HIGH INTENSITY
HEALTH

NYC 2018

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- None of the content herein has been evaluated by the Food and Drug Administration. These ideas are not intended to diagnose, treat, cure, or prevent any disease.

OUTLINE

- **Essential Non-Nutritional** Factors that **Affect Ketosis**
- **Exercise and Time-Restricted Feeding**
- **Gut health on keto**: key insights all keto dieters should know
- Importance of the **Gut-Liver Axis on Ketosis**
- **Carb Cycling**, **Meal Prep** and **Seasonal Eating Tips**
- **Mindset & Meal Prep**


The Care Group, P.C.
Page Liked · November 8, 2009 ·
Allowed on timeline
Dr. Guillory and the staff — with Gerard Guillory, Mike Mutzel, Nhu Nguyen and Jessica Baldovsky.
1
1 Share
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The biggest challenge we face today is to prevent doing something we “think” is healthy but that will discovered to be unhealthy in hindsight a decade from now

IN 2002 **THROWING OUT THE EGG YOLK** WAS CONSIDERED **HEALTHY**

"If It's ***"gluten free"*** It's Healthy" - me circa 2012

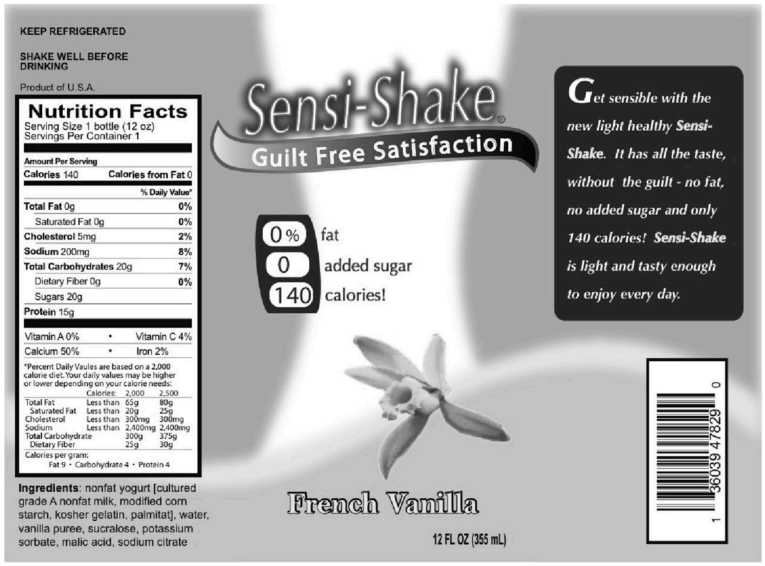


Figure 2. Sensible shake label.

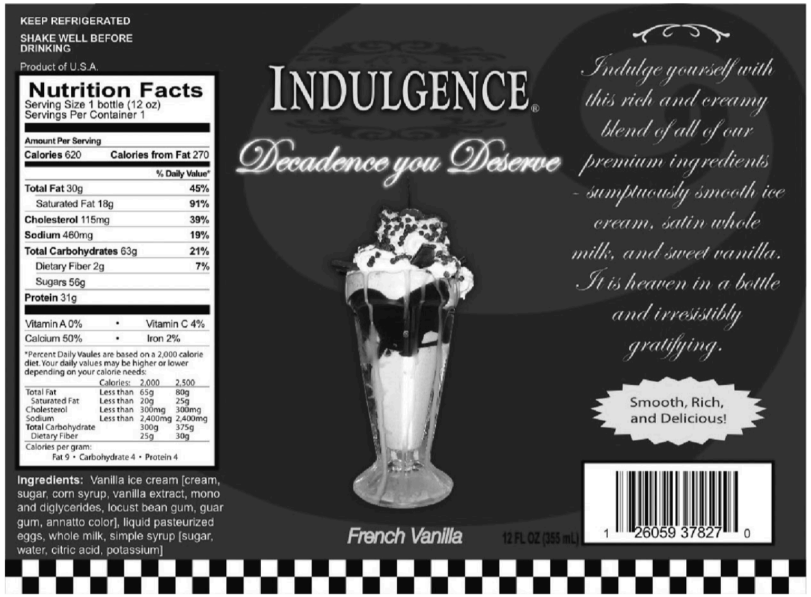


Figure 1. Indulgent shake label.

Health Psychology
2011, Vol. 30, No. 4, 424–429

0278-6133/11/$12.00 DOI: 10.1037/a0023467

BRIEF REPORT

Mind Over Milkshakes: Mindsets, Not Just Nutrients, Determine Ghrelin Response

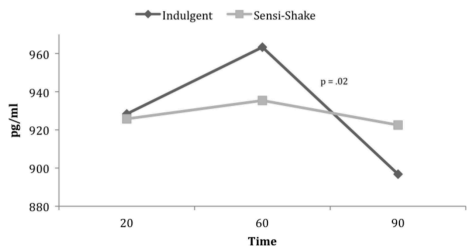
Alia J. Crum
Yale University

William R. Corbin
Arizona State University

Kelly D. Brownell and Peter Sal
Yale University

Objective: To test whether physiological satiation as measured by th depending on the mindset in which one approaches consumption of occasions, participants (n = 46) consumed a 380-calorie milkshake unc a 620-calorie "indulgent" shake or a 140-calorie "sensible" shake. Ghrel blood samples at 3 time points: baseline (20 min), anticipatory (60 min) During the first interval (between 20 and 60 min) participants we (misleading) label of the shake. During the second interval (between 6 asked to drink and rate the milkshake. ***Results:*** The mindset of indu steeper decline in ghrelin after consuming the shake, whereas the m relatively flat ghrelin response. Participants' satiety was consistent wi consuming rather than the actual nutritional value of what they consur food consumption on ghrelin may be psychologically mediated, an physiological responses to food.

Keywords: mindset, nutrition, ghrelin, hunger, product labeling



Participants' satiety was consistent with what they believed they were consuming rather than the actual nutritional value of what they consumed.

Applied Economic Perspectives and Policy Advance Access published July 10, 2012
Applied Economic Perspectives and Policy (2012) volume 0, number 0, pp. 1–22.
doi:10.1093/aepp/pps028

Featured Article

How Package Design and Packaged-based Marketing Claims Lead to Overeating

Pierre Chandon*

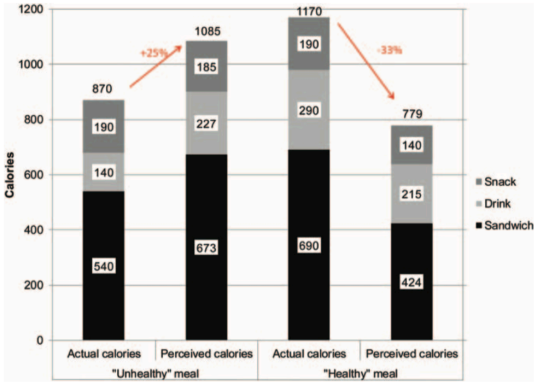
Pierre Chandon is Professor of Marketing and Director of the Social Science Research Centre at INSEAD, Fontainebleau, France.
*Correspondence may be sent to: pierre.chandon@insead.edu.

Accepted 11 June 2012.

Abstract *Because packaging reaches cons purchase and consumption, it has become an manufacturers and retailers. In this paper, health and nutrition claims made on packag foods appear healthier than they are, thereby lower perceived calorie intake. I then show h and sizes) biases people's perception of quan for supersized packages and portions that ap I examine the extent to which mandatory nu package claims, public promotion of mindful could limit the biasing effects of packaging on*

JEL Codes: I12, I18, M31, M37, M39, M8

Figure 1 Positive and Negative Health Halo Effects on Calorie Perception



Research Article

The Effect of Fitness Branding on Restrained Eaters' Food Consumption and Postconsumption Physical Activity

Joerg Koenigstorfer and Hans Baumgartner*

*Joerg Koenigstorfer is Professor of Sport & Health Management, Technische Universität München (e-mail: joerg.koenigstorfer@tum.de).

Hans Baumgartner is the Smeal Professor of Marketing, Smeal College of Business, Pennsylvania State University (e-mail: jxb14@psu.edu).

The authors thank Lisa E. Bolton, Rik Pieters, and Karen Page Winterich for their valuable feedback on the manuscript. The constructive comments of the review team are also greatly appreciated. This work was supported by a fellowship within the post

"…restrained eaters because it discourages physical activity despite an increase in consumption."

…id
…rs
have developed fitness-branded food that may lead restrained eaters (i.e., consumers who are chronically concerned about their body weight) to believe that they can achieve these two goals at the same time by consuming the food. The purpose o this research is to investigate the effects of fitness branding in food marketing (i.e., the integration of fitness into the brandin of food) on consumption and physical activity in restrained (vs. unrestrained) eaters. The authors show that fitness branding increases consumption volumes for restrained eaters unless consumers view the food as dietary forbidden. Restrained eate are also less physically active after consuming fitness-branded food, and food consumption volumes mediate this effect in restrained eaters. Fitness branding may therefore have undesirable effects on the weight-control behaviors of restrained eaters because it discourages physical activity despite an increase in consumption, which is contrary to the principle of ener balance.

SPRING INTO KETOSIS

NIH Public Access
Author Manuscript
Brain Res Rev. Author manuscript; available in PMC 2009 September 1.

Published in final edited form as:
Brain Res Rev. 2009 March ; 59(2): 293–315. doi:10.1016/j.brainresrev.2008.09.002.

THE NEUROPROTECTIVE PROPERTIES OF CALORIE RESTRICTION, THE KETOGENIC DIET, AND KETONE BODIES

Marwan A. Maalouf[1], Jong M. Rho[2], and Mark P. Mattson[3]
1*Cedars-Sinai Medical Center, Los Angeles, CA*
2*Barrow Neurological Institute and St. Joseph's Hospital & Medical Center, Phoenix, AZ.*
3*Laboratory of Neurosciences, National Institute on Aging, Intramural Research Program, Baltimore, MD.*

Abstract

The therapeutic potential of calorie restriction and the ketogenic diet have been repeatedly demonstrated in clinical settings and in various animal models of neurological disease. The underlying mechanisms involve an improvement in mitochondrial function, a decrease in the expression of apoptotic factors and an increase in the activity of neurotrophic factors. Clinical applications of ketogenic diets have been significantly hampered however by poor tolerability and potentially serious side-effects. Recent research aimed at identifying a mediator that can reproduce the neuroprotective effects of calorie restriction with less demanding changes to dietary intake suggests that ketone bodies might represent an appropriate candidate. Ketone bodies protect neurons against multiple types of neuronal injury and the underlying mechanisms are similar to those of calorie restriction and of the ketogenic diet. The present review describes the neuroprotective effects of calorie restriction, the ketogenic diet and ketone bodies and compare the molecular mechanisms of action of these interventions.

Keywords

calorie restriction; neuroprotection; ketone bodies; ketogenic diet; oxidative stress; mitochondria; brain

1. Introduction

The anticonvulsant properties of fasting have been recognized since antiquity, strongly suggesting that fasting, and more generally, calorie restriction probably represents the first effective treatment for epileptic seizures in medical history. In addition, more recent evidence suggests that the benefits of calorie restriction, elicited either by daily reduction of energy intake or by intermittent fasting, are not limited to epilepsy and might in fact include a generalized neuroprotective effect applicable to many acute and chronic neurological diseases. In view of the present obesity epidemic, however, the scientific and medical communities have realized that calorie restriction is often not a practical treatment option. Similarly, the ketogenic diet, a high-fat, low-carbohydrate diet designed to reproduce the effects of fasting, has been difficult to use in clinical settings despite proven efficacy. As a consequence, considerable effort has been invested to understand the mechanisms underlying the neuroprotective effects of calorie restriction (and potentially of the ketogenic diet) with the hope of developing alternative therapeutic options.

Correspondence: Jong M. Rho. Email: Jong.Rho@cshw.edu.

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Author Manuscript
Neurobiol Aging. Author manuscript; available in PMC 2013 February 1.

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Neurobiol Aging. 2012 February ; 33(2): 425.e19–425.e27. doi:10.1016/j.neurobiolaging.2010.10.006.

Dietary ketosis enhances memory in mild cognitive impairment

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[a] Department of Psychiatry and Behavioral Neuroscience, University of Cincinnati, Cincinnati, OH 45267-0559, USA
[b] Department of Nutritional Science, University of Cincinnati, Cincinnati, OH, 45267-0394, USA
[c] Touchstone Diabetes Research Center, University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, Texas 75390-8549

Abstract

We randomly assigned 23 older adults with Mild Cognitive Impairment to either a high carbohydrate or very low carbohydrate diet. Following the six-week intervention period, we observed improved verbal memory performance for the low carbohydrate subjects ($p = 0.01$) as well as reductions in weight ($p < 0.0001$), waist circumference ($p < 0.0001$), fasting glucose ($p = 0.009$), and fasting insulin ($p = 0.005$). Level of depressive symptoms was not affected. Change in caloric intake, insulin level, and weight were not correlated with memory performance for the entire sample, although a trend toward a moderate relationship between insulin and memory was observed within the low carbohydrate group. Ketone levels were positively correlated with memory performance ($p = 0.04$). These findings indicate that very low carbohydrate consumption, even in the short-term, can improve memory function in older adults with increased risk for Alzheimer's disease. While this effect may be attributable in part to correction of hyperinsulinemia, other mechanisms associated with ketosis such as reduced inflammation and enhanced energy metabolism also may have contributed to improved neurocognitive function. Further investigation of this intervention is warranted to evaluate its preventive potential and mechanisms of action in the context of early neurodegeneration.

Keywords

ketosis; low carbohydrate diet; memory; MCI; Alzheimer's disease

1. Introduction

Currently, there are 5.3 million cases of Alzheimer's disease (AD) with projections of as many as 16 million cases by the year 2050 (Alzheimer's Association, 2009). There is no

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Disclosure Statement
None of the authors has a conflict of interest influencing this work.

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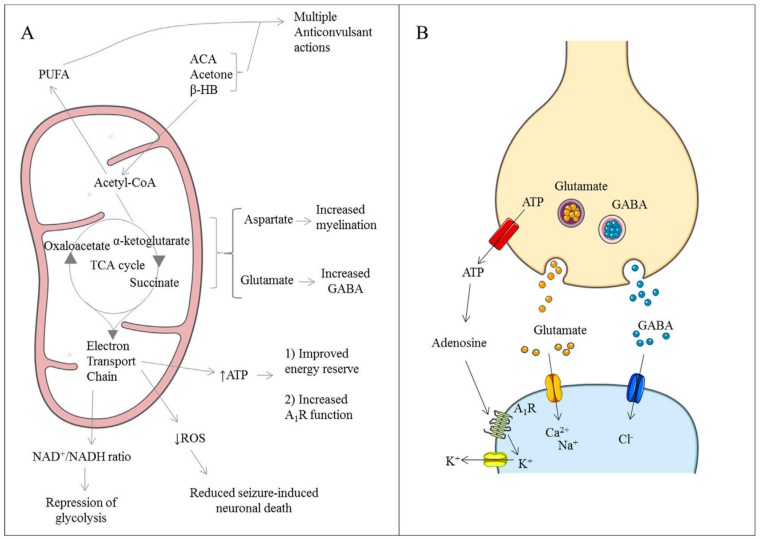
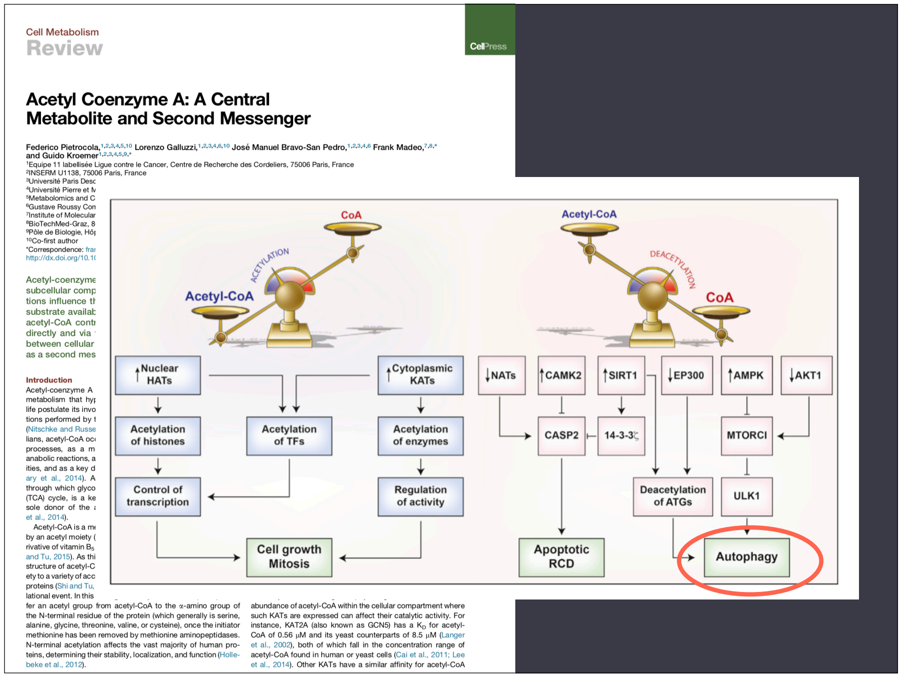
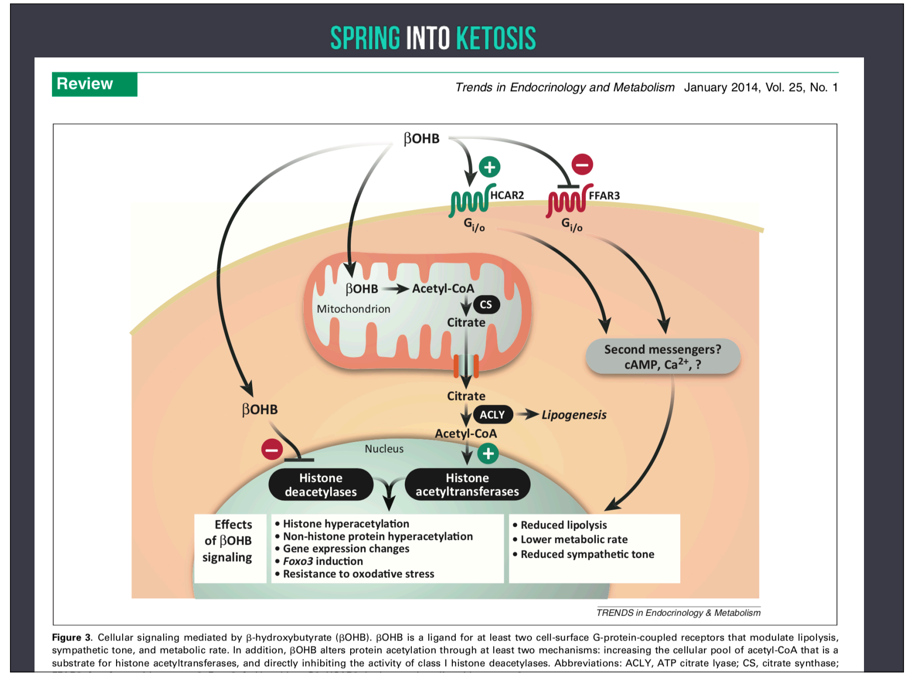
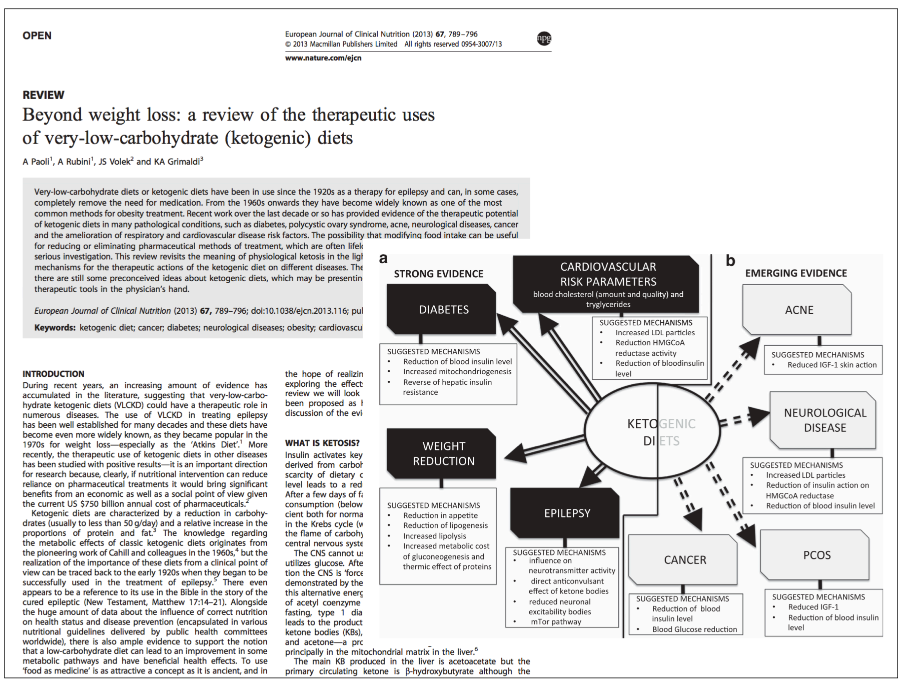
SPRING INTO KETOSIS

N.A. Youngson et al. / Seizure 52 (2017) 15–19




Cell Metabolism
Review
Acetyl Coenzyme A: A Central Metabolite and Second Messenger
Federico Pietrocola, Lorenzo Galluzzi, José Manuel Bravo-San Pedro, Frank Madeo, and Guido Kroemer
CoA
Acetyl-CoA
ACETYLATION
DEACETYLATION
↑Nuclear HATs
↑Cytoplasmic KATs
↓NATs
↑CAMK2
↑SIRT1
↓EP300
↑AMPK
↓AKT1
Acetylation of histones
Acetylation of TFs
Acetylation of enzymes
CASP2
14-3-3ζ
MTORC1
Control of transcription
Regulation of activity
Deacetylation of ATGs
ULK1
Cell growth Mitosis
Apoptotic RCD
Autophagy


SPRING INTO KETOSIS
Review
Trends in Endocrinology and Metabolism January 2014, Vol. 25, No. 1
βOHB
HCAR2
FFAR3
Gi/o
Mitochondrion
βOHB → Acetyl-CoA
CS
Citrate
Second messengers? cAMP, Ca2+, ?
ACLY
Lipogenesis
Nucleus
Histone deacetylases
Histone acetyltransferases
Effects of βOHB signaling
• Histone hyperacetylation
• Non-histone protein hyperacetylation
• Gene expression changes
• Foxo3 induction
• Resistance to oxodative stress
• Reduced lipolysis
• Lower metabolic rate
• Reduced sympathetic tone
TRENDS in Endocrinology & Metabolism
Figure 3. Cellular signaling mediated by β-hydroxybutyrate (βOHB). βOHB is a ligand for at least two cell-surface G-protein-coupled receptors that modulate lipolysis, sympathetic tone, and metabolic rate. In addition, βOHB alters protein acetylation through at least two mechanisms: increasing the cellular pool of acetyl-CoA that is a substrate for histone acetyltransferases, and directly inhibiting the activity of class I histone deacetylases. Abbreviations: ACLY, ATP citrate lyase; CS, citrate synthase;


OPEN
European Journal of Clinical Nutrition (2013) 67, 789–796
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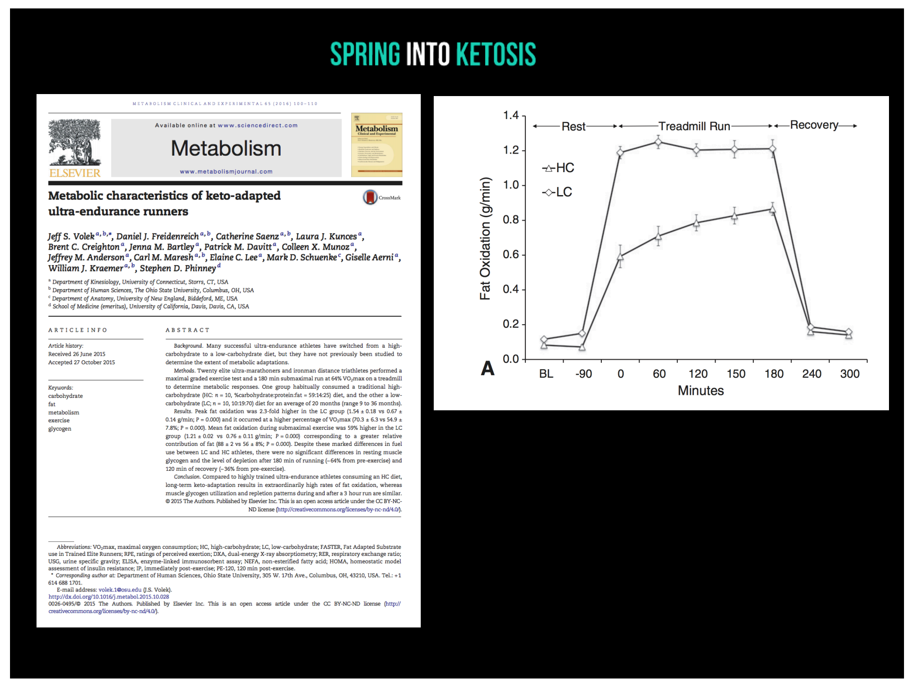
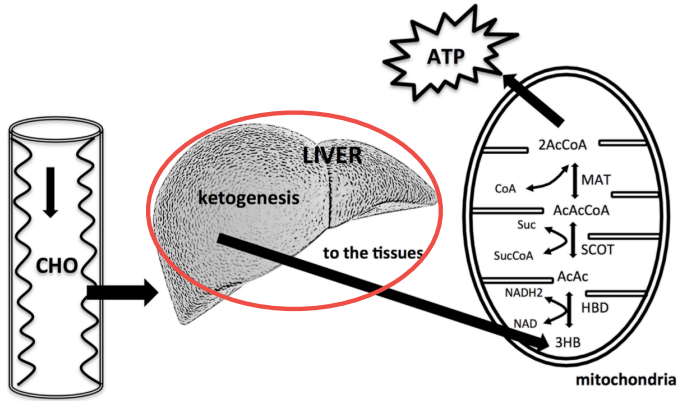
www.nature.com/ejcn
REVIEW
Beyond weight loss: a review of the therapeutic uses of very-low-carbohydrate (ketogenic) diets
A Paoli, A Rubini, JS Volek and KA Grimaldi
a
STRONG EVIDENCE
DIABETES
CARDIOVASCULAR RISK PARAMETERS
WEIGHT REDUCTION
EPILEPSY
KETOGENIC DIETS
b
EMERGING EVIDENCE
ACNE
NEUROLOGICAL DISEASE
CANCER
PCOS

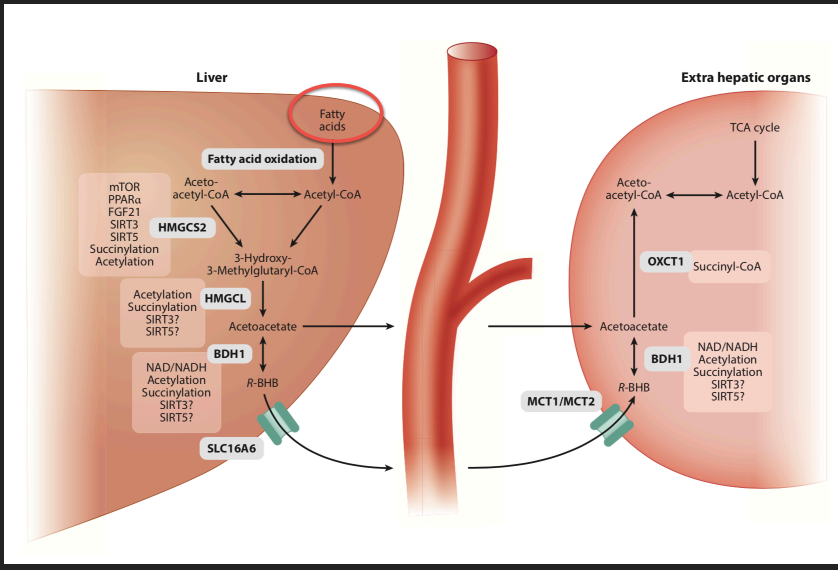

SPRING INTO KETOSIS
Metabolism
ELSEVIER
Metabolic characteristics of keto-adapted ultra-endurance runners
Jeff S. Volek, Daniel J. Freidenreich, Catherine Saenz, Laura J. Kunces, Brent C. Creighton, Jenna M. Bartley, Patrick M. Davitt, Colleen X. Munoz, Jeffrey M. Anderson, Carl M. Maresh, Elaine C. Lee, Mark D. Schuenke, Giselle Aerni, William J. Kraemer, Stephen D. Phinney
Rest
Treadmill Run
Recovery
HC
LC
Fat Oxidation (g/min)
A
Minutes

Ketones Are Made in the _____?



GURE 2 | A reduced availability of dietary carbohydrates leads to an creased liver production of KBs. The liver cannot utilize KBs because it ks the mitochondrial enzyme succinyl-CoA: 3-ketoacid (oxoacid) CoA nsferase (SCOT) necessary for activation of acetoacetate to acetoacetyl CoA. KBs are utilized by tissues, in particularly by brain. KBs enter the citric acid cycle after being converted to acetyl CoA by hydroxybutyrate dehydrogenase (HBD), succinyl-CoA: 3–CoA transferase (SCOT), and methylacetoacetyl CoA thiolase (MAT). Modified from Owen (2005), Paoli et al. (2014).

The Ketone GIGL



Newman, JC Annu. Rev. Nutr. 2017. 37:51–76

frontiers in PSYCHOLOGY

REVIEW ARTICLE
published: 02 February 2015
doi: 10.3389/fpsyg.2015.00027

Ketosis, ketogenic diet and food intake control: a complex relationship

***Antonio Paoli*[1]*, Gerardo Bosco[1], Enrico M. Camporesi[2,3] and Devanand Mangar[3,4]**

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[2] Department of Surgery, University of South Florida, Tampa, FL, USA
[3] TEAMHealth, Tampa, FL, USA
[4] Tampa General Hospital, Tampa, FL, USA

Edited by:
Tanya Zilberter, Infotonic Conseil, France
Reviewed by:
Tanya Zilberter, Infotonic Conseil, France
Jong Min Rho, University of Calgary, Canada
***Correspondence:**
Antonio Paoli, Nutrition and Exercise Physiology Laboratory, Department of Biomedical Sciences, University of Padova, Via Marzolo 3, 35031 Padova, Italy
e-mail: antonio.paoli@unipd.it

Though the hunger-reduction phenomenon reported during ketc
the underlying molecular and cellular mechanisms remain un
demonstrated to exert an anorexigenic effect via cholecysto
reducing orexigenic signals e.g., via ghrelin. However, ketone bc
to increase food intake through AMP-activated protein kinase
gamma-aminobutyric acid (GABA) and the release and produ
aim of this review is to provide a summary of our current kn
ketogenic diet (KD) on food control in an effort to unify the app
into a coherent picture.

Keywords: ketones, ketogenic diet, hunger, brain, hypothalamus, appetite

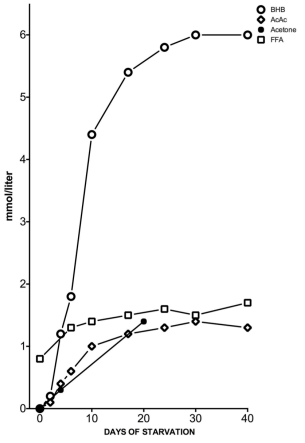

Table 1 | Blood levels during a normal diet, ketogenic diet, and diabetic ketoacidosis (Paoli et al., 2012).

Blood levels	Normal diet	Ketogenic diet	Diabetic ketoacidosis
Glucose (mg/dL)	80–120	65–80	>300
Insulin (μU/L)	6–23	6.6–9.4	≅ 0
KB conc (mmol/L)	0.1	7–8	>25
pH	7.4	7.4	<7.3

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subfornical
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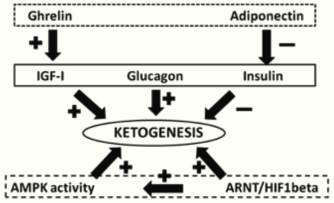
or not. These experiments demons

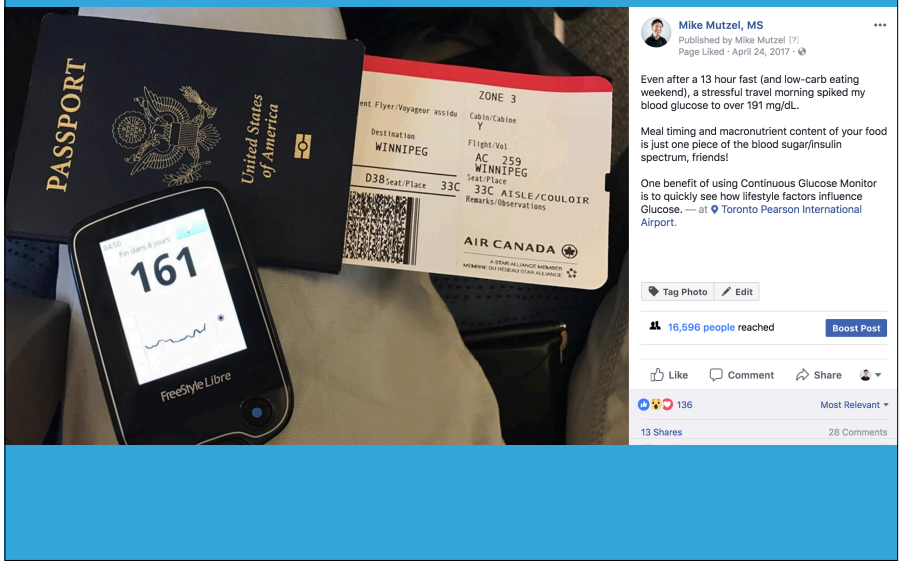
	Effect on ketogenesis	Mechanisms of action
Environmental factors		
Low carbohydrate diet	↑	Low insulin, leptin
Low calorie diet	↑	Low insulin, leptin, T3 High IGF-I, ghrelin
Physical activity	↑	Low insulin High AMPK gene expression High IGF-I
Moderate alcohol consumption	↑	High adiponectin, ghrelin Low insulin
Intrinsic factors		
AMPK activity	↑	Directly induces ketogenesis
ARNT/HIF1beta	↑	Stimulates AMPK activity
Medication		
Insulin therapy	↓	High insulin
Metformin	↑	Low insulin
Sulfonylureas	↓	High insulin
SGLT2 inhibitors	↑	Low insulin High glucagon
Octreotide analogues	↓	Low GH, glucagon

Ghrelin and Growth
Hormone: Ghrelin is a potent stimulus for Growth hormone secretion
High GH and Ghrelin increase insulin sensitivity and lower insulin

Adiponectin: increases inulin sensitivity, so maybe supportive of ketogenesis
*Omega-3 FA Increase



Kruljac, I. Ketosis in type 2 diabetes mellitus: complication or compensatory mechanism?2016 Endocrine Oncology and Metabolism



Mike Mutzel, MS
Published by Mike Mutzel [?]
Page Liked · April 24, 2017 ·

Even after a 13 hour fast (and low-carb eating weekend), a stressful travel morning spiked my blood glucose to over 191 mg/dL.

Meal timing and macronutrient content of your food is just one piece of the blood sugar/insulin spectrum, friends!

One benefit of using Continuous Glucose Monitor is to quickly see how lifestyle factors influence Glucose. — at Toronto Pearson International Airport.

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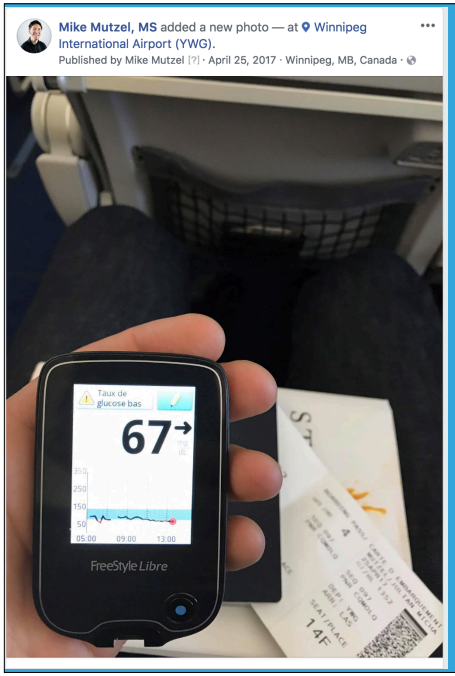
16,596 people reached · Boost Post

Like · Comment · Share

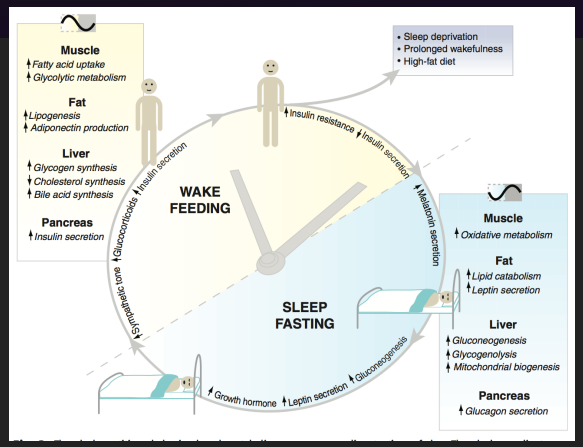
136 · Most Relevant

13 Shares · 28 Comments



WHAT ABOUT STRESS?

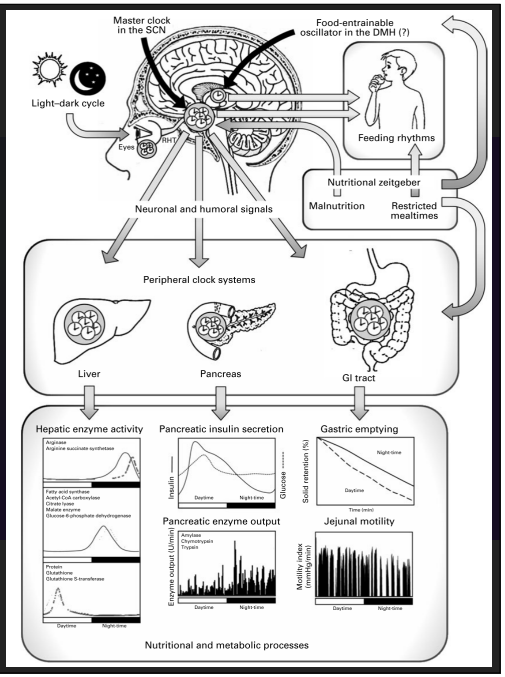
CIRCADIAN CLOCK SYSTEM IN THE CONTEXT OF THE KETOGENIC DIET



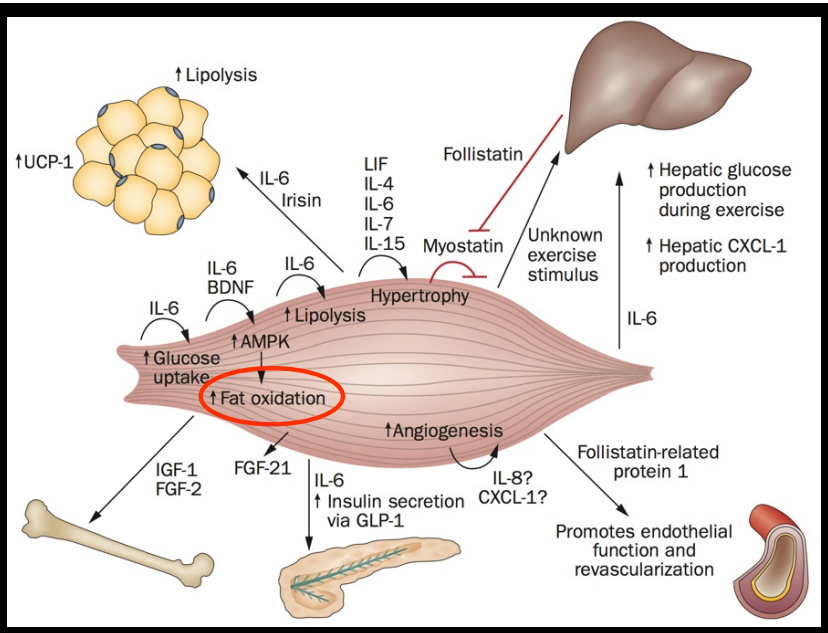
Bass, J., & Takahashi, J. S. (2010). Circadian Integration of Metabolism and Energetics. *Science*, *330*(6009), 1349–1354. doi:10.1126/science.1195027

Meet Your Biological Clock

- Pancreatic section diminishes at night
- GI motility declines at night
- Fatty acid related enzymes increase at night

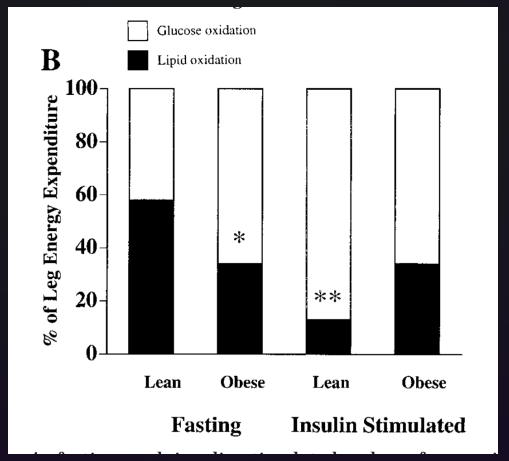


Cagampang, F. R., & Bruce, K. D. (2012). The role of the circadian clock system in nutrition and metabolism. British Journal of Nutrition, 108(03), 381–392. doi:10.1017/S0007114512002139



2012. Endocrinology, 8(8), 457–465.

Metabolic Flexibility



"WHEN THE SUPPLY OF FAT TO THE LIVER WAS INCREASED BEYOND A CRITICAL VALUE, THE RATE OF FATTY ACID OXIDATION DECLINED. UNDER THESE CIRCUMSTANCES, INTERMEDIATE METABOLITES ACCUMULATED, VERY MUCH LIKE THE SITUATION IN A TRAFFIC JAM."

Skeletal muscle fatty acid metabolism in association with insulin resistance, obesity, and weight loss

RESISTANCE + AEROBICS

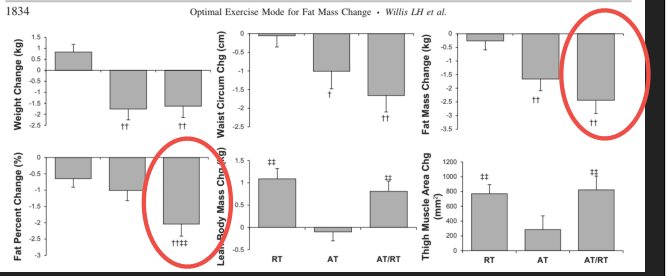
J Appl Physiol 113: 1831–1837, 2012.
First published September 27, 2012; doi:10.1152/japplphysiol.01370.2011.

Effects of aerobic and/or resistance training on body mass and fat mass in overweight or obese adults

Leslie H. Willis,[1] Cris A. Slentz,[1] Lori A. Bateman,[1] A. Tamlyn Shields,[5] Lucy W. Piner,[1] Connie W. Bales,[3,4] Joseph A. Houmard,[5] and William E. Kraus[2]

[1]*Division of Cardiology, Duke University Medical Center, Durham, North Carolina,* [2]*Duke Center for Living, Duke University Medical Center, Durham, North Carolina,* [3]*Division of Geriatric Medicine, Duke University Medical Center, Durham, North Carolina,* [4]*Geriatric Research, Education and Clinical Center, Durham Veterans Affairs Medical Center, Durham, North Carolina,* [5]*Department of Exercise and Sports Science and Human Performance Laboratory, East Carolina University, Greenville, North Car*

Submitted 7 November 2011; accepte



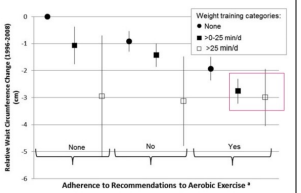
Willis LH, Slentz CA, Bateman LA, Shi Bales CW, Houmard JA, Kraus WE. Effec resistance training on body mass and fat mass ir adults. *J Appl Physiol* 113: 1831–1837, 2012. tember 27, 2012; doi:10.1152/japplphysiol.(guidelines on exercise for weight loss and include resistance training as part of the exerc few studies have compared the effects of similar a resistance training on body mass and fat mass STRRIDE AT/RT, a randomized trial, compa resistance training, and a combination of the t optimal mode of exercise for obesity reduction. sedentary, overweight or obese adults who were three 8-mo exercise protocols: *1)* RT: resista aerobic training, and *3)* AT/RT: aerobic and resi

Original Article
EPIDEMIOLOGY/GENETICS

Obesity

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]



Objective: Findings on weight training and waist circumference (WC) change ar examined prospectively whether weight training, moderate to vigorous aerobic ac ment of one activity for another were associated with favorable changes in WC an

Methods: Physical activity, WC, and BW were reported in 1996 and 2008 in a U.S. men in the Health Professionals Follow-up Study. Multiple linear regressic tution) to assess these associations were used.

Results: After adjusting for potential confounders, a significant inverse dose-resp weight training and WC change (*P*-trend <0.001) was observed. Less age-ass seen (−0.67 (−0.16 weight MVAA

Concl increase. Studies on frequency/volume of weight training and WC change are warranted.

"Substituting 20 min/day of weight training for any other discretionary activity had the strongest inverse association with wast circumference change."

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

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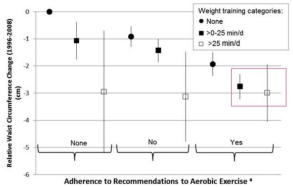

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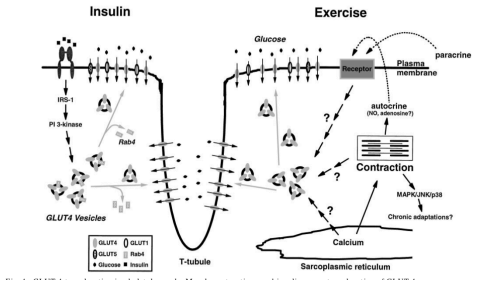
invited review

Exercise regulation of glucose transport in skeletal muscle

TATSUYA HAYASHI,[1] JØRGEN F. P. WOJTASZEWSKI,[2] AND LAURIE J. GOODYEAR[1]
[1]*Research Division, Joslin Diabetes Center, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts 02215; and* [2]*Copenhagen Muscle Research Centre, August Krogh Institute, University of Copenhagen, DK-2100 Copenhagen, Denmark*

Hayashi, Tatsuya, Jørgen F. P. Wojtaszewski and Laurie J. Goodyear. Exercise regulation of glucose t
Am. J. Physiol. 273 (*Endocrinol. Metab.*
Exercise increases the rate of glucose uptake
muscles. This effect of exercise is similar
glucose uptake, and the mechanism through
skeletal muscle glucose uptake involves t
glucose transporters to the plasma membr
Most studies suggest that exercise and insu
containing vesicles and/or mobilize differen
originating from unique intracellular loc
intracellular signaling pathways that lea
stimulated GLUT-4 translocation. Insulin ut
3-kinase-dependent mechanism, whereas
initiated by calcium release from the sarco
the activation of other signaling interm
evidence for autocrine- or paracrine-media
The period after exercise is characterized
muscle glucose uptake to insulin, which can
the face of carbohydrate deprivation. The
insulin-independent mechanisms to increas
muscle has important clinical implications,
diseases that are associated with periphera
non-insulin-dependent diabetes mellitus.



muscle contraction; glucose transporters; GLUT-4; calcium; insulin

Combining fish-oil supplements with regular aerobic exercise improves body composition and cardiovascular disease risk factors[1–3]

Alison M Hill, Jonathan D Buckley, Karen J Murphy, and Peter RC Howe

ABSTRACT
Background: Regular exercise and consuming long-chain n−3 fatty acids (FAs) from fish or fish oil can independently improve cardiovascular and metabolic health, but combining these lifestyle modifications may be more effective than either treatment alone.
Objective: We examined the individual and combined effects of n−3 FA supplements and regular exercise on body composition and cardiovascular health.
Design: Overweight volunteers [body mass index (BMI; in kg/m^2): >25] with high blood pressure, cholesterol, or triacylglycerols were randomly assigned to one of the following interventions: fish oil (FO), FO and exercise (FOX), sunflower oil (SO; control), or SO and exercise (SOX). Subjects consumed 6 g tuna FO/d (≈1.9 g n−3 FA) or 6 g SO/d. The exercise groups walked 3 d/wk for 45 min at 75% age-predicted maximal heart rate. Plasma lipids, blood pressure, and arterial function were assessed at 0, 6, and 12 wk. Body composition was assessed by dual-energy X-ray absorptiometry at 0 and 12 wk only.
Results:
HDL cho
sodilatio
0.05). Be
($P < 0.0$
Conclusi
body fat
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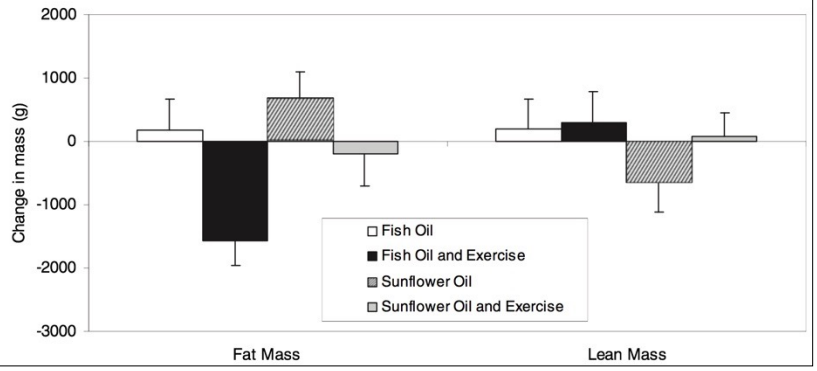
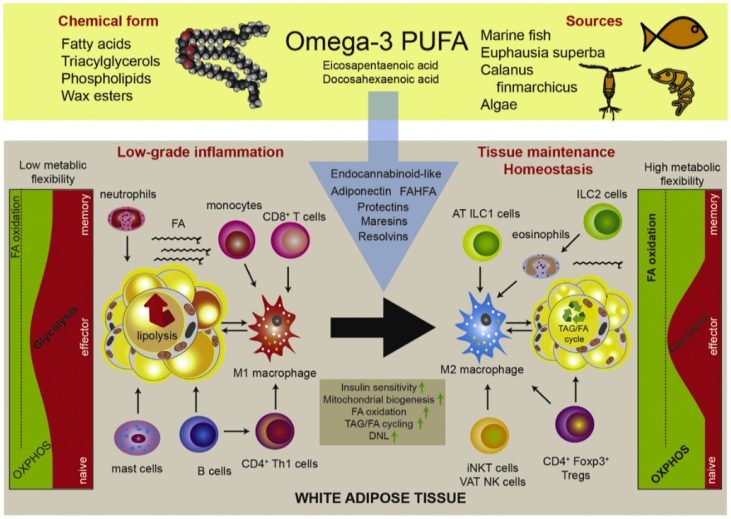
aimed at
lar disea

and the rate of mortality from coronary artery disease (4). Furthermore, regular consumption of n−3 FAs, particularly docosahexaenoic acid (DHA), has the capacity to ameliorate several cardiovascular risk factors, including elevated blood pressure and triacylglycerols, platelet aggregation, endothelial dysfunction, and arrhythmia (5). Evidence also suggests that n−3 FAs may have a favorable effect on metabolism by modulating gene expression (6). Indeed, animal studies have found that fat mass deposition is reduced after feeding with n−3 FAs (7, 8). In human studies, however, the evidence is less clear: some (9, 10) but not all studies indicate that n−3 FAs can reduce body fat with (11) or without (12) concomitant dietary restriction.

Physical activity is often recommended for weight loss, although most studies find that physical activity alone produces relatively small changes in body weight (13–15). The extent of weight loss that can be achieved through exercise may be small, but it is clear that physical activity plays a key role in preventing weight gain; however, as much as 60–90 min/d of moderate-





Mar. Drugs **2015**, *13*, 996-1009; doi:10.3390/md13020996

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Article

Effects of *n*-3 Polyunsaturated Fatty Acids (ω-3) Supplementation on Some Cardiovascular Risk Factors with a Ketogenic Mediterranean Diet

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Abstract: Background: the ketogenic diet (KD) has become a widely used nutritional approach for weight loss. Some of the KD's positive effects on metabolism and cardiovascular risk factors are similar to those seen after *n*-3 polyunsaturated fatty acids (ω-3) supplementation. We hypothesized that a ketogenic Mediterranean diet with phytoextracts combined with ω-3 supplementation may have increased positive effects on cardiovascular risk factors and inflammation. Methods: We analyzed 34 male overweight subjects; aged between 25 and 65 years who were overall healthy apart from overweight. The subjects followed a ketogenic diet protocol for four weeks; with (KDO3) or without (KD) ω-3 supplementation. Results: All subjects experienced a significant loss of body weight and body fat and there was no significant differences between treatment (body weight: