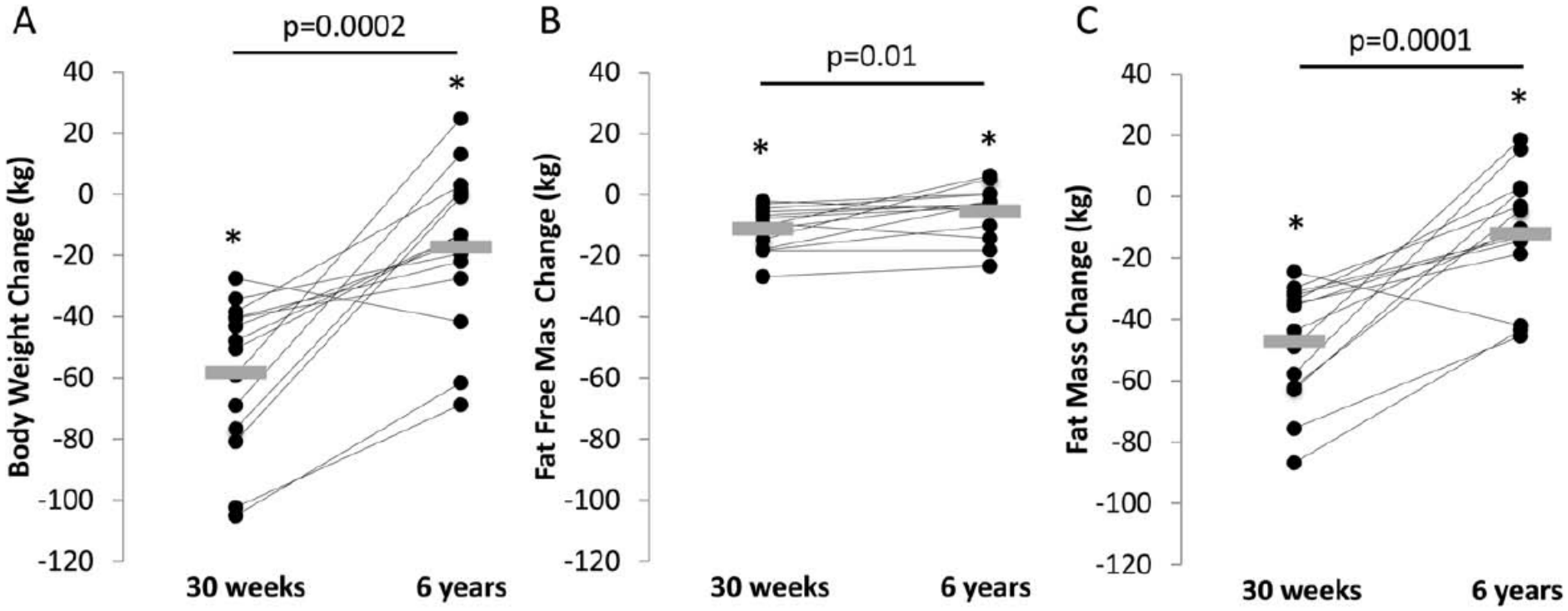


Figure 2 Individual (•) and mean (gray rectangles) changes in (**A**) body weight, (**B**) fat-free mass, and (**C**) fat mass at the end of "The Biggest Loser" 30-week weight loss competition and after 6 years. Horizontal bars and corresponding *P* values indicate comparisons between 30 weeks and 6 years. *$P < 0.05$ compared with baseline.

Why did they fail to keep it off?

- “Reality TV” is NOT real.
- No real gain in muscle mass.
- Serious drop off in exercise program.
- And their metabolic rate dropped significantly.
- Without exercise you can’t cut calories enough!

Relax, don't do it…

OPEN ACCESS Freely available online

PLOS ONE

Relaxation Response Induces Temporal Transcriptome Changes in Energy Metabolism, Insulin Secretion and Inflammatory Pathways

Manoj K. Bhasin[1,4,5☯], Jeffery A. Dusek[6☯], Bei-Hung Chang[7,8☯], Marie G. Joseph[5], John W. Denninger[1,2], Gregory L. Fricchione[1,2], Herbert Benson[1,3¶], Towia A. Libermann[1,4,5*¶]

1 Benson-Henry Institute for Mind Body Medicine at Massachusetts General Hospital, Boston, Massachusetts, United States of America, **2** Department of Psychiatry, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, United States of America, **3** Department of Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, United States of America, **4** Department of Medicine, Division of Interdisciplinary Medicine and Biotechnology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts, United States of America, **5** BIDMC Genomics and Proteomics Center, Beth Israel Deaconess Medical Center, Boston, Massachusetts, United States of America, **6** Institute for Health and Healing, Abbott Northwestern Hospital, Minneapolis, Minnesota, United States of America, **7** VA Boston Healthcare System, Boston, Massachusetts, United States of America, **8** Department of Health Policy and Management, Boston University School of Public Health, Boston, Massachusetts, United States of America

Epigenetics, again!

Now is that gratitude???

Spirituality in Clinical Practice
2015, Vol. 2, No. 1, 5–17

2326-4500/15/$12.00 http://dx.doi.org/10.1037/scp0000050

The Role of Gratitude in Spiritual Well-Being in Asymptomatic Heart Failure Patients

Paul J. Mills, Laura Redwine, Kathleen Wilson, Meredith A. Pung, Kelly Chinh, Barry H. Greenberg, Ottar Lunde, Alan Maisel, and Ajit Raisinghani
University of California, San Diego

Alex Wood
University of Stirling

Deepak Chopra
University of California, San Diego, and
Chopra Center for Wellbeing, Carlsbad, California

Gratitude improved, sleep, mood, self-sufficiency, and inflammatory biomarkers.

Think positive!

TABLE 2 Optimism and Pessimism as Predictors of Clinical Outcomes

First Author (Ref. #)	Year	n	Follow-Up (yrs)	Endpoints	Adjusted RR (95% CI)*
Pessimism as a risk factor					
Brummet et al. (13)	2006	6,958	40.0	ACM	1.42 (1.13–1.77)
Grossbart et al. (14)	2009	7,216	32.0	ACM	1.32 (1.13–1.77)
Optimism as a buffer					
Kubzansky et al. (15)	2004	1,306	10.0	MI/CV death	0.44 (0.26–0.74)
Giltay et al. (16)	2004	941	9.1	CV death	0.27 (0.12–0.57)
Giltay et al. (17)	2006	554	15.0	CV death	0.45 (0.29–0.68)
Tindle et al. (18)	2009	97,253	8.0	CV death	0.76 (0.64–0.90)
Nabi et al. (19)	2010	23,216	7.0	Stroke	0.52 (0.29–0.93)
Kim et al. (20)	2011	6,044	2.0	Stroke	0.90 (0.84–0.97)†

*Risk ratios are primarily for first versus third tercile or fourth quartile. †For each unit increase in optimism.

ACM = all-cause mortality; CI = confidence interval; CV = cardiovascular; RR = risk ratio; MI = myocardial infarction.

Change your habits-change your life!

- ***<u>Winning is a habit.</u>***
  Watch your thoughts, they become your beliefs.
  Watch your beliefs, they become your words.
  Watch your words, they become your actions.
  Watch your actions, they become your habits.
  Watch your habits, they become your character.

Adam 1 (or Eve)

- Richest…
- Busiest…
- Best…
- Most stuff…

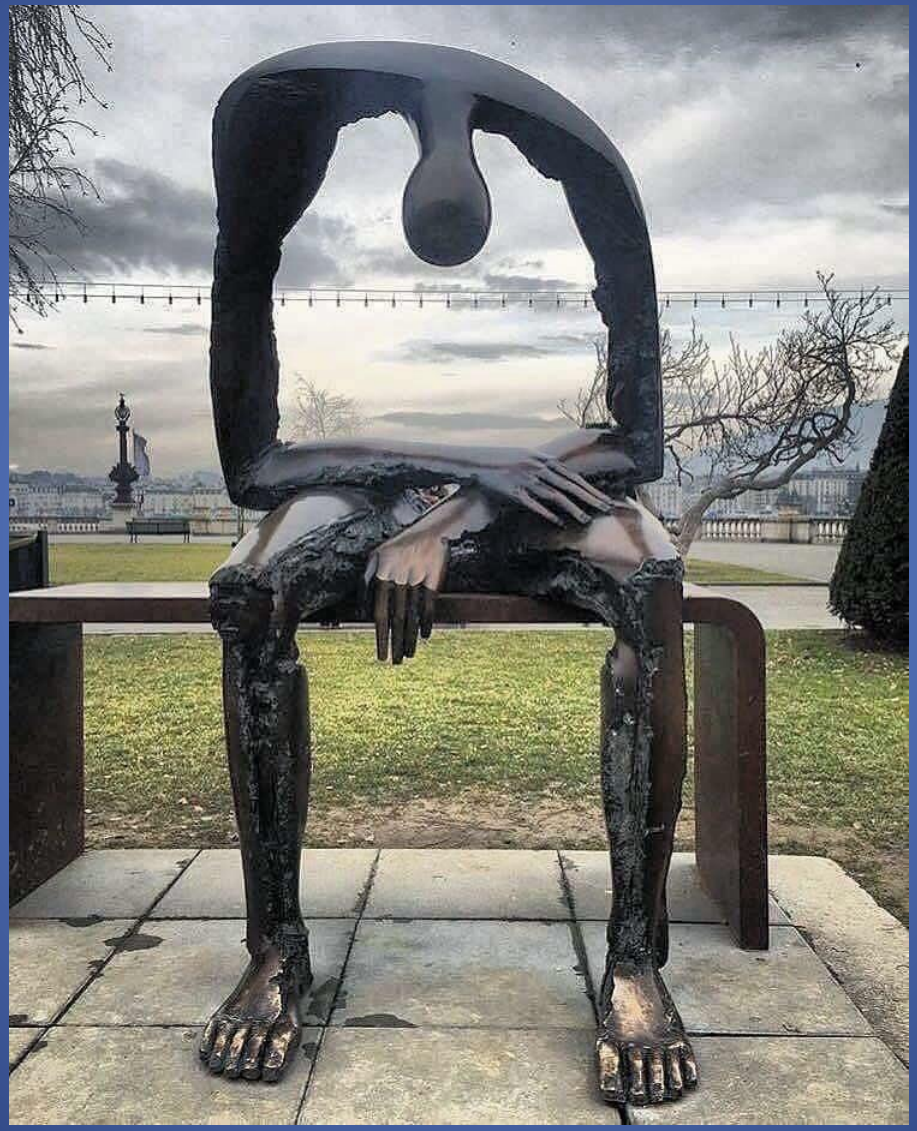
Adam 2 (or Eve)

- Happiest!
- Deepest relationship!
- Most caring!
- Most giving!

The Power of Choice

- "I am not a product of my circumstances. I am a product of my decisions.
  S. Covey

Life is hOllOw withOut GOD.



100 years vs eternity?

“Your mission if you should choose to accept it…”

- Think positive.
- Don’t stress-waste of time and energy.
- Laugh, be happy.
- Helping others helps YOU.
- Be social.
- Have purpose.

Nothing gives purpose like God!

The Dickens Process

- Tony Robbins.
- What are your beliefs COSTING you?
- In the past?
- In the present?
- What will they cost you in YOUR future?

Sleep duration and mortality – Does weekend sleep matter?

Torbjörn Åkerstedt[1,2] | Francesca Ghilotti[3,4] | Alessandra Grotta[5,6] | Hongwei Zhao[7] | Hans-Olov Adami[5] | Ylva Trolle-Lagerros[3,8] | Rino Bellocco[4,5]

J Sleep Res. 2018;e12712.
https://doi.org/10.1111/jsr.12712

Work stress and risk of death in men and women with and without cardiometabolic disease: a multicohort study

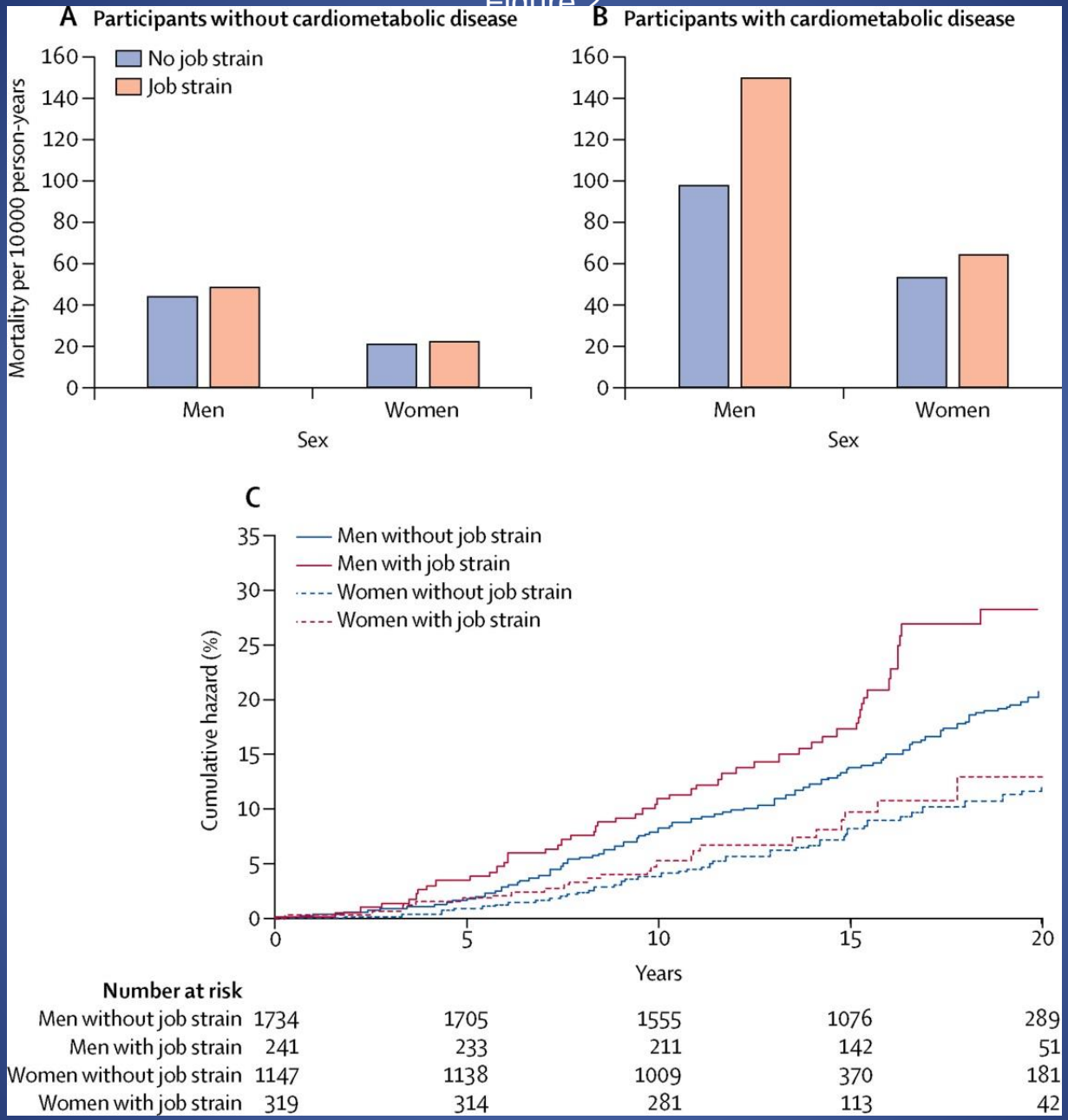
Prof Mika Kivimäki, FMedSci, Jaana Pentti, MSc, Jane E Ferrie, PhD, Prof G David Batty, DSc, Solja T Nyberg, PhD, Markus Jokela, PhD, Prof Marianna Virtanen, PhD, Prof Lars Alfredsson, PhD, Prof Nico Dragano, PhD, Eleonor I Fransson, PhD, Prof Marcel Goldberg, MD, Prof Anders Knutsson, PhD, Prof Markku Koskenvuo, MD, Aki Koskinen, MSc, Prof Anne Kouvonen, PhD, Ritva Luukkonen, PhD, Tuula Oksanen, MD, Prof Reiner Rugulies, PhD, Prof Johannes Siegrist, PhD, Archana Singh-Manoux, PhD, Sakari Suominen, MD, Prof Töres Theorell, MD, Ari Väänänen, PhD, Prof Jussi Vahtera, MD, Prof Peter J M Westerholm, MD, Prof Hugo Westerlund, PhD, Marie Zins, PhD, Prof Timo Strandberg, MD, Prof Andrew Steptoe, DSc, Prof John Deanfield, FRCP

The Lancet Diabetes & Endocrinology

DOI: 10.1016/S2213-8587(18)30140-2

ELSEVIER

Figure 2



Number at risk

Men without job strain	1734	1705	1555	1076	289
Men with job strain	241	233	211	142	51
Women without job strain	1147	1138	1009	370	181
Women with job strain	319	314	281	113	42

The Lancet Diabetes & Endocrinology DOI: (10.1016/S2213-8587(18)30140-2)


YOU DISGUST ME RONALD!
YOU'RE NOT EVEN SCARY!
I'VE KILLED MORE PEOPLE THAN YOU


I RUN BECAUSE
PUNCHING
PEOPLE IS
FROWNED UPON

Associations of Fitness, Physical Activity, Strength, and Genetic Risk With Cardiovascular Disease

Longitudinal Analyses in the UK Biobank Study

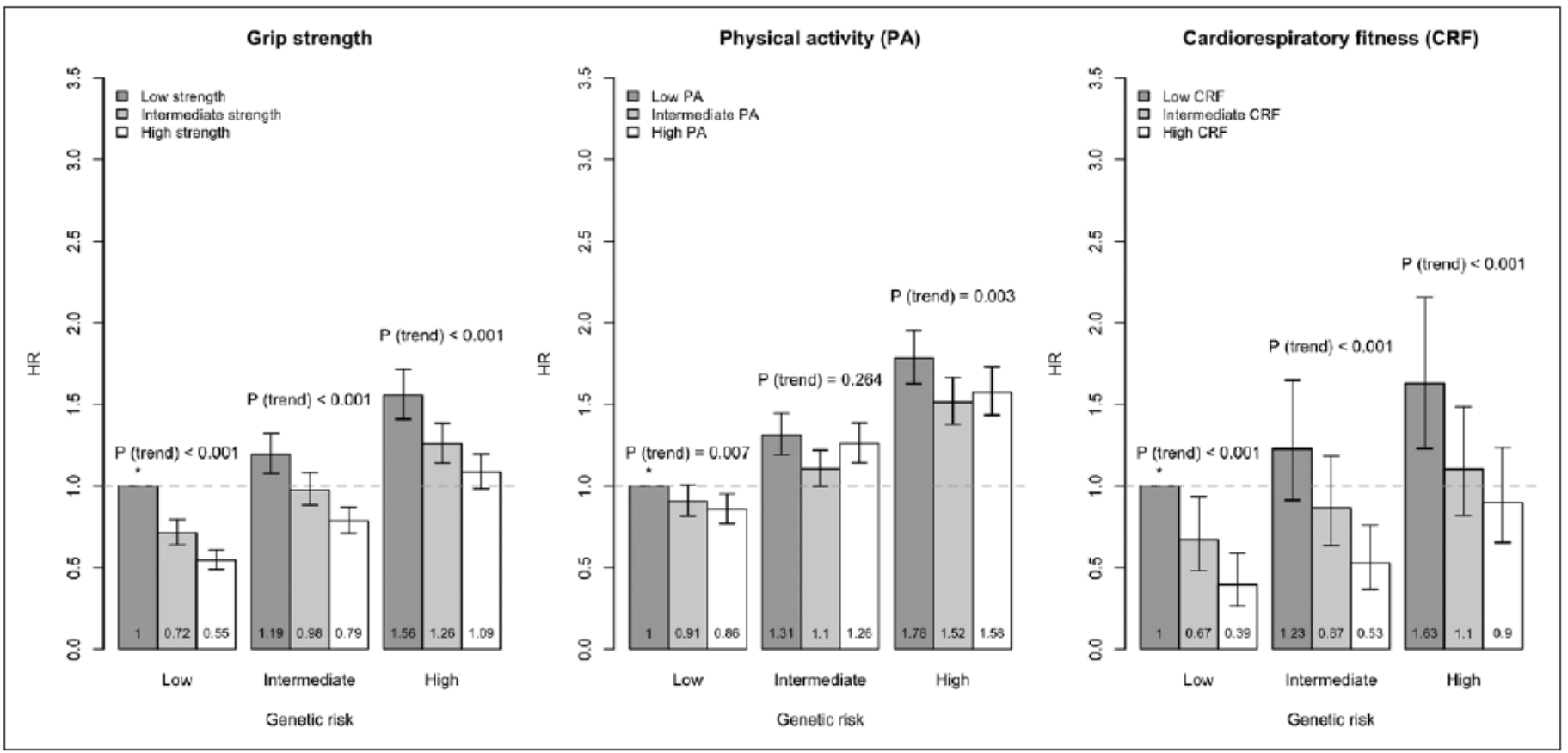
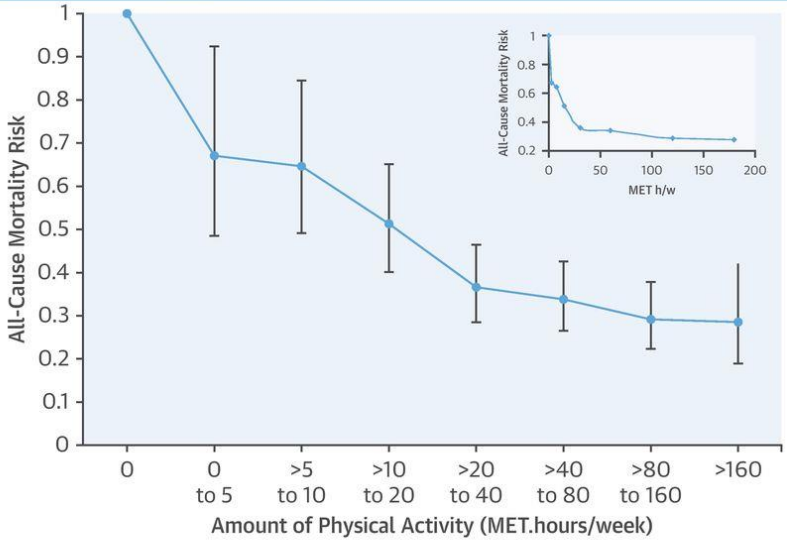


Figure 1. Associations of grip strength, physical activity, and cardiorespiratory fitness with coronary heart disease by genetic risk.

Circulation. 2018;137:00–00. DOI: 10.1161/CIRCULATIONAHA.117.032432

CENTRAL ILLUSTRATION: Habitual Physical Activity and Mortality in Patients With Stable Coronary Artery Disease

All-cause mortality risk associated with each doubling of habitual physical activity volume, and by linear increase in physical activity



Characteristics associated with greatest potential to benefit from increase in physical activity

↑ ABC-CHD risk score

- ↑ Age
- Smoker
- Diabetes
- Peripheral artery disease
- ↑ Troponin T
- ↑ NT-proBNP
- ↑ LDL cholesterol

Stewart, R.A.H. et al. J Am Coll Cardiol. 2017;70(14):1689-700.

Ralph A.H. Stewart et al. JACC 2017;70:1689-1700

JACC

JAMA Internal Medicine | Original Investigation

Association of "Weekend Warrior" and Other Leisure Time Physical Activity Patterns With Risks for All-Cause, Cardiovascular Disease, and Cancer Mortality

Gary O'Donovan, PhD; I-Min Lee, ScD; Mark Hamer, PhD; Emmanuel Stamatakis, PhD

Table 3. Cox Proportional Hazards Regression for Associations Between Physical Activity Pattern and All-Cause Mortality by Sex[a]

Physical Activity Pattern[b]	HR (95% CI)	
	Men (n = 29 181)	Women (n = 34 410)
Inactive	1 [Reference]	1 [Reference]
Insufficiently active	0.71 (0.64-0.78)	0.68 (0.71-0.74)
Weekend warrior	0.78 (0.64-0.95)	0.72 (0.55-0.94)
Regularly active	0.63 (0.54-0.73)	0.57 (0.47-0.68)

JAMA Intern Med. 2017;177(3):335-342. doi:10.1001/jamainternmed.2016.8014
Published online January 9, 2017.

Journal of the American College of Cardiology
© 2011 by the American College of Cardiology Foundation
Published by Elsevier Inc.

Vol. 57, No. 18, 2011
ISSN 0735-1097/$36.00
doi:10.1016/j.jacc.2010.12.025

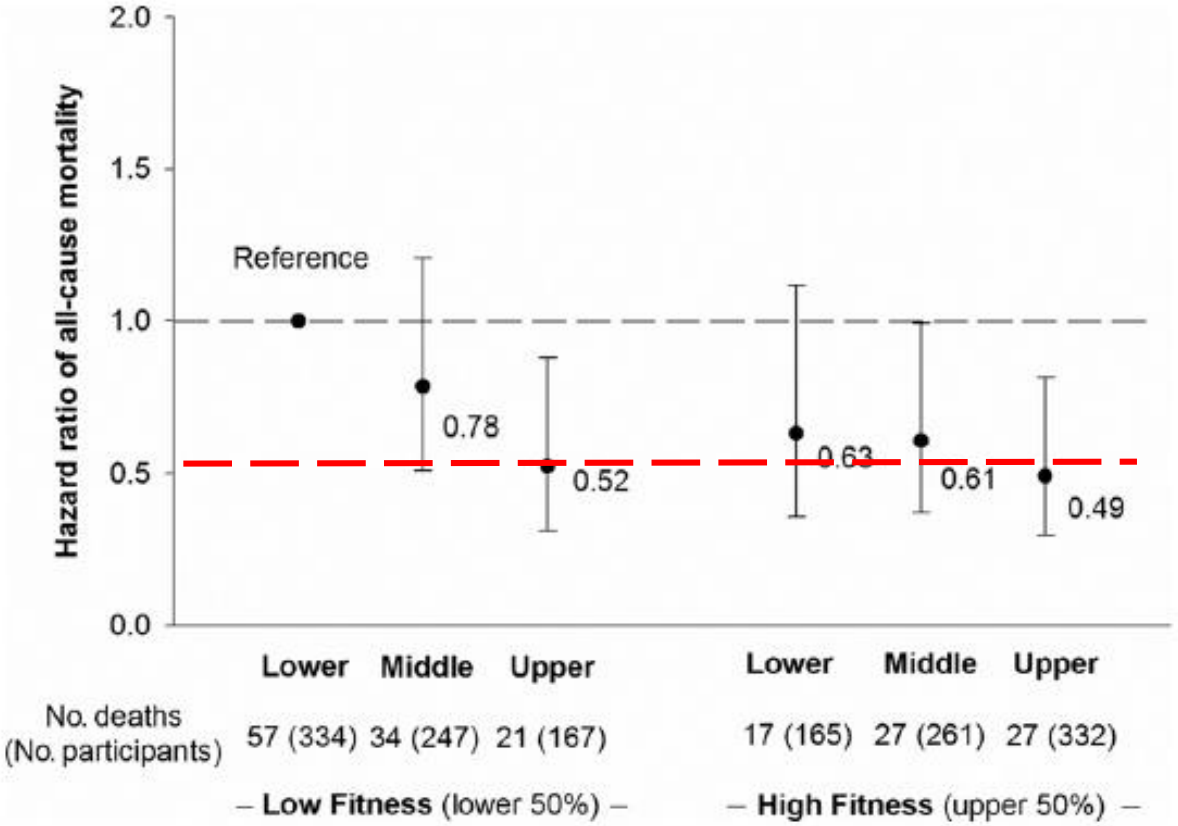
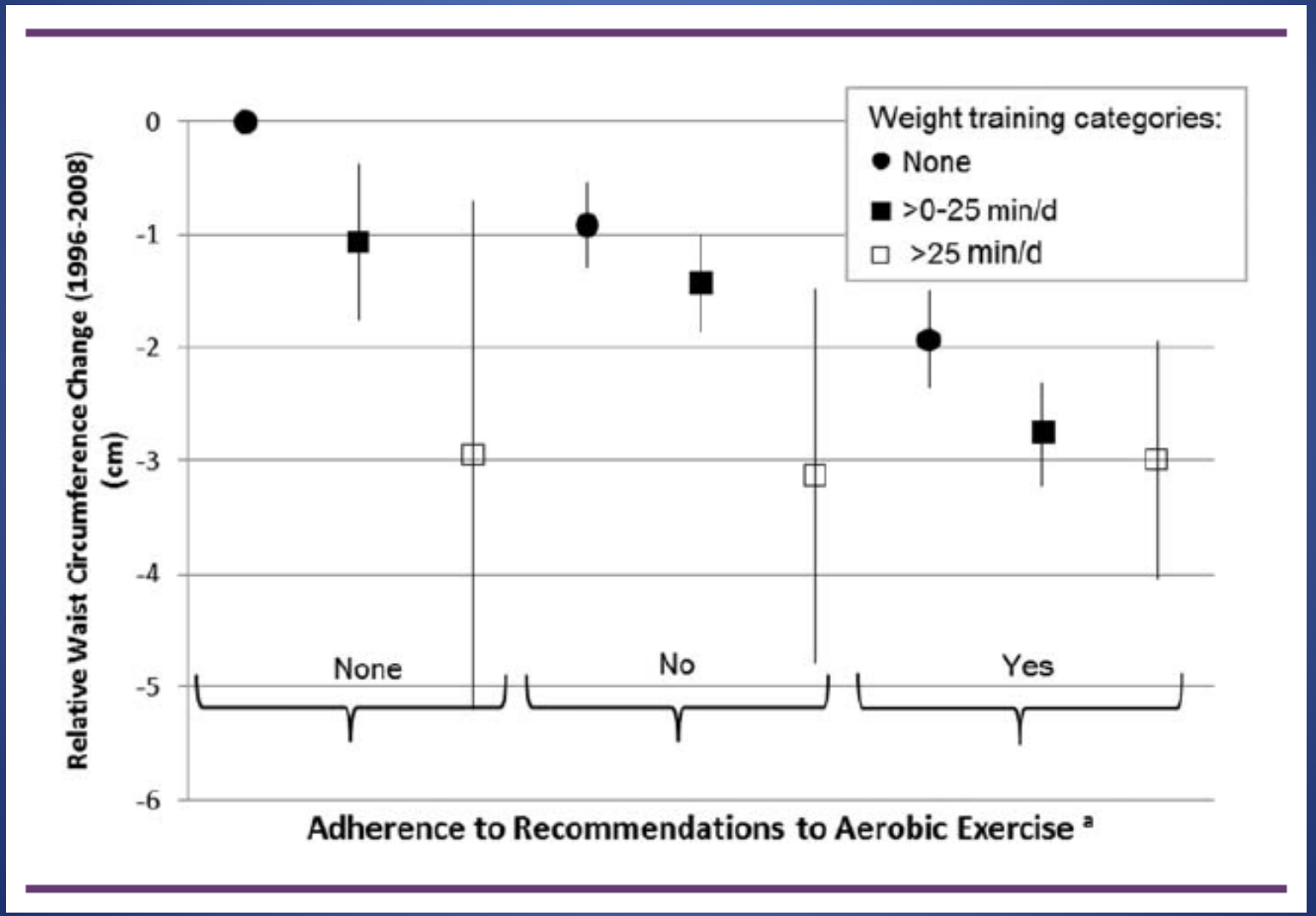


Figure 1 **Muscular Strength, Cardiorespiratory Fitness, and Mortality in Hypertension**

Combined association of muscular strength (thirds) and cardiorespiratory fitness (low fitness, high fitness) with hazard ratio of all-cause mortality after adjustment for age, physical activity, current smoking, alcohol intake, body mass index, systolic and diastolic blood pressure, total cholesterol, diabetes, abnormal electrocardiogram, and family history of cardiovascular disease. **Error bars** represent 95% confidence interval.

Weight Training, Aerobic Physical Activities, and Long-Term Waist Circumference Change in Men

Rania A. Mekary[1,2], Anders Grøntved[1,3], Jean-Pierre Despres[4], Leandro Pereira De Moura[5,6], Morteza Asgarzadeh[1], Walter C. Willett[1,7,8], Eric B. Rimm[1,7,8], Edward Giovannucci[1,7,8], and Frank B. Hu[1,7,8]



Obesity (2014) **00**, 00-00. doi:10.1002/oby.20949

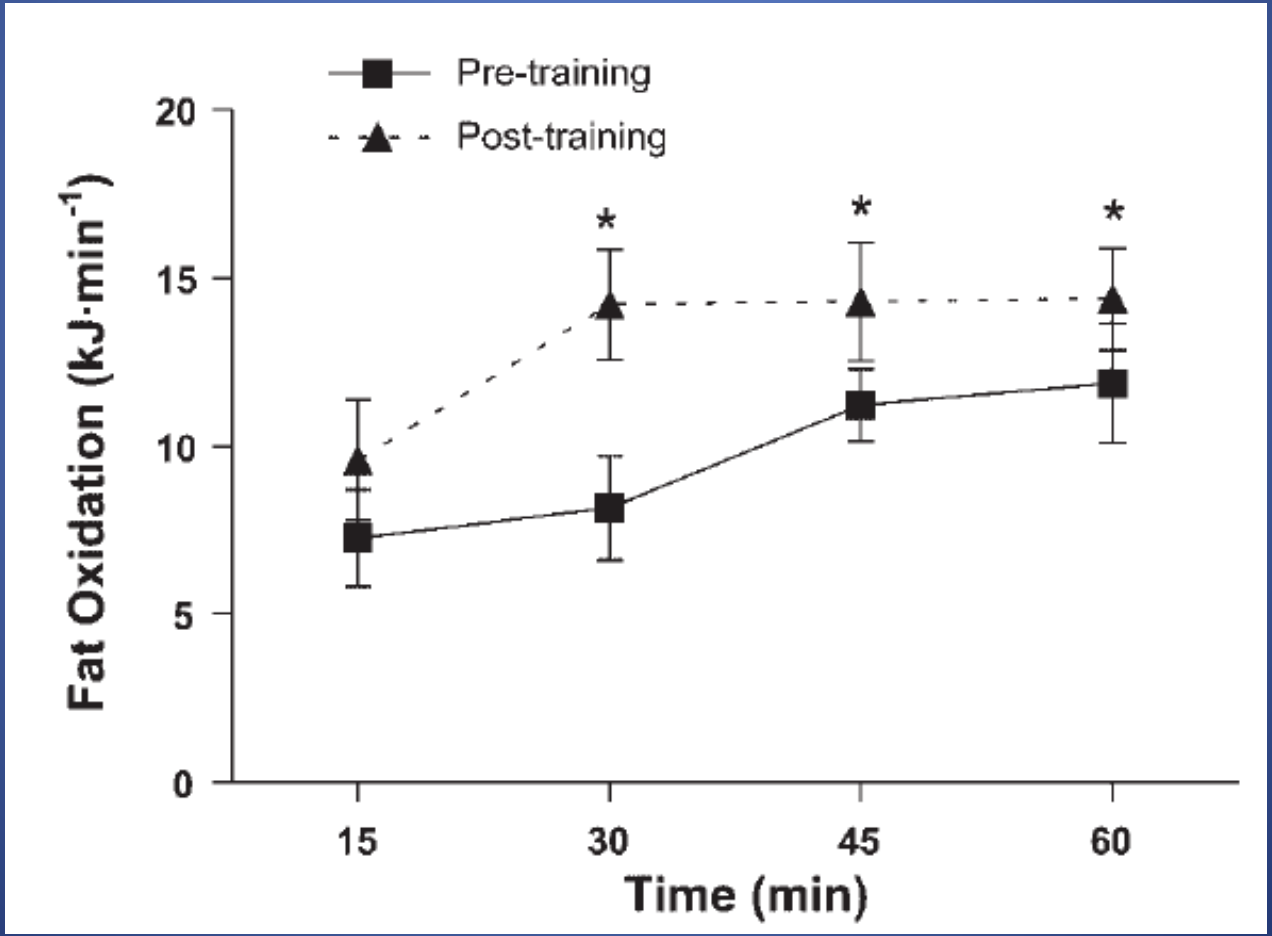
HIIT it!

- High Intensity Interval Training.
- Short bouts of near max effort (really max effort) with longer recovery periods.
- Many different programs, but most studies show that you only need 4-5 cycles to get the benefit!

Two weeks of high-intensity aerobic interval training increases the capacity for fat oxidation during exercise in women

Jason L. Talanian,[1] Stuart D. R. Galloway,[2] George J. F. Heigenhauser,[3] Arend Bonen,[1] and Lawrence L. Spriet[1]

[1]Department of Human Health and Nutritional Sciences, University of Guelph, Guelph, Ontario, Canada, [2]Department of Sport Studies, University of Stirling, Stirling, Scotland; and [3]Department of Medicine, McMaster University, Hamilton, Ontario, Canada



J Appl Physiol 102: 1439–1447, 2007.

2.5 mins a day vs 45?

Physiological Reports

Open Access

Physiological Reports ISSN 2051-817X

ORIGINAL RESEARCH

Total daily energy expenditure is increased following a single bout of sprint interval training

Kyle J. Sevits[1], Edward L. Melanson[2,3], Tracy Swibas[3], Scott E. Binns[4], Anna L. Klochak[4], Mark C. Lonac[4], Garrett L. Peltonen[4], Rebecca L. Scalzo[4], Melani M. Schweder[4], Amy M. Smith[1], Lacey M. Wood[4], Christopher L. Melby[1] & Christopher Bell[4]

1 Department of Food Science and Human Nutrition, Colorado State University, Fort Collins, Colorado
2 Division of Endocrinology Metabolism and Diabetes, University of Colorado Anschutz Medical Campus, Denver, Colorado
3 Division of Geriatrics, University of Colorado Anschutz Medical Campus, Denver, Colorado
4 Department of Health and Exercise Science, Colorado State University, Fort Collins, Colorado

Physiol Rep, 1 (5), 2013, e00131, doi: 10.1002/phy2.131

A 45-Minute Vigorous Exercise Bout Increases Metabolic Rate for 14 Hours

AMY M. KNA

[1]Human Perfo
[2]University of
NC; and [3]Bioi
Kannapolis, N

VID C. NIEMAN[1]

apolis, NC;
pus, Kannapolis,
earch Campus,

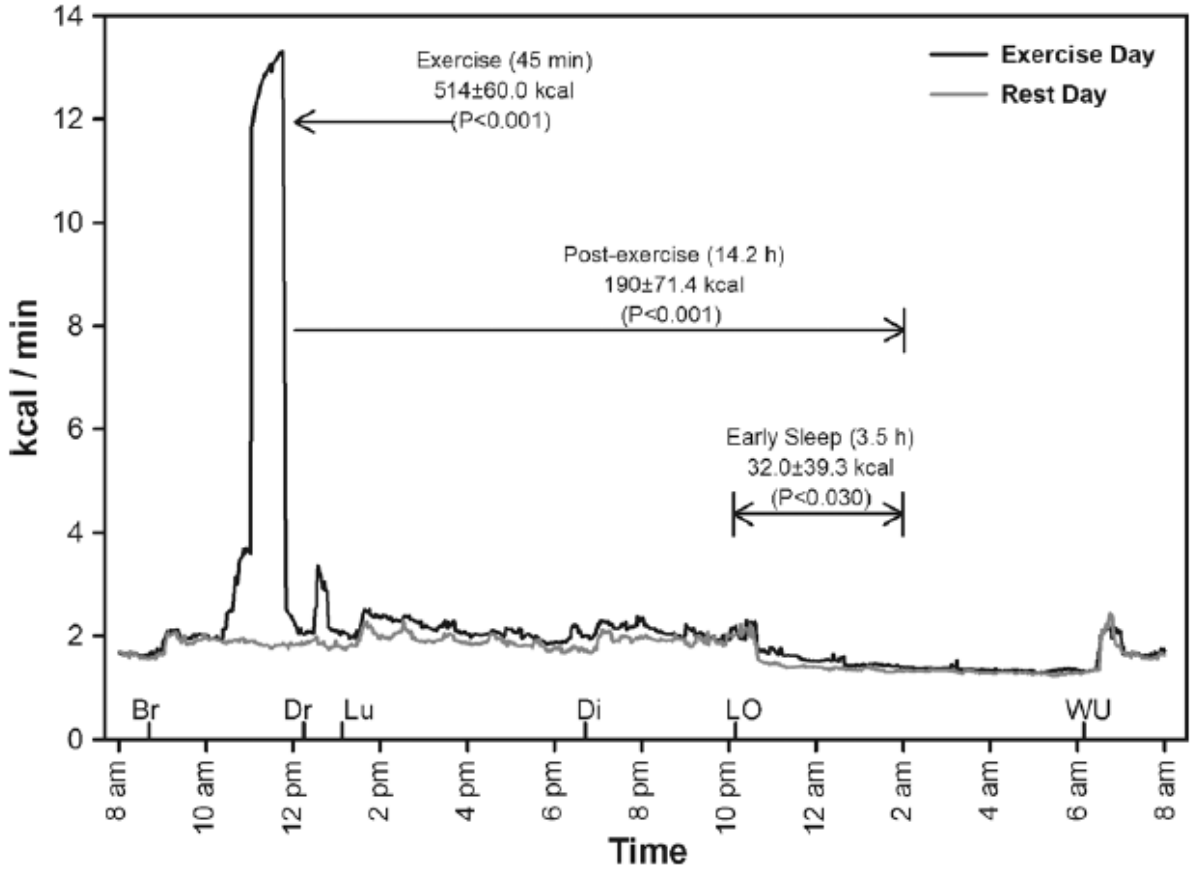


FIGURE 1—Average 24-h energy expenditure on rest and exercise days. Forty-five minutes of cycling resulted in 519 ± 60.9 kcal of energy expended above rest day ($P < 0.001$), whereas 190 ± 71.4 kcal was expended above levels on the rest day for 14.2 h after exercise ($P < 0.001$). Net energy expenditure difference from the start of sleep to 18 h after exercise was 32.0 ± 39.3 kcal ($P = 0.030$).

Med. Sci. Sports Exerc., Vol. 43, No. 9, pp. 1643–1648, 2011.

ORIGINAL ARTICLE

Mark D. Schuenke · Richard P. Mikat
Jeffrey M. McBride

Effect of an acute period of resistance exercise on excess post-exercise oxygen consumption: implications for body mass management

Eur J Appl Physiol (2002) 86: 411–417
DOI 10.1007/s00421-001-0568-y

Muscle burns more calories than fat!