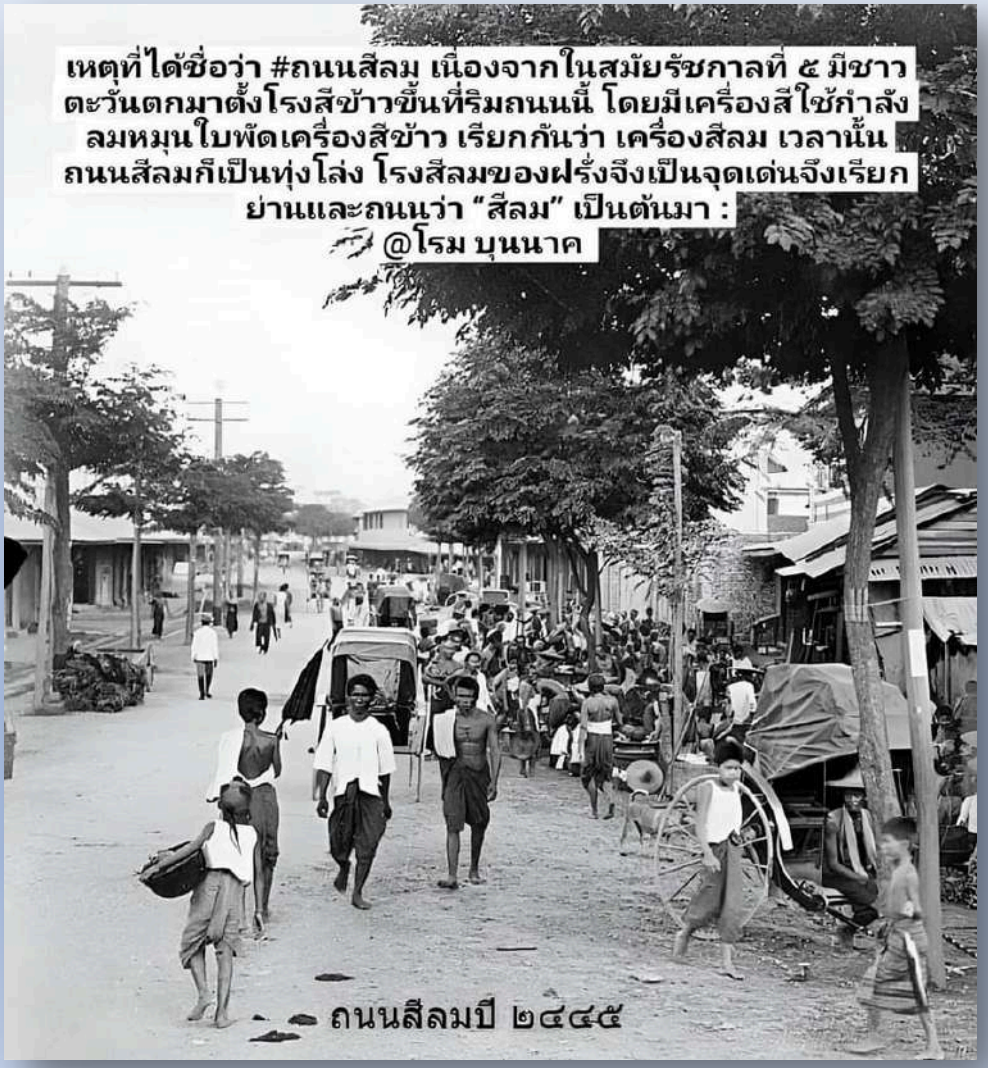
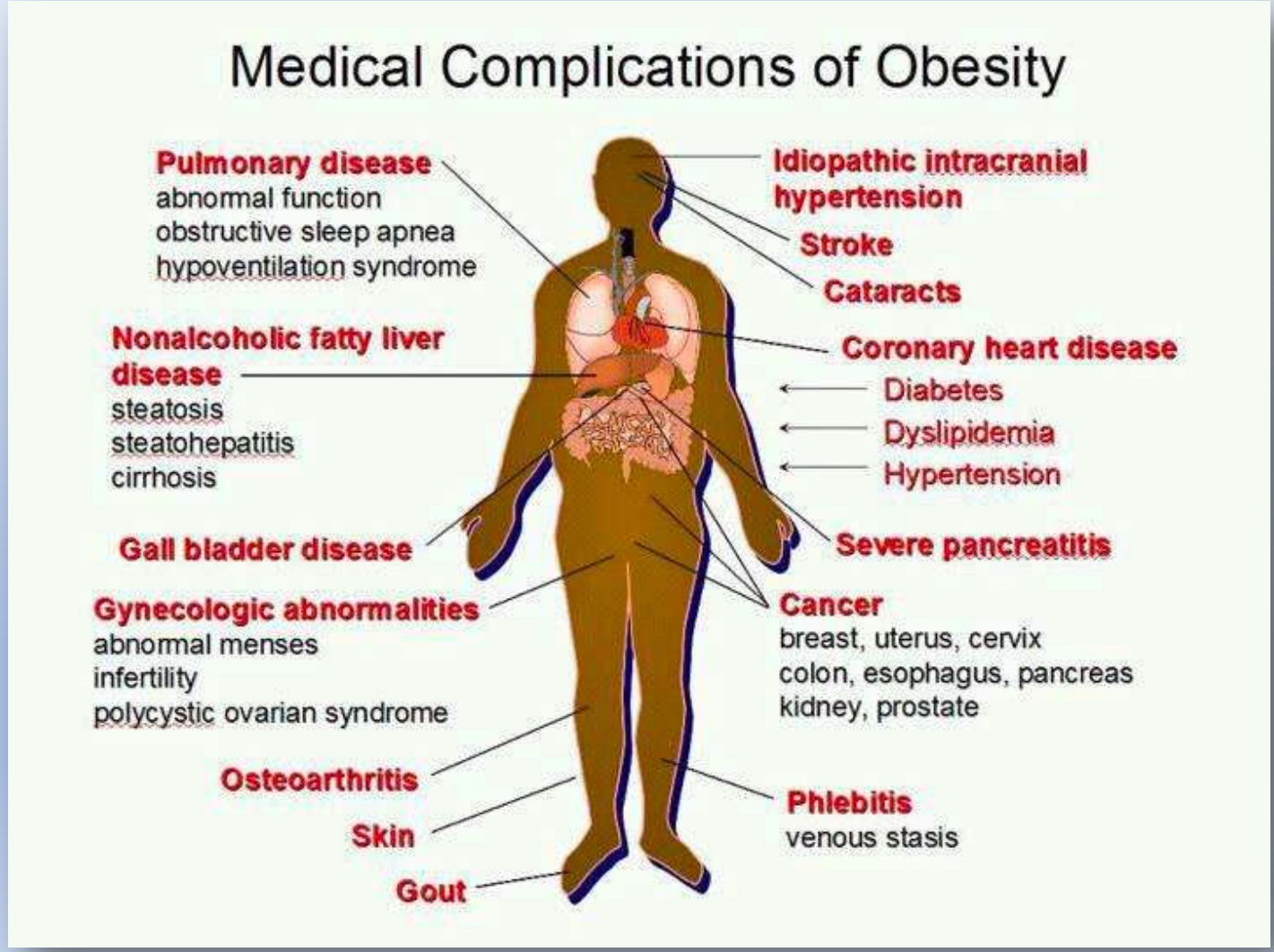
WHY ARE WE GETTING SICK?

New paradigm and New hope.

นพ.ธนศักดิ์ ยิ้มเกิด

WE SHOULD NOT BE THIS FAT AND THIS SICK.



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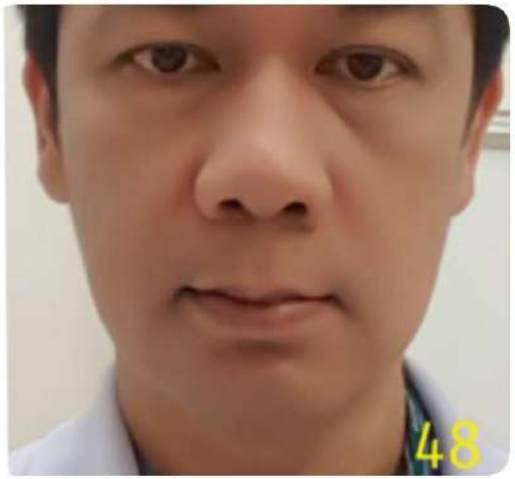
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53

นับแคลไม่เก่ง

นับแคลเก่ง

ระยะเวลา 4 เดือน ยินดีกับคุณมานะ

Requesting physician : THANASAK KHIKERD,M.D.
Date/time collected : 04 Feb 2021 07:37

TEST	RESULT	Previous Result	Previous Resul
03 Clinical Chemistry.			
Hb A1c	(04/02/21 08:51)	(03/12/20 10:41)	(28/10/20 10:00
HbA1c	5.5	6.0	7.5
Mean Blood Glucose	112	125	168
Lipid Profile	(04/02/21 08:51)	(03/12/20 10:43)	(09/09/20 11:01
Total Cholesterol	249 ↑	221	206
Triglyceride	52	75	148
HDL cholesterol : (32503)	67	50	38
LDL Cholesterol : (32504)	192 ↑	177	138

Programs	Normal	Unit	Nov-21	Jul-21	Feb-21	Nov-20	Jul-20	Feb-20	Oct-19	Jul-19	May-19	Mar-19	Feb-19	Nov-18
Cholesterol	150-250	mg/dL	191	187	200	174	192	201		183	177	183		125
Triglyceride	<150	mg/dL	34	42	100	41	56	47		63	82	81		99
HDL	>50	mg/dL	64	65	72	72	59	64		46	40	38		32
LDL	<150	mg/dL	120	114	108	94	122	128		124	121	129		73
TG/HDL	<2		0.53	0.65	1.39	0.57	0.95	0.73		1.37	2.05	2.13		3.09
LDL/HDL	<3		0.53	1.75	1.50	1.31	2.07	2.00		2.70	3.03	3.39		2.28
Che/HDL	<4		2.98	2.88	2.78	2.42	3.25	3.14		3.98	4.43	4.82		3.91
Sugar	60-100	mg/dL	78	90	86	82	75	83	98	102	132	141	139	121
HbA1c	4.8-6.0	%	5.2	5.2	5.3	5.1	5.5	5.2	5.3	5.9	6.3	6.4	6.6	7.5
Blood Pressure	< 120/80	mmHg	117/66	122/59	125/69	126/67	109/69	130/62						
Weight		kg	86.5	85	83.5	82.5	83	88.3	95.2	105.6	111.9	117.7	119.7	130

DIABETES UK
KNOW DIABETES. FIGHT DIABETES.

DIABETES REMISSION

American Diabetes Association®
Connected for Life

Diabetes remission in people with type 2 diabetes means that your blood sugar levels are healthy without needing to take any diabetes medication.

People with type 2 diabetes should be considered in remission after sustaining normal blood glucose (sugar) levels for three months or more, according to a new consensus statement from the American Diabetes Association® (ADA), the Endocrine Society, the European Association for the Study of Diabetes and Diabetes UK jointly published in *Diabetes Care*, the *Journal of Clinical Endocrinology & Metabolism*, *Diabetologia*, and *Diabetic Medicine*, respectively.

แนวทางการดูแลผู้ป่วยเบาหวาน ชนิดที่ 2
ให้เข้าสู่ โรคเบาหวานระยะสงบ
ด้วยการปรับเปลี่ยนพฤติกรรมอย่างเข้มงวด
สำหรับบุคลากรทางการแพทย์และสาธารณสุข

(Diabetes remission in type 2 diabetes with intensive lifestyle intervention guide for healthcare providers)

สมาคมแพทย์เวชปฏิบัติทั่วไป/เวชศาสตร์ครอบครัวแห่งประเทศไทย ราชวิทยาลัยแพทย์เวชศาสตร์ครอบครัวแห่งประเทศไทย สมาคมโรคเบาหวานแห่งประเทศไทยฯ สมาคมผู้ให้ความรู้โรคเบาหวาน สมาคมต่อมไร้ท่อแห่งประเทศไทย สมาคมนักกำหนดอาหารแห่งประเทศไทย สมาคมผู้ให้อาหารทางหลอดเลือดดำและทางเดินอาหารแห่งประเทศไทย กองโรคไม่ติดต่อ กรมควบคุมโรค กระทรวงสาธารณสุข และคณะทำงานผู้เชี่ยวชาญ

แนวทางการดูแลผู้ป่วยเบาหวาน ชนิดที่ 2
ให้เข้าสู่โรคเบาหวานระยะสงบ
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(Diabetes remission in type 2 diabetes with intensive lifestyle intervention guide for healthcare providers)

ข้อมูลทางบรรณานุกรมของสำนักหอสมุดแห่งชาติ
National Library of Thailand Cataloging in Publication Data

แนวทางการดูแลผู้ป่วยเบาหวาน ชนิดที่ 2 ให้เข้าสู่โรคเบาหวานระยะสงบด้วยการ ปรับเปลี่ยนพฤติกรรมอย่างเข้มงวดสำหรับบุคลากรทางการแพทย์ และสาธารณสุข.-- กรุงเทพฯ : ราชวิทยาลัยแพทย์ เวชศาสตร์ครอบครัวแห่งประเทศไทย, 2565.

26 หน้า.
1. ผู้ป่วยเบาหวาน -- การดูแล. 2. เบาหวาน -- การดูแล. I. ชื่อเรื่อง.

616.462
ISBN (ebook) 978-616-92988-3-0

บรรณาธิการ : คณะทำงานพัฒนาแนวทางการรักษาผู้ป่วยโรคเบาหวานชนิดที่ 2 รายใหม่ให้เข้าสู่ระยะสงบของโรคด้วยการปรับเปลี่ยนพฤติกรรมร่วมกับการตรวจระดับน้ำตาลในเลือดด้วยตนเอง

จัดทำรูปเล่ม : รศ.นพ.กรภัทร มยุระสาคร
พัชรินทร์ โพธิ์ทอง (พีพี มีเดีย ดีไซน์ แอนด์ พริ้นท์)
โทรศัพท์ 08 8422 5940

ออกแบบปก : พัชรินทร์ โพธิ์ทอง

พิมพ์ครั้งที่ 1 : พฤศจิกายน 2565

จัดทำและเผยแพร่โดย
ราชวิทยาลัยแพทย์เวชศาสตร์ครอบครัวแห่งประเทศไทย
เลขที่ 2 ชั้น 11 อาคารเฉลิมพระบารมี ๕๐ ปี ซอยศูนย์วิจัย
แขวงบางกะปิ เขตห้วยขวาง กรุงเทพมหานคร 10310
https://www.facebook.com/RCFPT/

สอบถามข้อมูลได้ที่
02-716-6651-2
02-716-6653
info@thaifammed.org

กรมควบคุมโรค
Department of Disease Control

สำนักงานป้องกันควบคุมโรคที่ 9 จังหวัดนครราชสีมา

ประชุมเชิงปฏิบัติการพัฒนาศักยภาพบุคลากรทางการแพทย์ และการสาธารณสุข ในการเข้าถึง ป้องกัน ควบคุมโรคเบาหวานชนิดที่ 2 ให้เข้าสู่โรคเบาหวานระยะสงบ ด้วยการปรับเปลี่ยนพฤติกรรมอย่างเข้มงวด (DM Remission)

ระหว่างวันที่ 13 – 14 กุมภาพันธ์ 2567 เวลา 08.00 - 20.00 น.

กลุ่มเป้าหมาย

ทีมสหวิชาชีพ : แพทย์ทั่วไปหรือหมอครอบครัว,หัวหน้ากลุ่มภารพยาบาล, หัวหน้าคลินิก NCDs, เภสัชกร, พยาบาลประจำคลินิก NCDs, พยาบาลรับผิดชอบงาน NCDs สสอ./รพสต. และ นักโภชนาการ/นักโภชนากร รวม 7 ท่าน/ทีม

รุ่นที่ 1 ทีมสหวิชาชีพจังหวัดนครราชสีมาและจังหวัดชัยภูมิ
สถานที่ : ณ ห้องประชุมโรงแรมสีมาธานี อำเภอเมืองนครราชสีมา จังหวัดนครราชสีมา

รุ่นที่ 2 ทีมสหวิชาชีพจังหวัดบุรีรัมย์และจังหวัดสุรินทร์
สถานที่ : ณ ห้องประชุมโรงแรมเซ็นทารา โคราช อำเภอเมืองนครราชสีมา จังหวัดนครราชสีมา

ประธานเปิดโครงการประชุม

นายแพทย์ภูวเดช สุระโคตร
ผู้ตรวจราชการกระทรวงสาธารณสุข เขตสุขภาพที่ 9

กล่าวรายงานและวัตถุประสงค์ของโครงการฯ

นายแพทย์ทวีชัย วิษณุโยธิน
ผู้อำนวยการ
สำนักงานป้องกันควบคุมโรคที่ 9 จังหวัดนครราชสีมา

วิทยากร

นายแพทย์ธนศักดิ์ ยิ้มเกิด
ที่ปรึกษาโครงการรักษาเบาหวานเข้าสู่ภาวะสงบของ สปสช. / DietDoctor Thailand

นายแพทย์ภูวดล พลพวก
รองนายแพทย์สสจ.พิษณุโลก
ด้านเวชกรรมป้องกัน
และผู้อำนวยการโรงพยาบาลบางระกำ

นายแพทย์ชัชวาล ลีลาเจริญพร
ข้าราชการบำนาญ

ทีมวิทยากร โรงเรียนเบาหวานภูวดลโมเดล

ทีมวิทยากร โรงเรียนเบาหวานวิทยา อำเภอพิมาย

ประโยชน์ที่จะได้รับ :
บุคลากรทางการแพทย์ การสาธารณสุข มีความรู้เรื่องการเฝ้าระวัง ป้องกันควบคุมโรคเบาหวานชนิดที่ 2 ให้เข้าสู่โรคเบาหวานระยะสงบ ด้วยการปรับเปลี่ยนพฤติกรรมอย่างเข้มงวด (DM Remission) สามารถนำไปใช้ในการรักษาผู้ป่วยโรคเบาหวานในคลินิกได้

เนื้อหา :
รูปแบบการอบรมมีทั้งภาคทฤษฎีและภาคปฏิบัติ โดยวิทยากรที่มีความรู้ความเชี่ยวชาญและประสบการณ์ที่เกี่ยวข้องกับการดูแลรักษาผู้ป่วยเบาหวานชนิดที่ 2 ให้เข้าสู่โรคเบาหวานระยะสงบ

ประกาศนียบัตร :
ผู้ผ่านการอบรมได้รับประกาศนียบัตร (ในรูปแบบไฟล์)

กลุ่ม LINE REMISSION R9

กรอกแบบฟอร์มส่งรายชื่อผู้เข้าร่วมอบรม

หนังสือเชิญอบรมรุ่นที่ 1

หนังสือเชิญอบรมรุ่นที่ 2

ติดต่อสอบถาม
สำนักงานป้องกันควบคุมโรคที่ 9 จังหวัดนครราชสีมา (สคร.9) เลขที่ 529 หมู่ 9
ถนนราชสีมา-โชคชัย ตำบลหนองบัวศาลา อำเภอเมืองนครราชสีมา จังหวัดนครราชสีมา 30000
กลุ่มโรคไม่ติดต่อ งาน NCDs โทร.0 4421 2900 ต่อ 115
นางนำริชาติ จิตกลาง พยาบาลวิชาชีพชำนาญการ โทรศัพท์ 09 3662 8899
นางสาวพัดชา พันธุ์สุข นักวิชาการสาธารณสุขปฏิบัติการ โทรศัพท์ 06 4465 5511

กรมควบคุมโรค
Department of Disease Control

ข่าวประชาสัมพันธ์

สำนักงานป้องกันควบคุมโรคที่ 9 นครราชสีมา
The Office of Disease Prevention and Control 9th Nakhon Ratchasima

สคร.9 นครราชสีมา ประชุมเชิงปฏิบัติการพัฒนาศักยภาพบุคลากรทางการแพทย์และเจ้าหน้าที่สาธารณสุข ในการเข้าถึงป้องกันควบคุมโรคเบาหวานชนิดที่ 2 ให้เข้าสู่โรคเบาหวานระยะสงบ ด้วยการปรับเปลี่ยนพฤติกรรมอย่างเข้มงวด (DM Remission) รุ่นที่ 1 และรุ่นที่ 2

วันที่ 13-14 กุมภาพันธ์ พ.ศ.2567 สำนักงานป้องกันควบคุมโรคที่ 9 จังหวัดนครราชสีมา โดยกลุ่มโรคไม่ติดต่อ จัดประชุมเชิงปฏิบัติการพัฒนาศักยภาพบุคลากรทางการแพทย์ และเจ้าหน้าที่สาธารณสุข ในการเข้าถึง ป้องกันควบคุมโรคเบาหวานชนิดที่ 2 ให้เข้าสู่โรคเบาหวานระยะสงบ ด้วยการปรับเปลี่ยนพฤติกรรมอย่างเข้มงวด (DM Remission) โดยได้รับเกียรติจาก นายแพทย์ภูวเดช สุระโคตร ผู้ตรวจราชการกระทรวงสาธารณสุข เขตสุขภาพที่ 9 เป็นประธานการประชุม กล่าวรายงานโดย นายแพทย์ทวีชัย วิษณุโยธิน ผู้อำนวยการสำนักงานป้องกันควบคุมโรคที่ 9 จังหวัดนครราชสีมา ซึ่งมีวัตถุประสงค์เพื่อพัฒนาศักยภาพบุคลากรทางการแพทย์ สาธารณสุข ในการเฝ้าระวัง ป้องกันควบคุมโรคเบาหวานชนิดที่ 2 ให้เข้าสู่โรคเบาหวานระยะสงบ ด้วยการปรับเปลี่ยนพฤติกรรมอย่างเข้มงวด (DM Remission) วิทยากรประกอบไปด้วย วิทยากรจากทีมโรงเรียนเบาหวานวิทยา อำเภอพิมาย นำโดยนายแพทย์ชัชวาล ลีลาเจริญพร วิทยากร Diet Doctor Thailand นายแพทย์ ธนศักดิ์ ยิ้มเกิด และทีมวิทยากรภูวดลโมเดล นำโดย นายแพทย์ภูวดล พลพวก ผู้อำนวยการโรงพยาบาลบางระกำ จังหวัดพิษณุโลก กลุ่มเป้าหมายที่เข้าร่วมประชุม ได้แก่ แพทย์/หมอครอบครัว/เภสัชกร/พยาบาล/นักวิชาการสาธรณสุข/นักโภชนาการ/โภชนากร รุ่นที่ 1 จัดประชุมฯ ณ ห้องประชุมโรงแรมสีมาธานี อำเภอเมือง จังหวัดนครราชสีมา มีผู้เข้าร่วมประชุม 250 คน และรุ่นที่ 2 จัดประชุมฯ ณ ห้องประชุมโรงแรมเซ็นทารา โคราช อำเภอเมือง จังหวัดนครราชสีมา มีผู้เข้าร่วมประชุม 200 คน

FACEBOOK WEBSITE

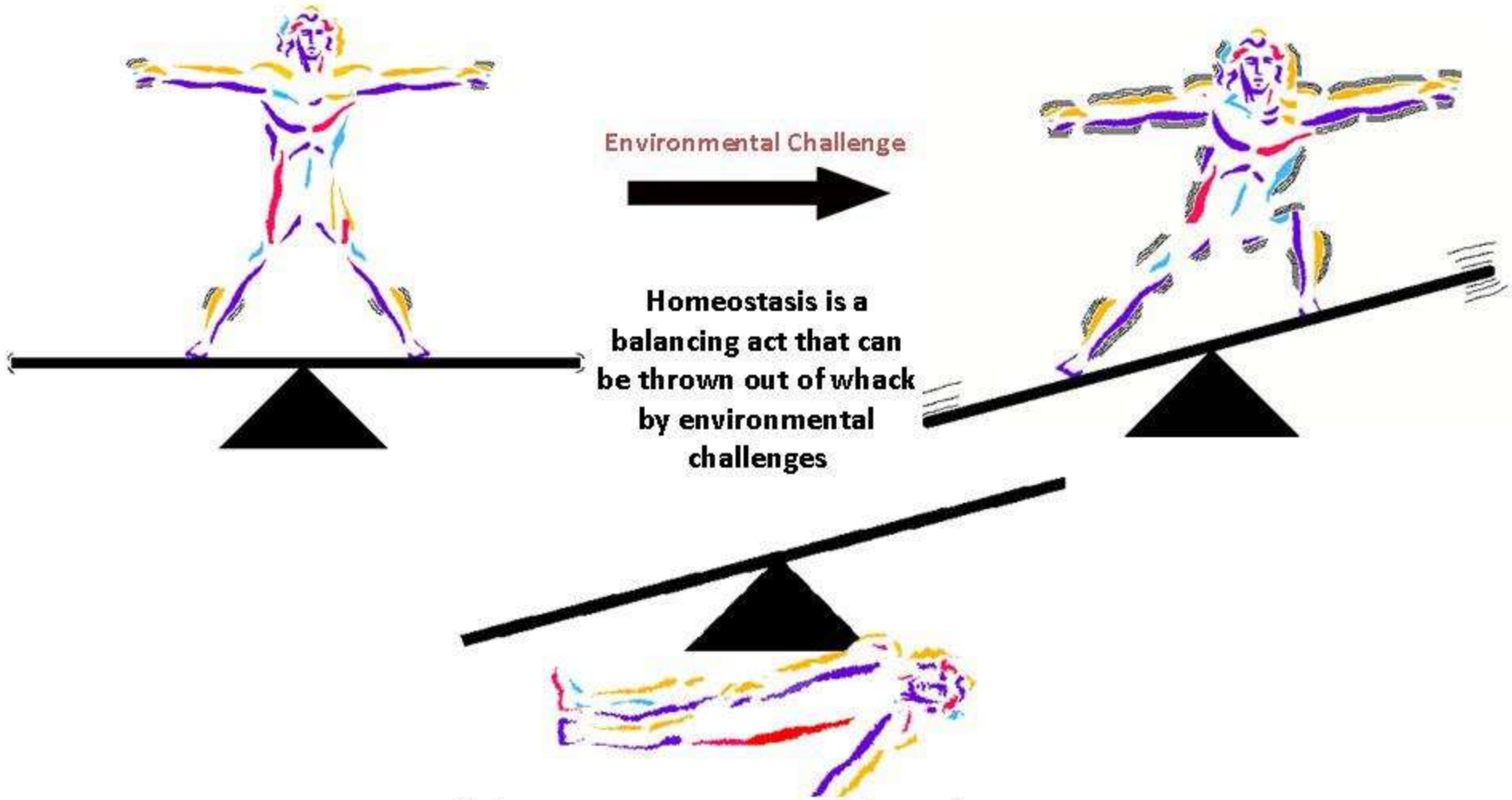
กลุ่มสื่อสารความเสี่ยงโรคและภัยสุขภาพ
สำนักงานป้องกันควบคุมโรคที่ 9 นครราชสีมา
ถนนราชสีมา-โชคชัย ตำบลหนองบัวศาลา อำเภอเมือง จังหวัดนครราชสีมา 30000
0 4421 2900 ต่อ 305 0 4421 8018

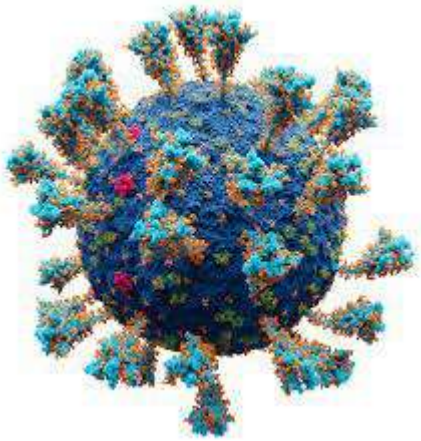
1422
สายด่วนกรมควบคุมโรค

WHY ARE WE GETTING SICK?

New paradigm and New hope.

นพ.ธนศักดิ์ ยิ้มเกิด


Environmental Challenge
Homeostasis is a balancing act that can be thrown out of whack by environmental challenges
If the system cannot restore balance, it can lead to death! (Credit: tollecausam.com)



ไข้
ไอ
เจ็บคอ
น้ำมูก
ปวดกล้ามเนื้อ

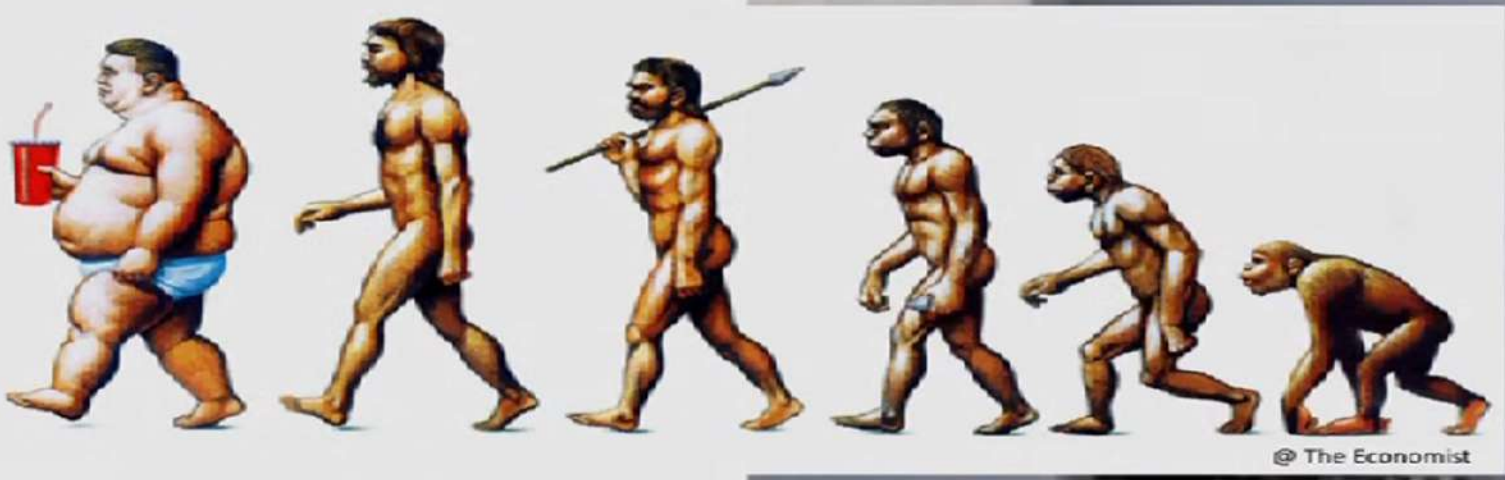
Health → Sick

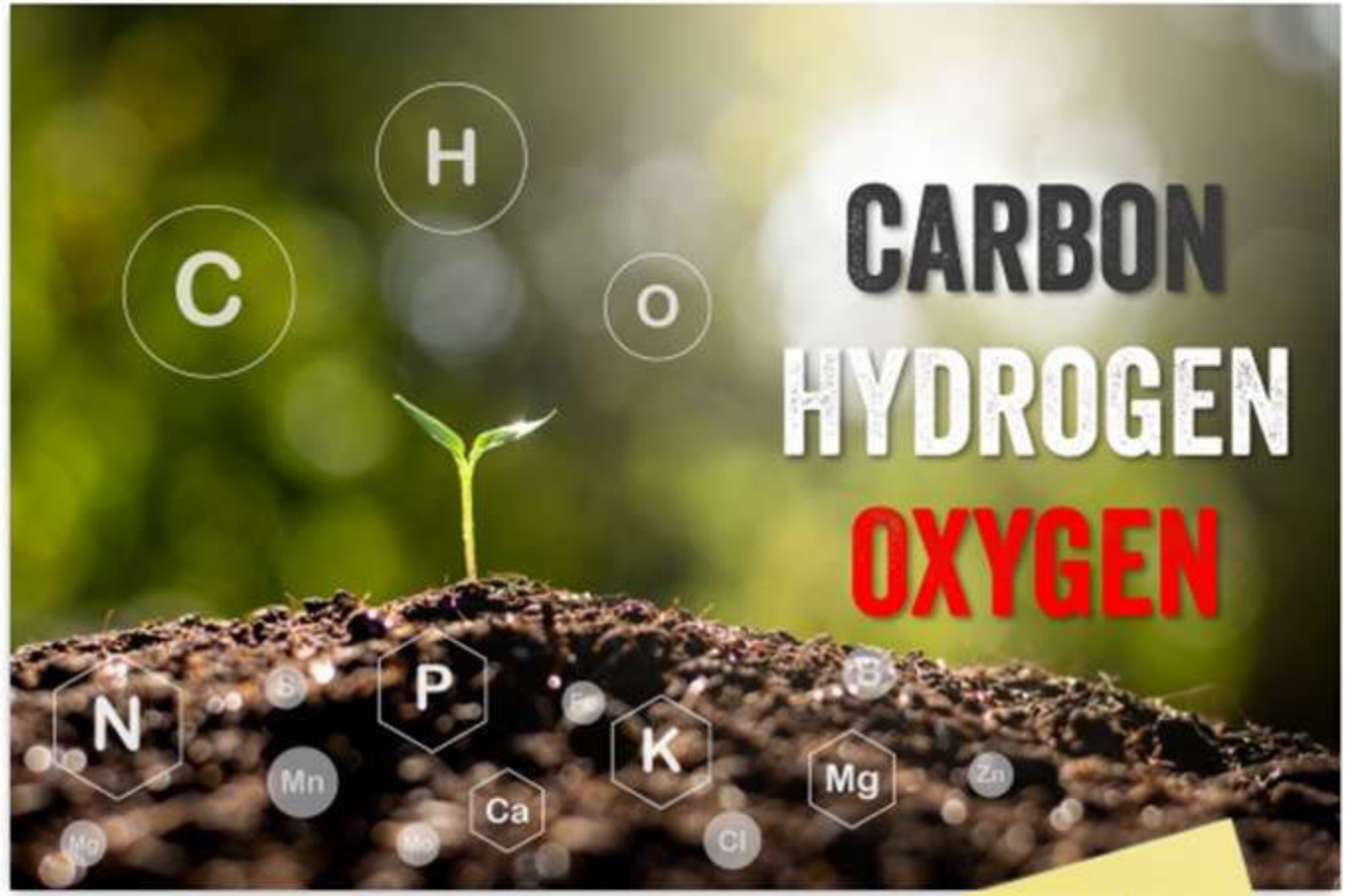
สาเหตุ → ผลกระทบต่อสุขภาพ → อาการแสดง

Diet & Lifestyle →

นน.ตัว
ความดัน
น้ำตาลในเลือด
ผลตรวจเลือดเปลี่ยนไป
เส้นเลือดอุดตัน
ศักยภาพของร่างกาย
มะเร็ง เป็นต้น

Human Diet
And
Why we get sick?

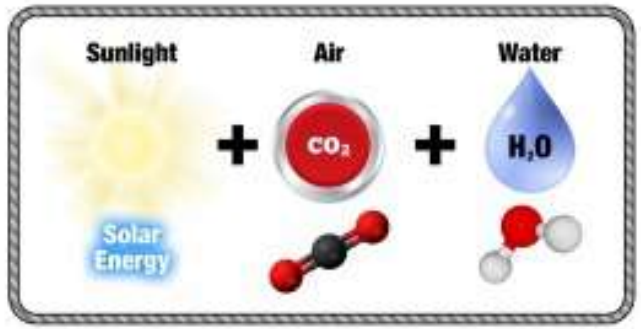


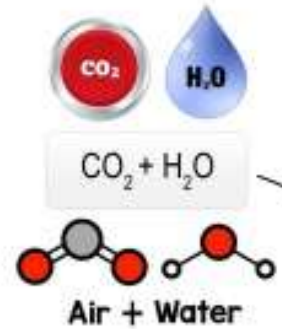


CARBON, HYDROGEN, AND OXYGEN ARE THE THREE MAIN ELEMENTS OF LIFE.

PLANTS GET THESE FROM AIR AND WATER.

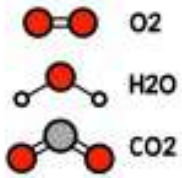
All dietary energy is solar energy, captured by plants, as the high-energy carbon-carbon and carbon-hydrogen bonds in carbohydrates and hydrocarbons (carbohydrates and fats).





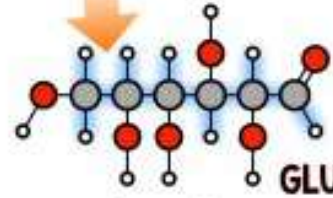
Plants store solar energy using the three main elements of life: carbon, hydrogen, and oxygen.

- Carbon
- Hydrogen
- Oxygen



Energy-containing carbon-carbon and carbon-hydrogen bonds

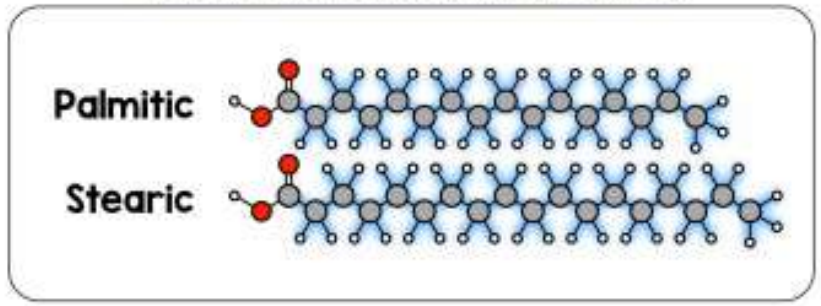
Solar Energy stored as high-energy bonds



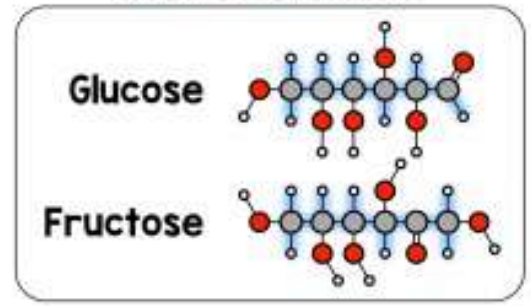
GLUCOSE

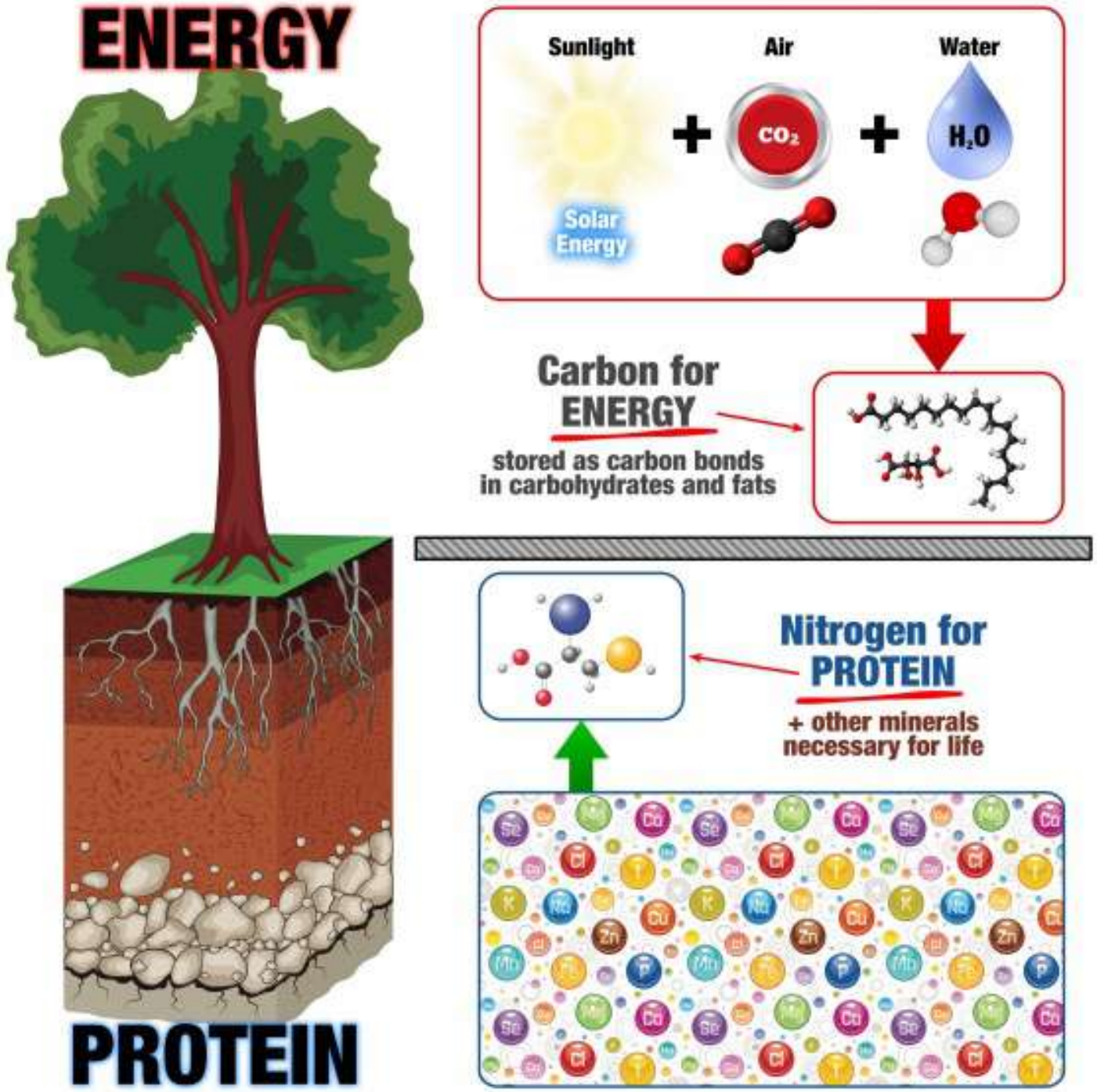
Photosynthesis

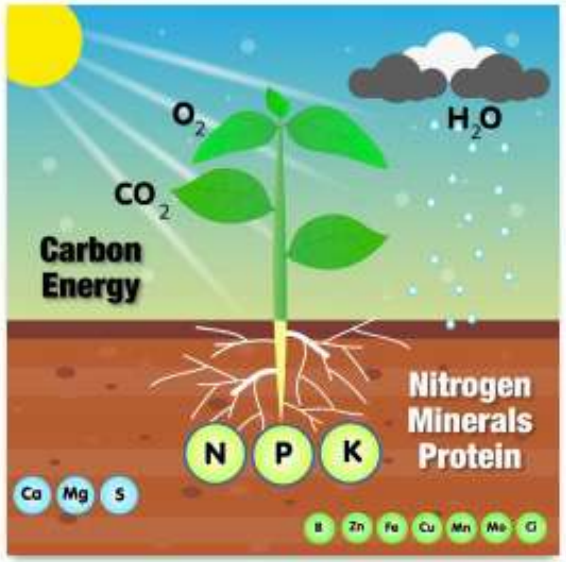
HYDROCARBONS (FATTY ACIDS)



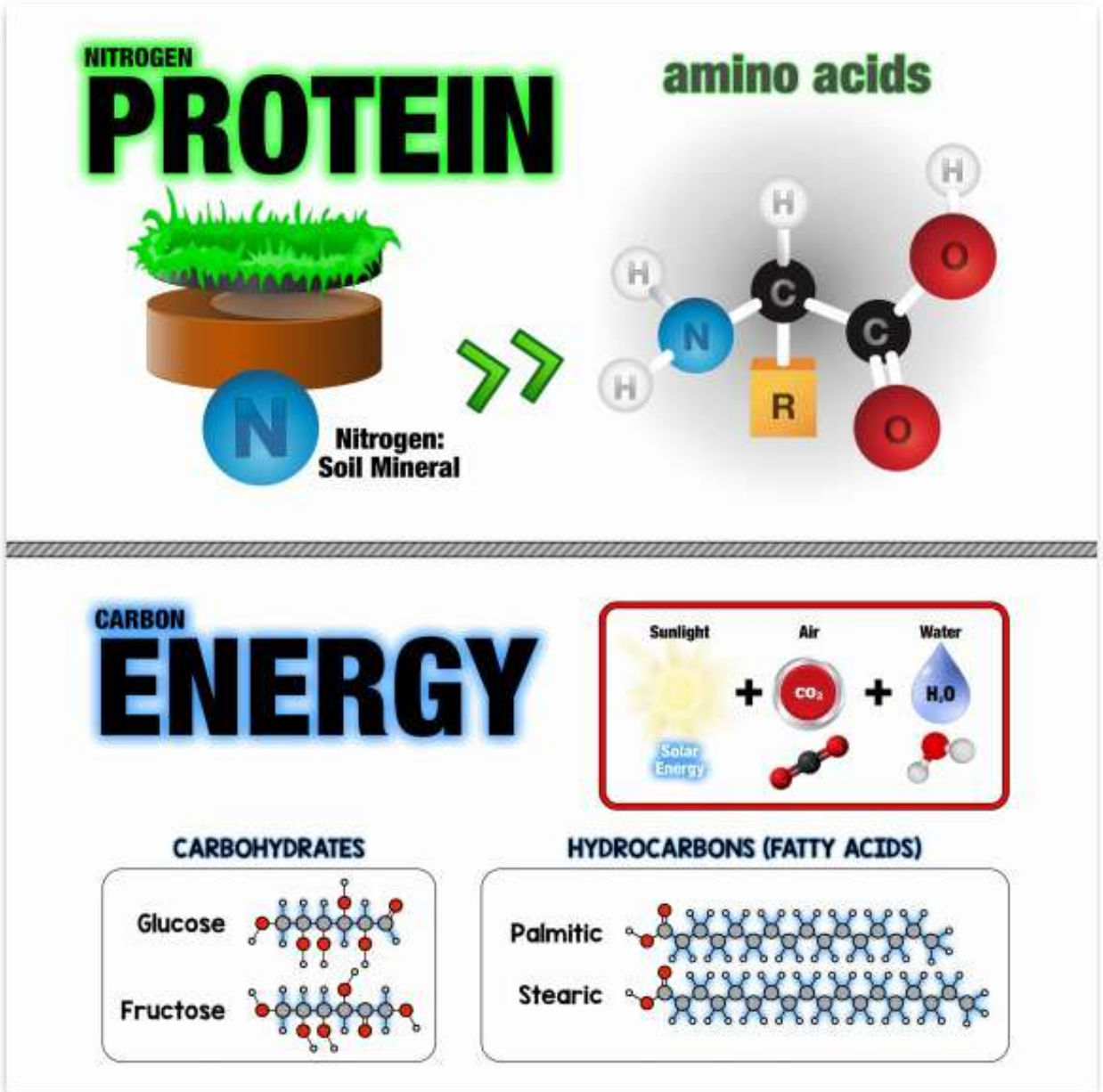
CARBOHYDRATES



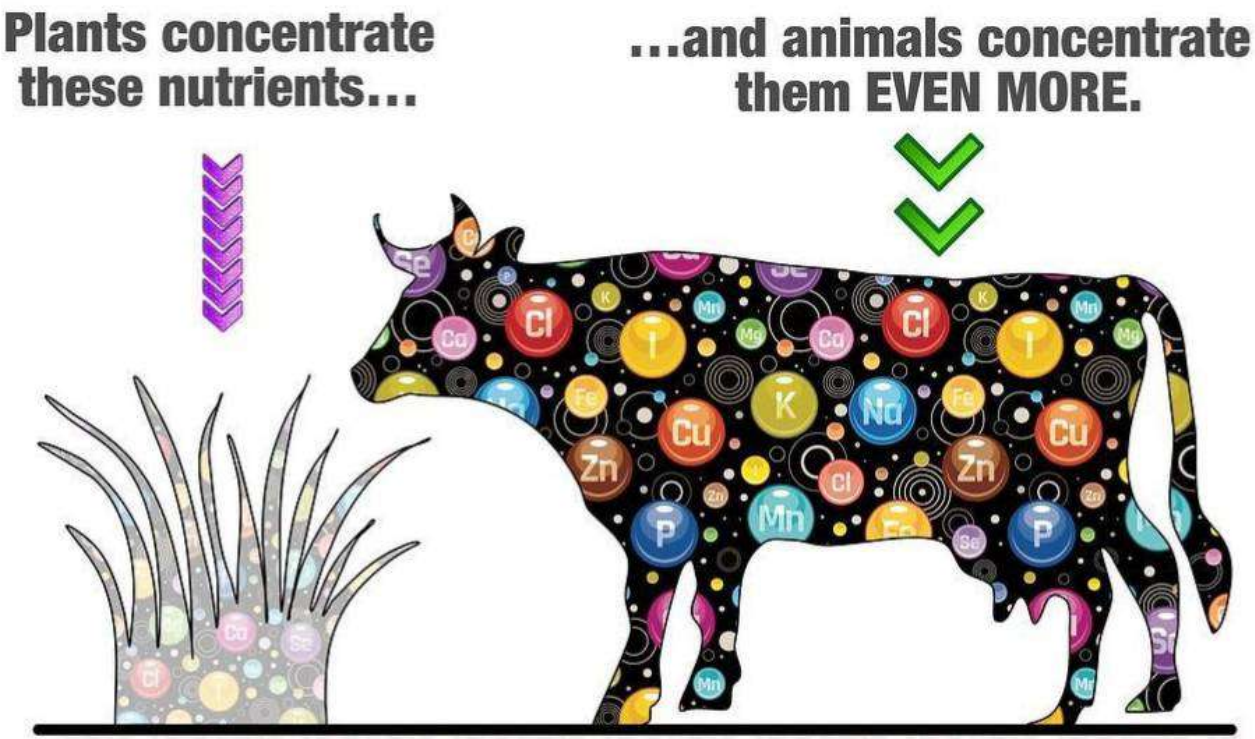

ENERGY
Sunlight
Air
Water
CO_2
H_2O
Solar Energy
Carbon for ENERGY
stored as carbon bonds in carbohydrates and fats
Nitrogen for PROTEIN
+ other minerals necessary for life
PROTEIN

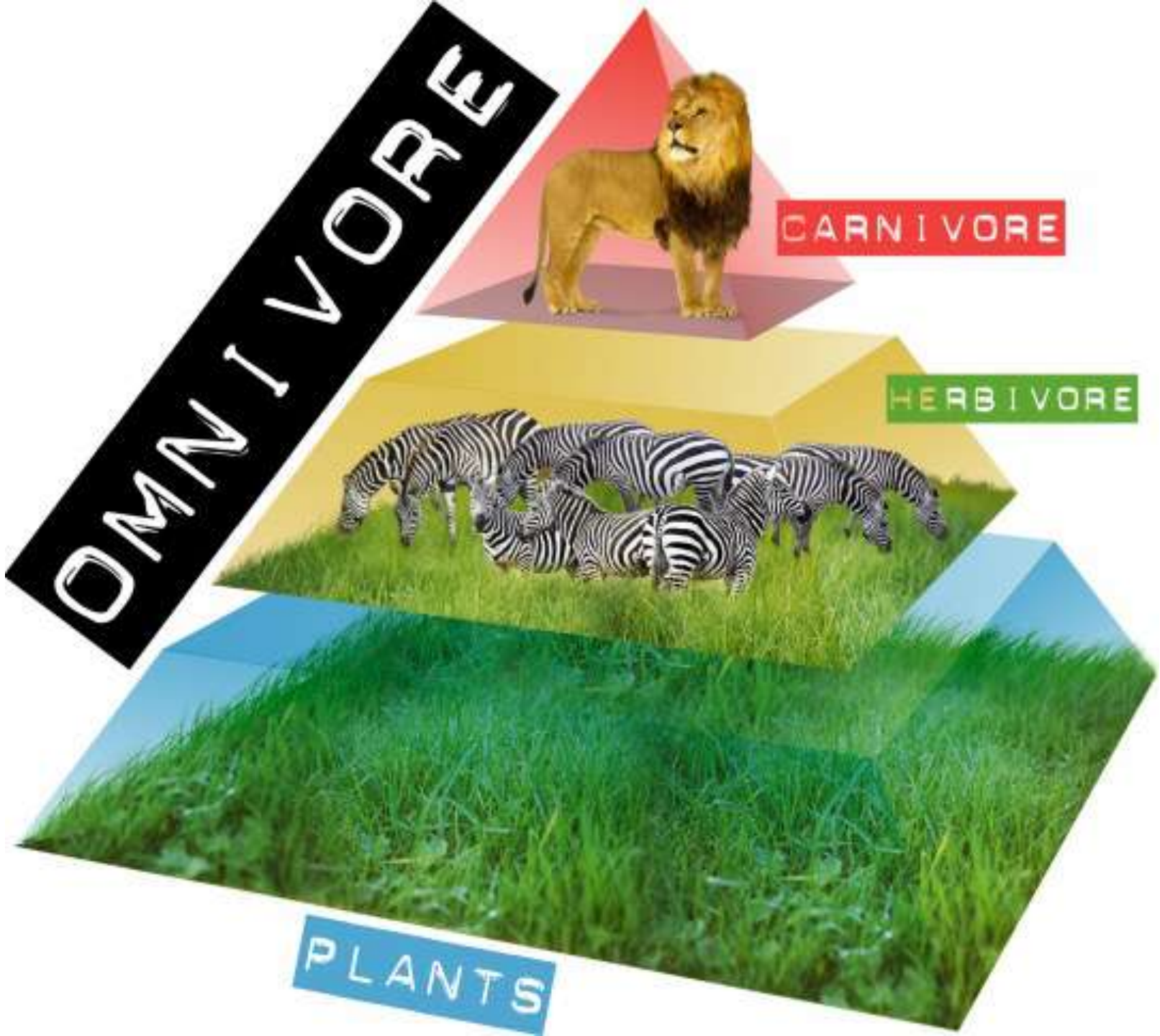


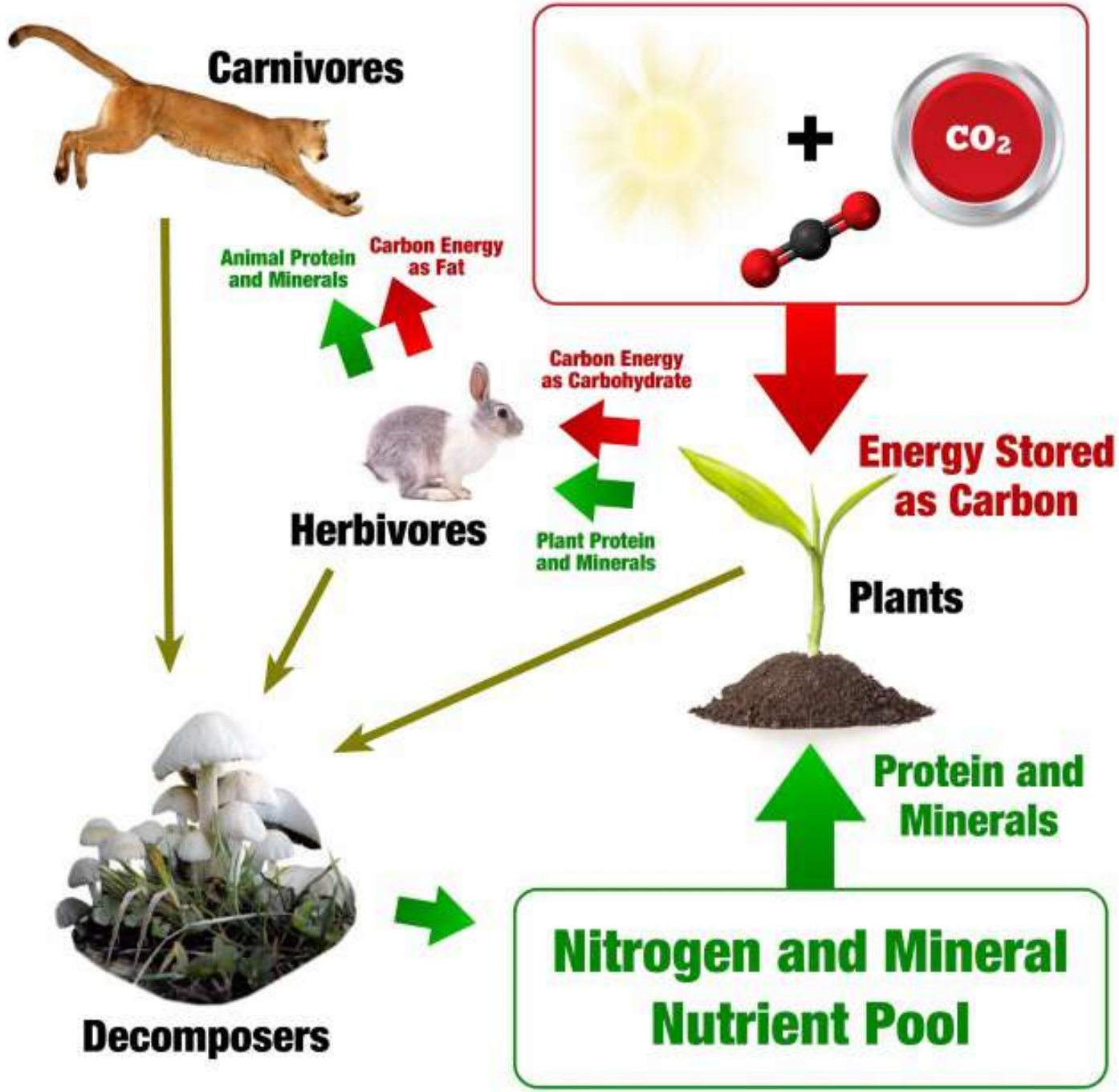
Your body, as well, has a protein quantity and an energy quantity. Your basic body composition goal should be to achieve the HIGHEST lean mass at the LOWEST fat mass, so the protein to energy ratio of your body is going to be an important concept going forward.

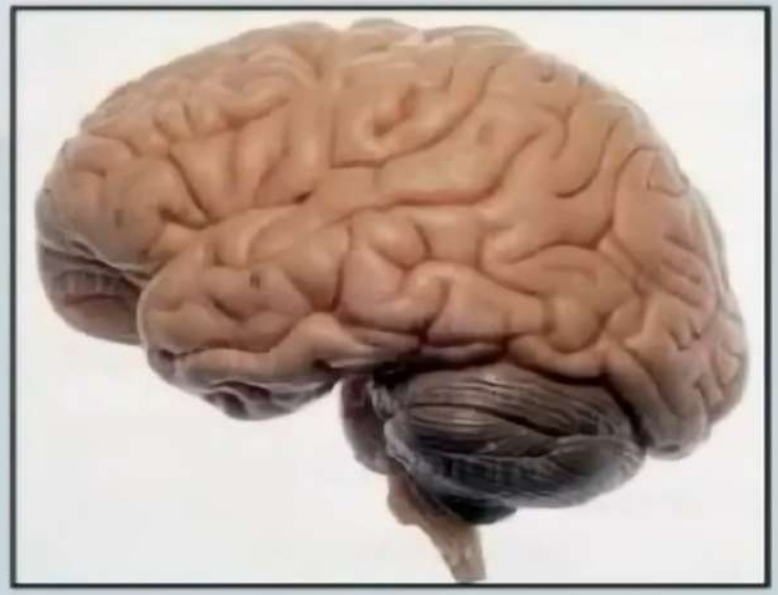


All life on earth depends on nitrogen and minerals from the nutrient pool (soil etc).




OMNIVORE
CARNIVORE
HERBIVORE
PLANTS

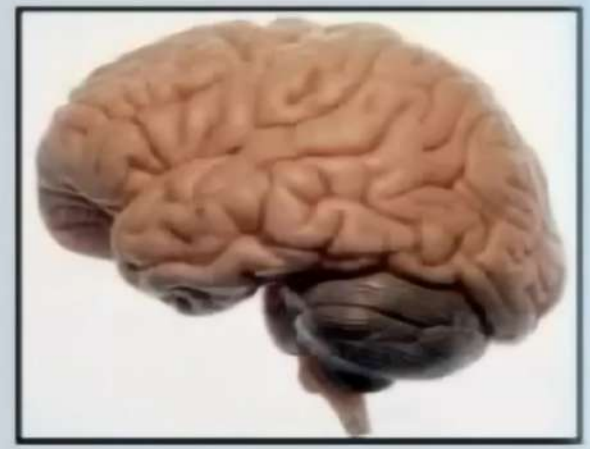

Carnivores
Animal Protein and Minerals
Carbon Energy as Fat
Carbon Energy as Carbohydrate
Herbivores
Plant Protein and Minerals
Energy Stored as Carbon
Plants
Protein and Minerals
Decomposers
Nitrogen and Mineral Nutrient Pool



Why Did It Happen?

Where Does Energy For Brain Come From?

- **Our total energy intake is in accordance with Kleiber's Law**
- Brain is ~2% of total body weight
- Brain uses ~20-25% of total resting energy
- Small gut to compensate

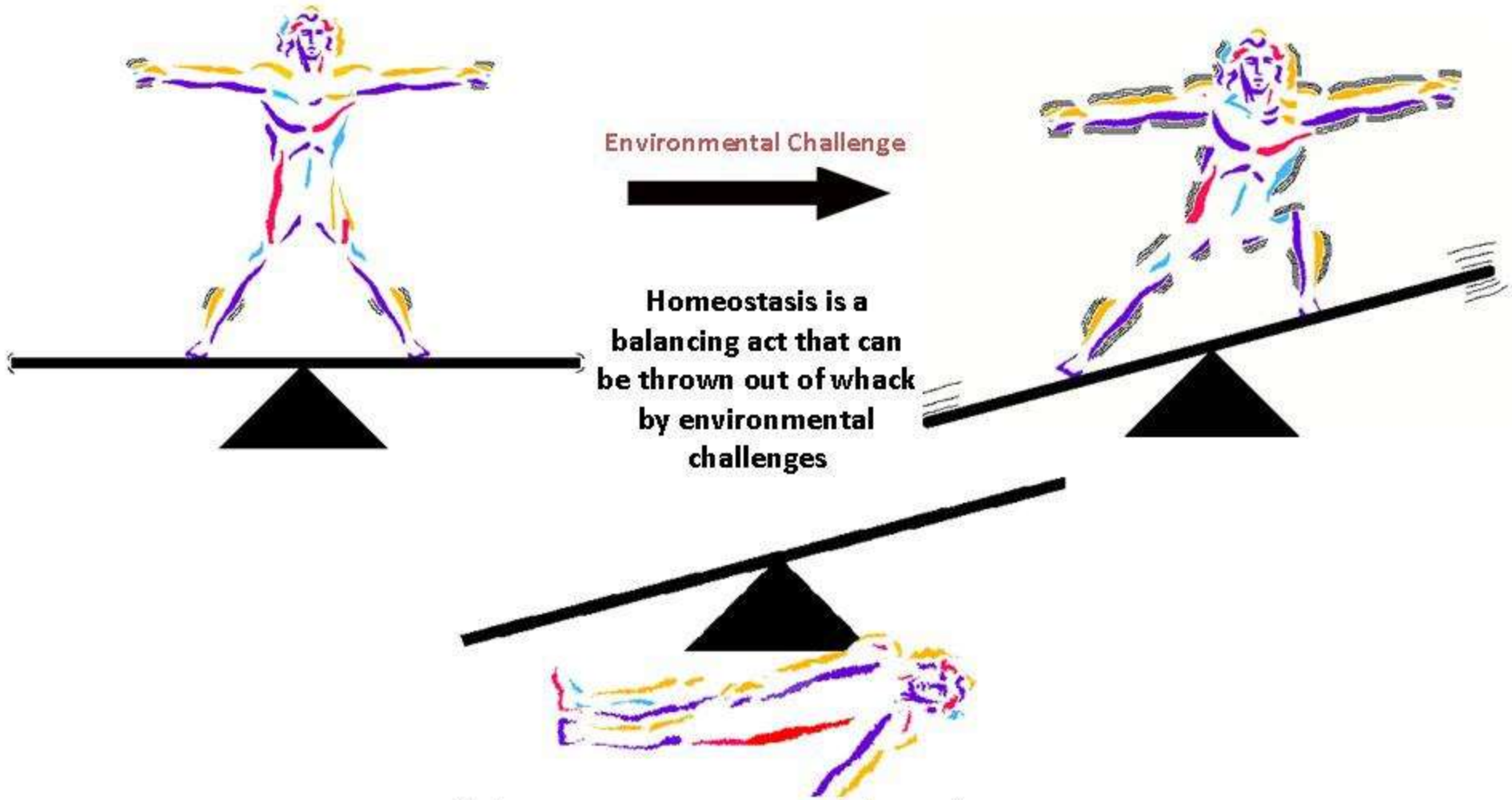


- Very nutrient and energy dense diet needed
- Fat is the only macronutrient
- Animals the only practical source

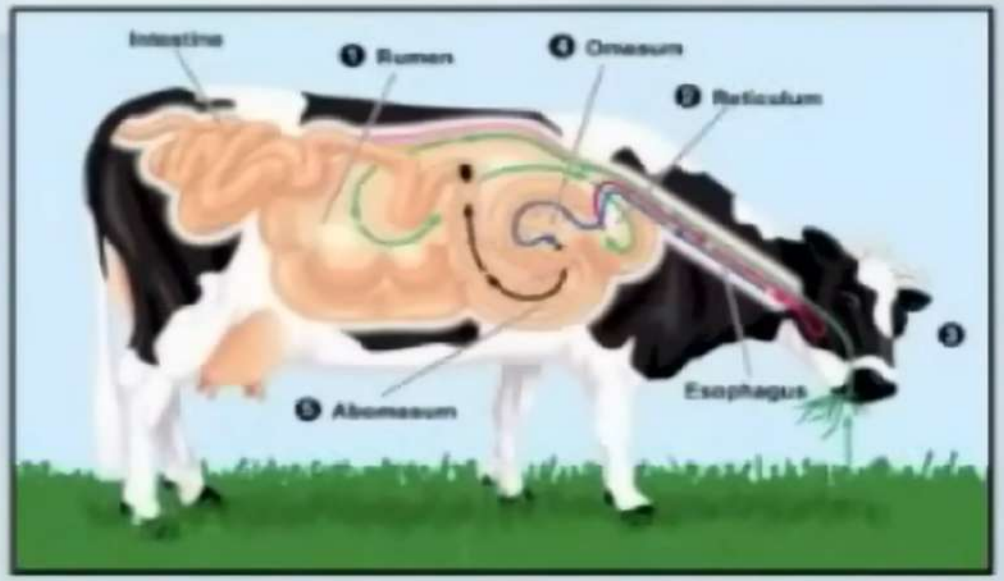
Vegetable Fats Are Not Suitable

- **20** and **22** carbon fatty acids (AA, DTA, EPA, DHA) are essential for brain development – found only in animal foods
- Longest chain in vegetable oils – linoleic acid (n-6) and alpha-linolenic acid (n-3) are **18** carbon fatty acids
- **Obligate carnivores and humans "maintain an inefficient ability to chain elongate and desaturate 18 carbon fatty acids to their product 20 and 22 carbon fatty acids . . ."**
- **". . . preformed dietary 20 and 22 carbon fatty acids (found only in animal foods) were increasingly incorporated in lieu of endogenously synthesized fats derived from 18 carbon plant fatty acids."**
- **Our brain growth could never have happened without these fats**

Emken, R.A., Adlof, R.O., Rohwedder, W.K., and Gulley, R.M., Comparison of linolenic and linoleic acid metabolism in man: Influence of dietary linoleic acid. In: Sinclair, A., and Gibson, R. (Eds.), *Essential Fatty Acids and Eicosanoids*. Invited Papers from the Third International Conference. AOCS Press, Champaign IL: 1992, pp. 23–25.


Environmental Challenge
Homeostasis is a balancing act that can be thrown out of whack by environmental challenges
If the system cannot restore balance, it can lead to death! (Credit: tollecausam.com)

Nutrient Absorption And Utilisation In Ruminants



'Foregut digesters'

All proteins, carbs and fibre fermented in the stomach

http://vetmedicine.about.com/

Nutrient Absorption And Utilisation In Ruminants

"[short chain fatty acids] . . . are of paramount importance in that they provide **greater than 70%** of the ruminant's energy supply."

70-80% kcals fat (saturated)
20-30% kcals protein

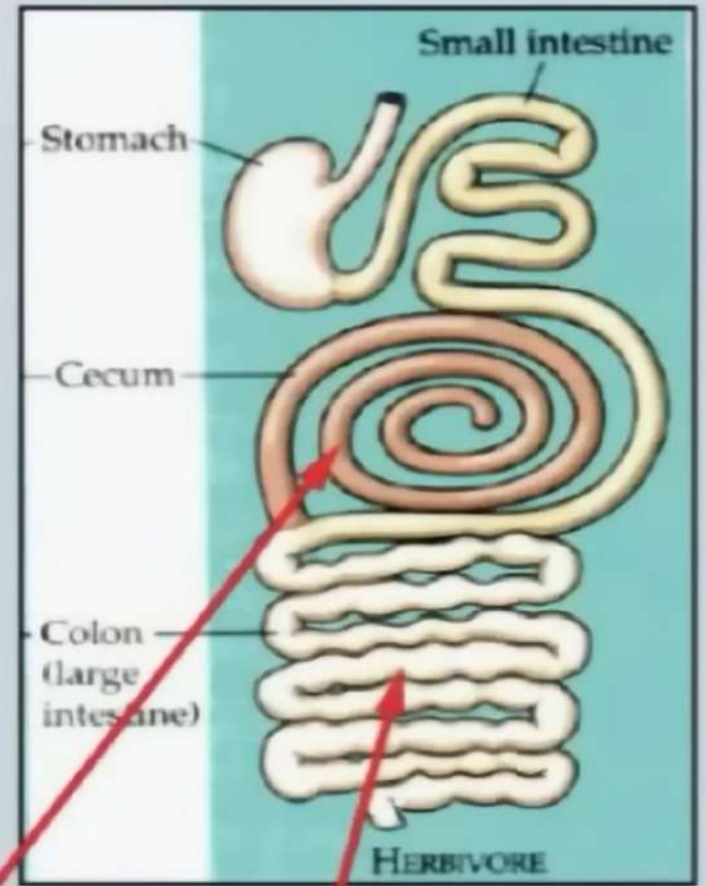
NO CARBS!

http://vetmedicine.about.com/

Gorilla's Diet

74 g of intake is vegetable fibre

Gorilla is a "hindgut digester"



Bacterial fermentation in the gorilla's cæcum and colon converts vegetable fibre into short-chain fatty acids (SCFA) @ ~2 kcals/g (fibre)

Popovich DG, et al. The Western Lowland Gorilla Diet Has Implications for the Health of Humans and Other Hominoids. *J Nutr* 1997; 127: 2000-2005

Gorilla's Diet

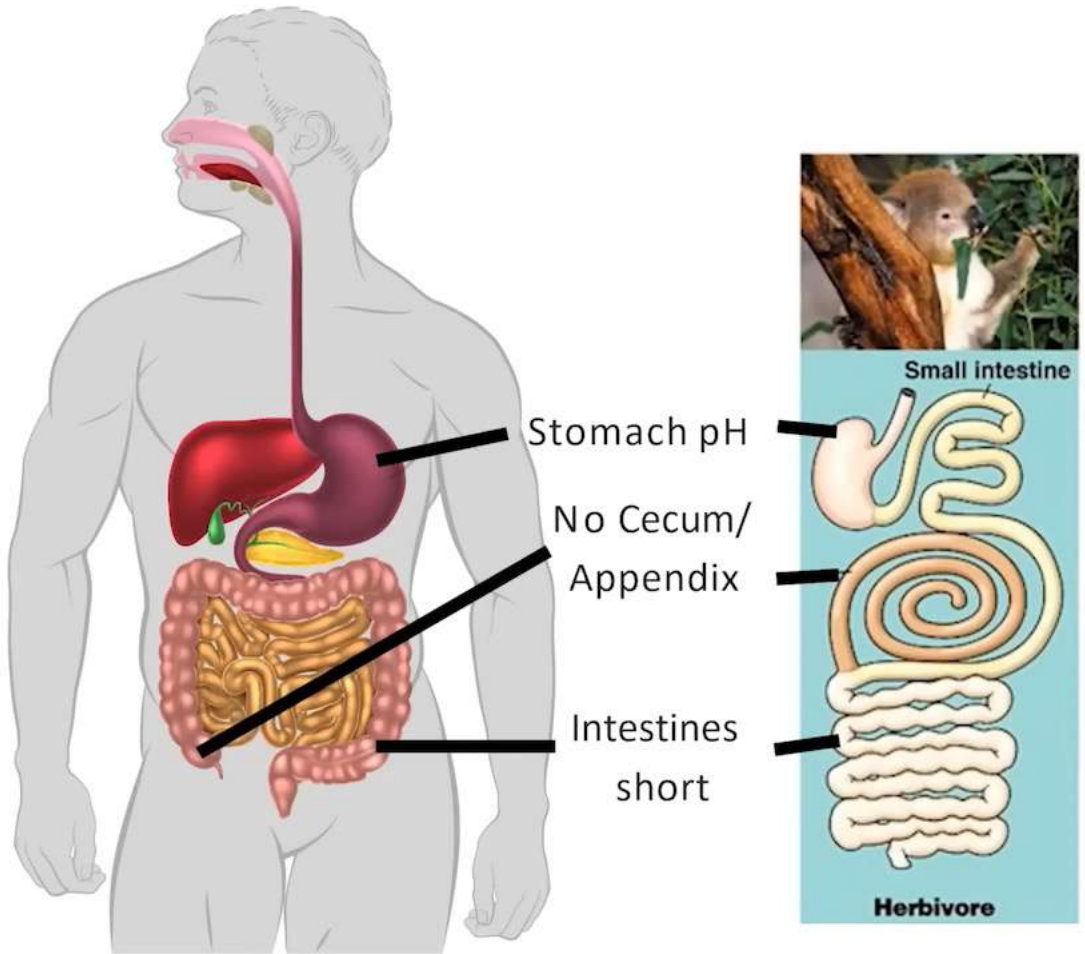
Overall energy (kcal) per 1 kg

Protein	47 kcals	58%	**20.5%** Prot
Available carbs	30 kcals	37%	**13.1%** Carb
Fat	4.5 kcals	5%	
SCFA from fibre	**148.0** kcals	1.9% 64·5%	**66·4%** Fat

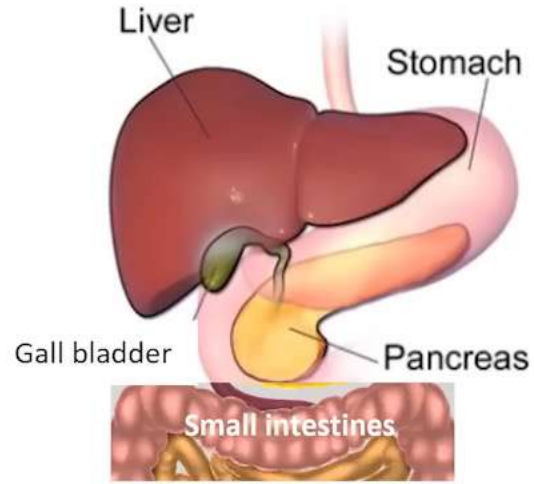
Total **229.5 kcals**

Short chain fatty acids are 100% saturated

We are Carnivores…Biologically



Digestion /GI Track



5 organs to absorb fat

Cannot break down fiber

Colon diseases: Diverticulosis

needs 'rested bowel'

IBD - elemental diet to reverse

In pursuit of nutrients from marrow and brain, early hominins likely smashed animal bones with percussive tools, such as the flint hammerstones in the top row. Flaked stone tools, such as the ax-head fragment in the lower photo, may have been crafted for other tasks. Image: Frank Basford/Wikimedia Commons (Top/Bottom)

Climate Change!

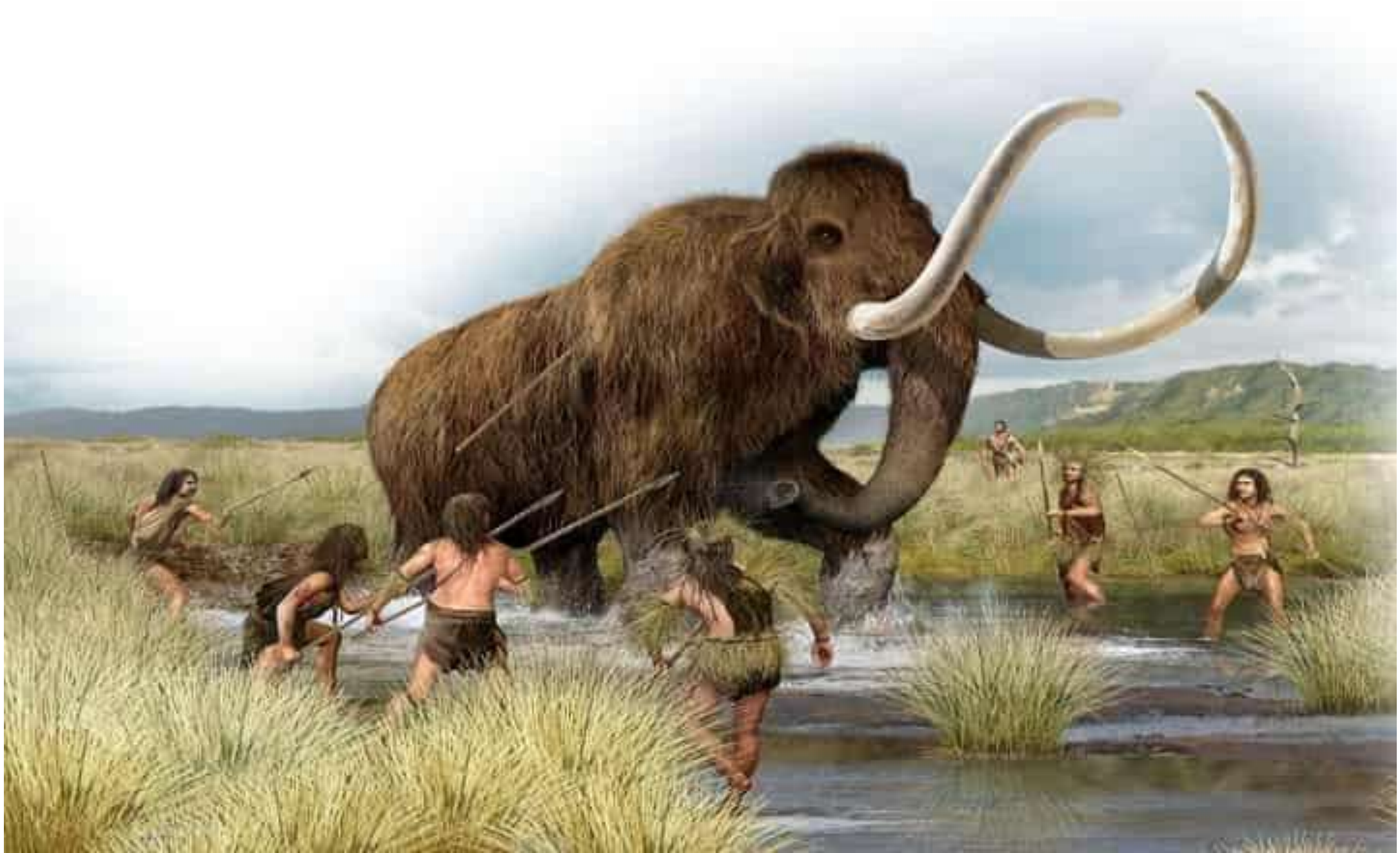
- Ice Ages 2.5 million years
- Long cold winters; short cool summers
- Few plants for short periods
- Heavy reliance on animal foods

Ice Age Food Animals

Palæolithic animals carried a lot of body fat

Hominids smashed skulls and long bones for the fats within

These included 20 and 22 carbon fatty acids



OUR FAMILY TREE

7 MILLION YEARS OF HUMAN EVOLUTION

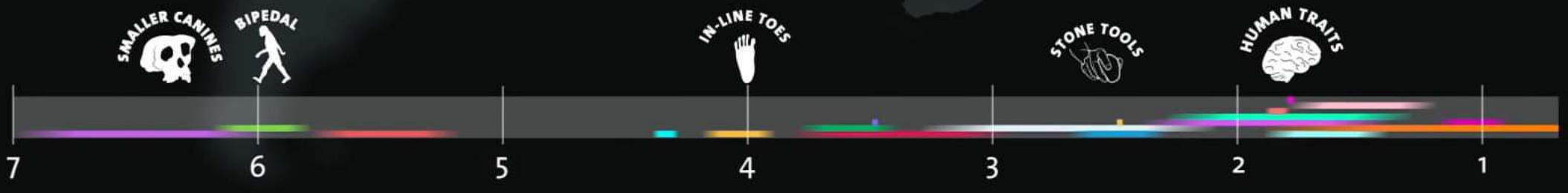
= FOSSIL SITE

= RANGE OF SPECIES (ESTIMATED)

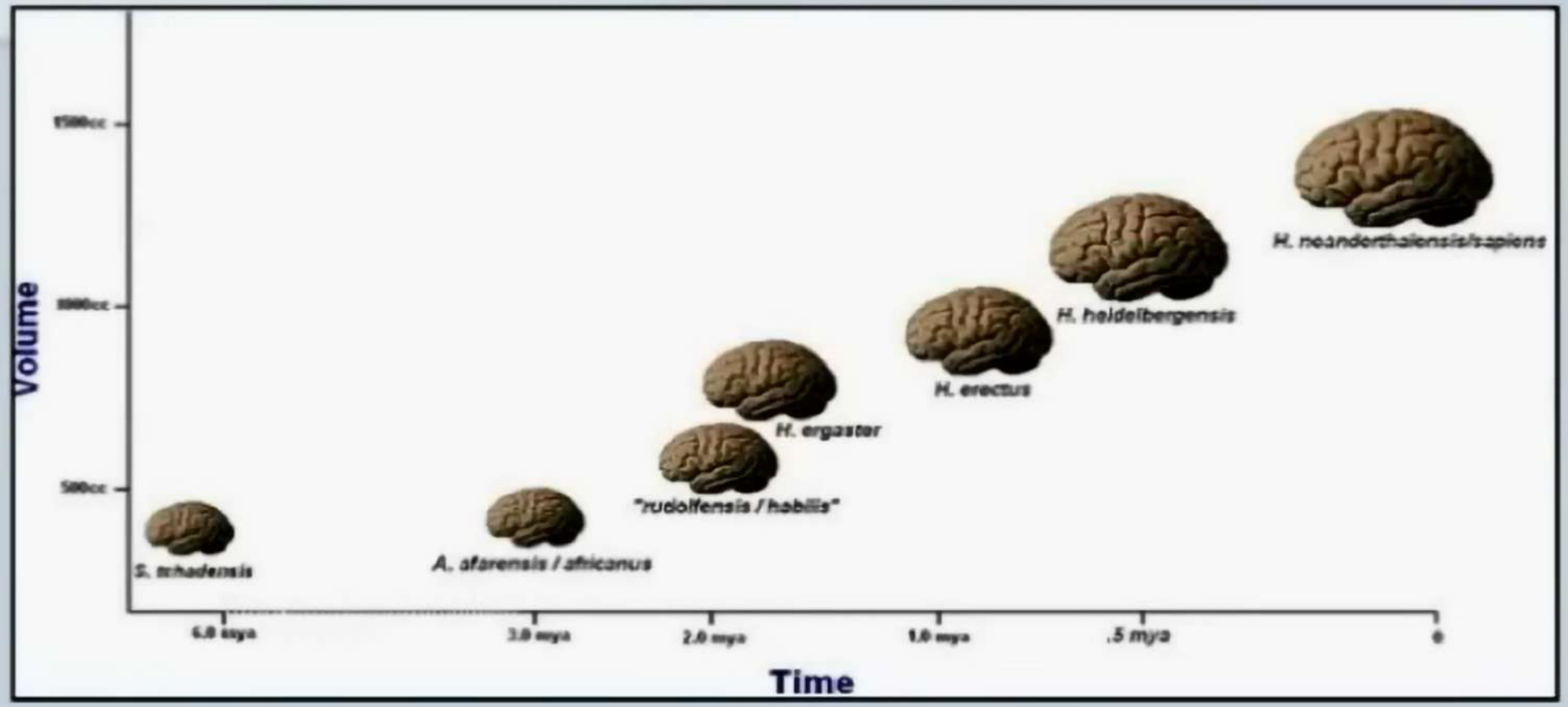
Homo heidelbergensis

Homo erectus

By 800,000 years ago, advances in cooking were fueling further brain growth.



If You Want to Get Ahead, Get a Brain

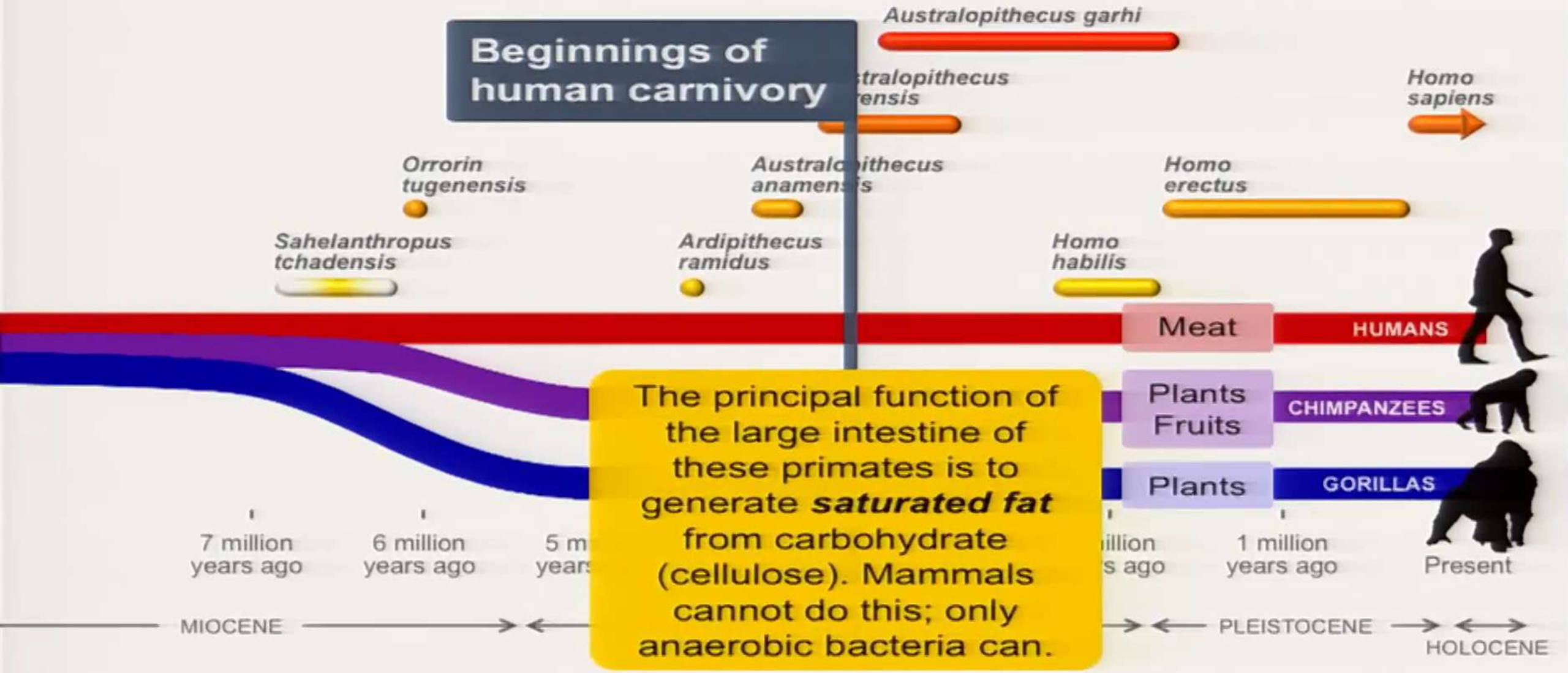


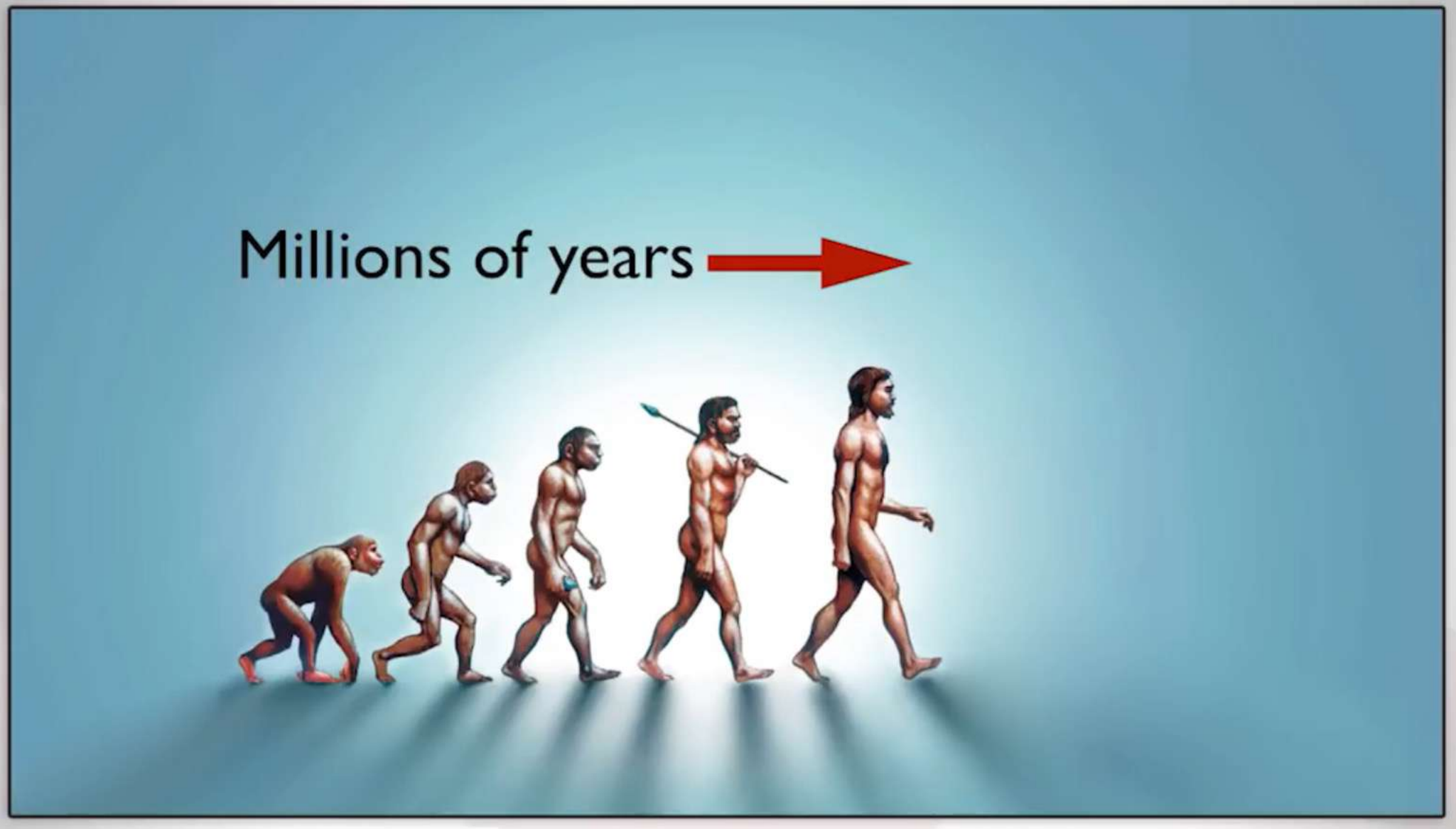
JIM Journal of Internal Medicine
Founded in 1863

Evolution of human diet

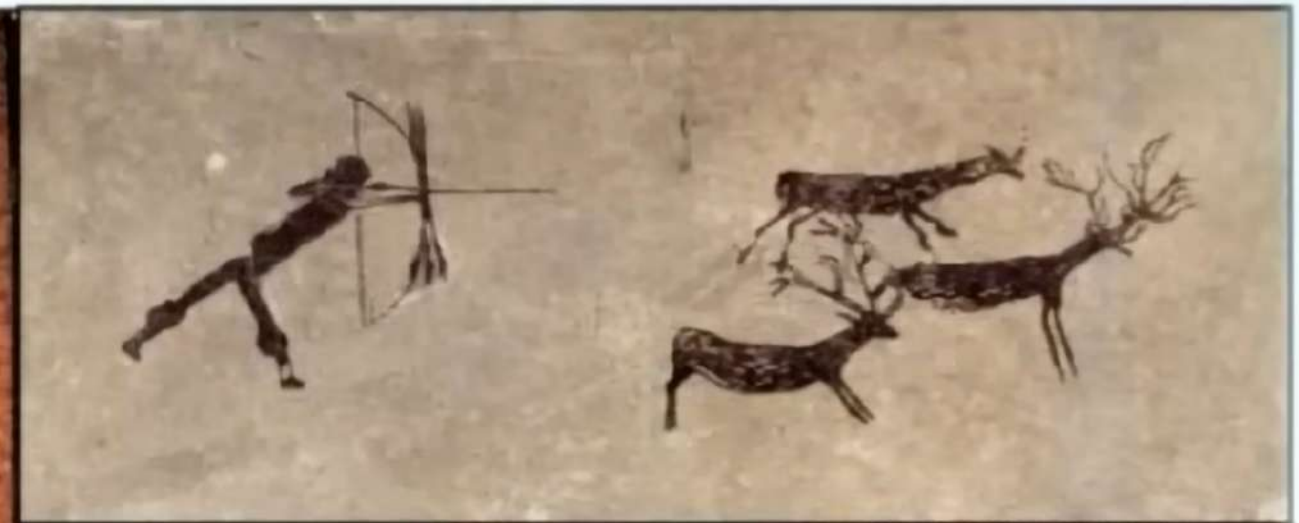
Forest apes	Woodland apes	First hominins	Homo habilis	Homo erectus	Homo sapiens
	15 Ma	8-6 Ma	3 Ma	1.8 Ma	800 kyr
Fruits	Fruits	Fruits	Omnivorous	Omnivorous Fire, cooking	Carnivorous Fire, cooking

DIETARY CHANGE DRIVES HUMAN EVOLUTION




Millions of years

Cave Paintings




Pūd

The Neolithic Revolution

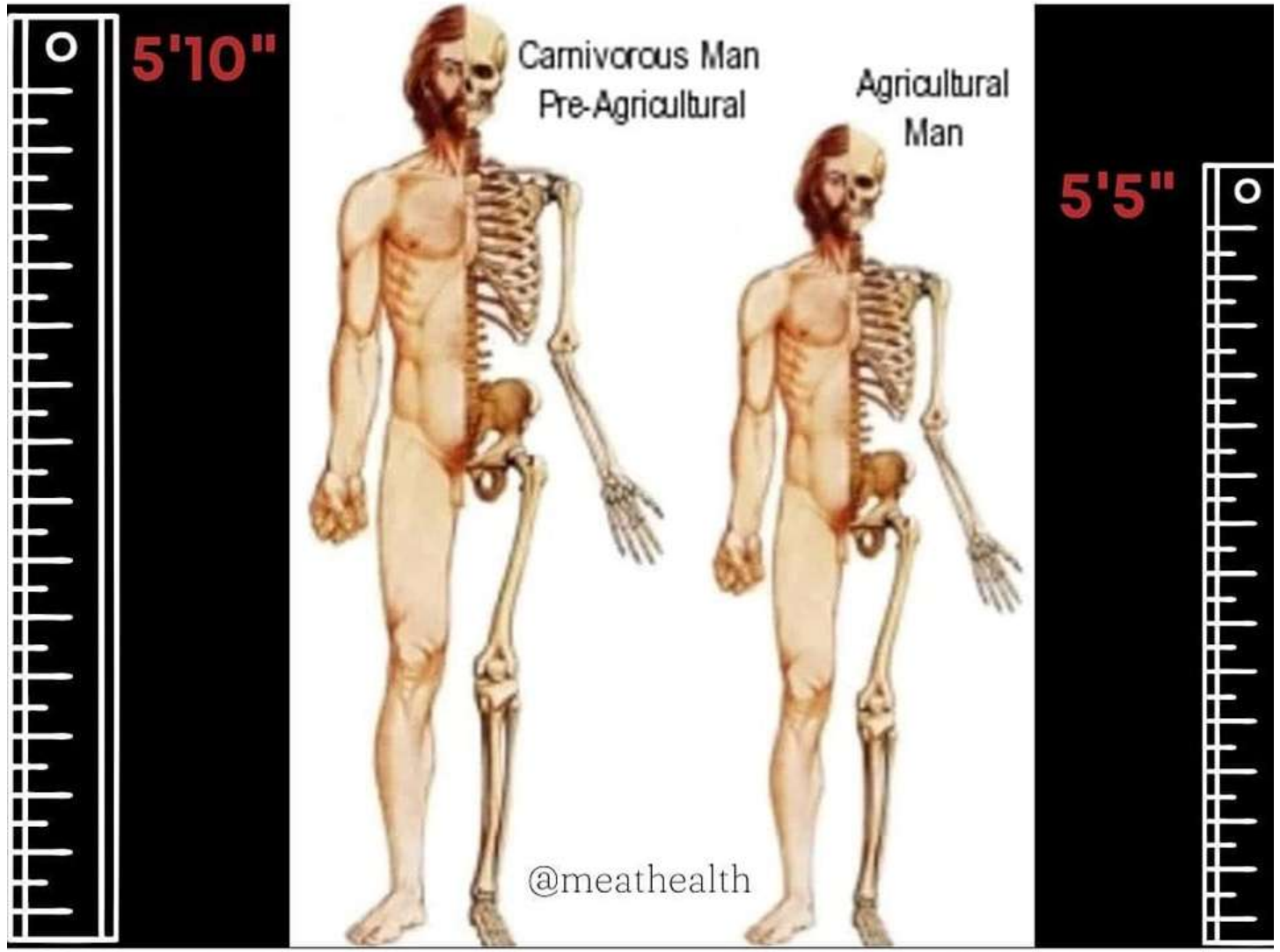
Food group	**Totals** (million tonnes) (estimated edible dry matter)
Cereals	**1,545**
Tubers (potatoes, etc)	136
Pulses (beans, lentils)	127
Meats, milk and eggs	**119**
Sugar	101
Fruits	34

Brain size shrunk by 11%

Eaton SB, et al. *Evolution, diet and health*. Presented in association with the scientific session, Origins and Evolution of Human Diet. 14th International Congress of Anthropological and Ethnological Sciences, Williamsburg, Virginia; 1998.

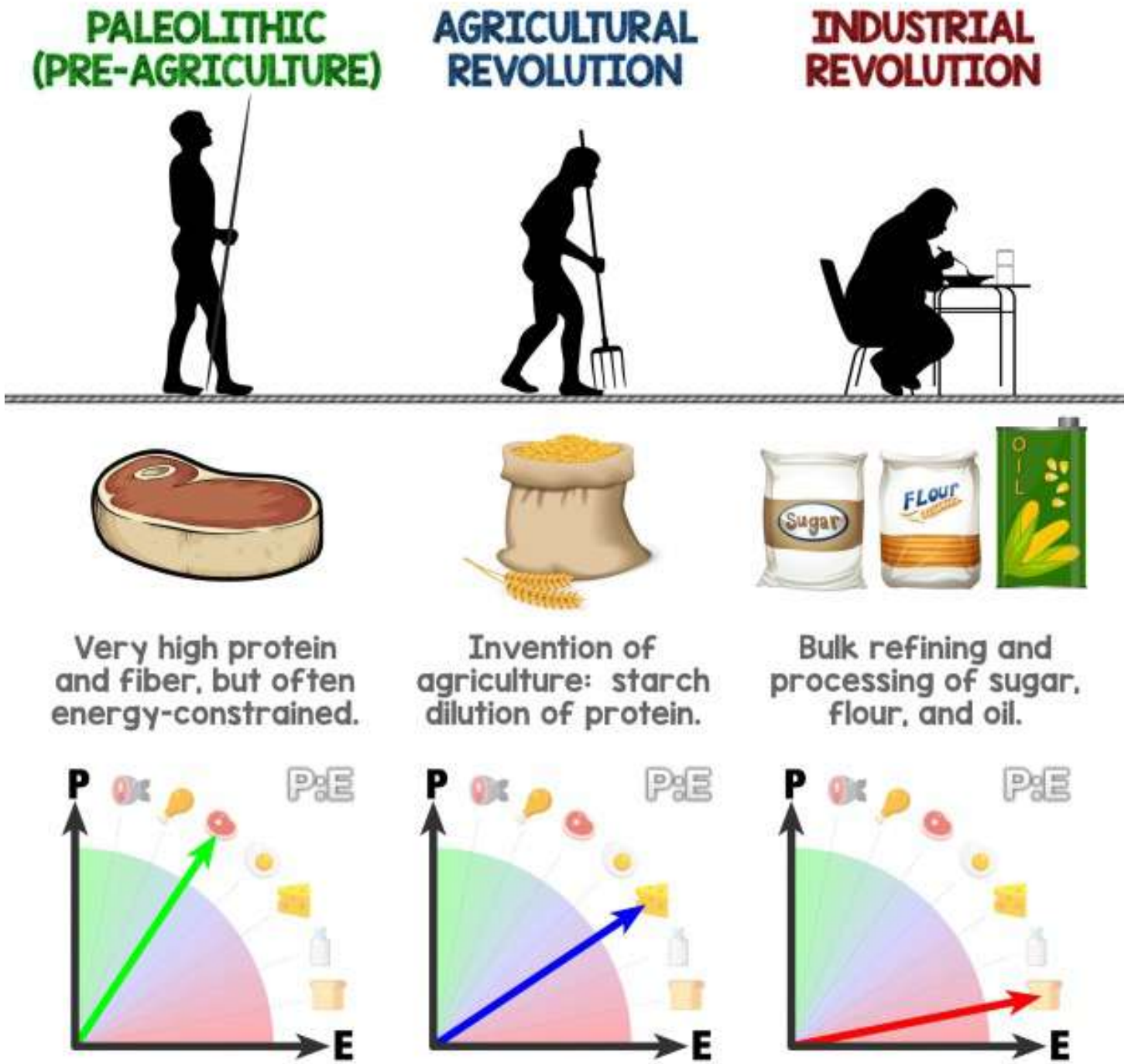
HEIGHT IS A BIOMARKER FOR POPULATION HEALTH.

ONCE THE AGRICULTURAL REVOLUTION BEGAN ABOUT 12,000 YEARS AGO, HEIGHTS STARTED TO DECREASE.



Contrasting hunter-gatherer average height with that of farmers.

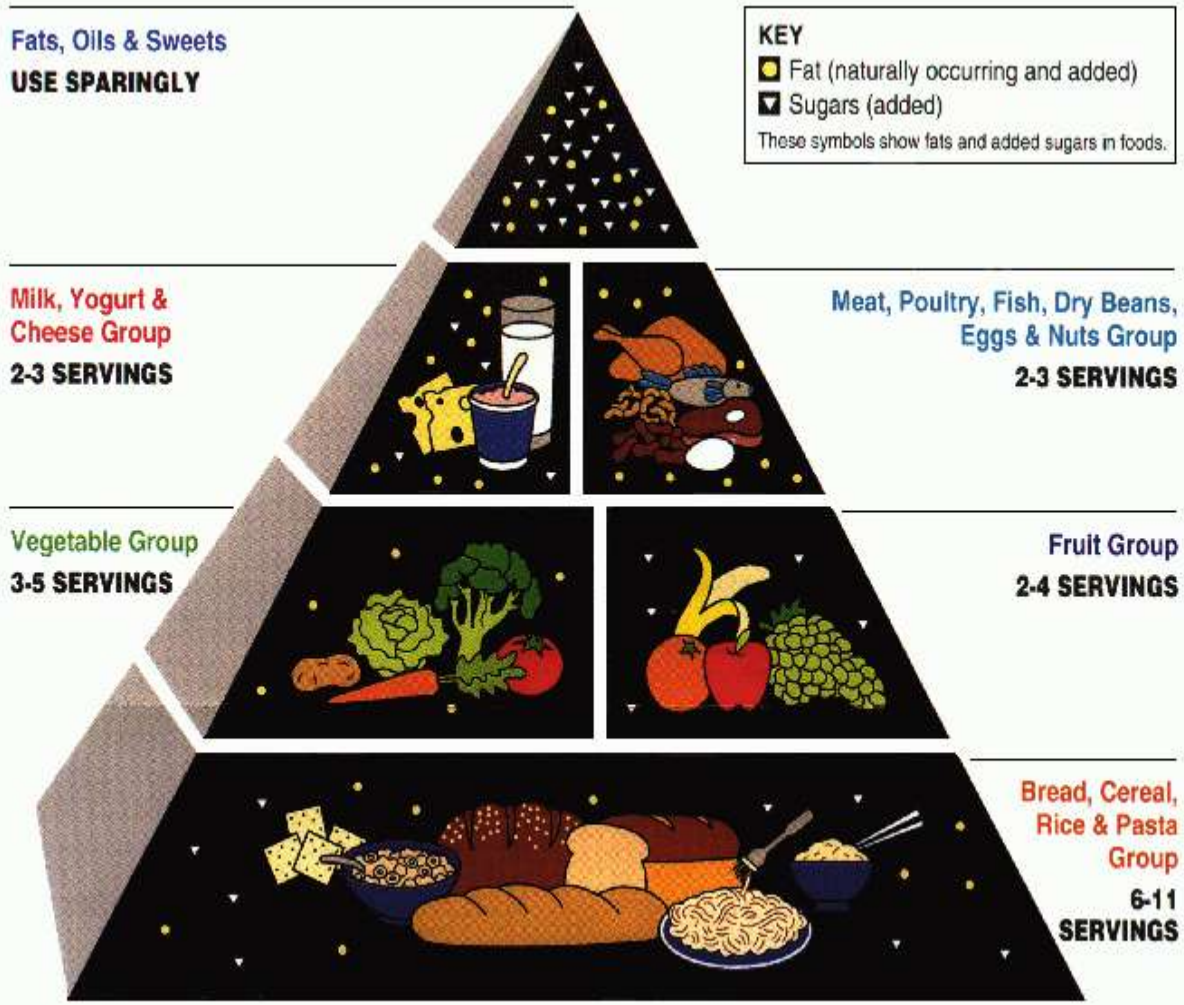
Evolutionary Lens



Dietary Goals For the United States 1977

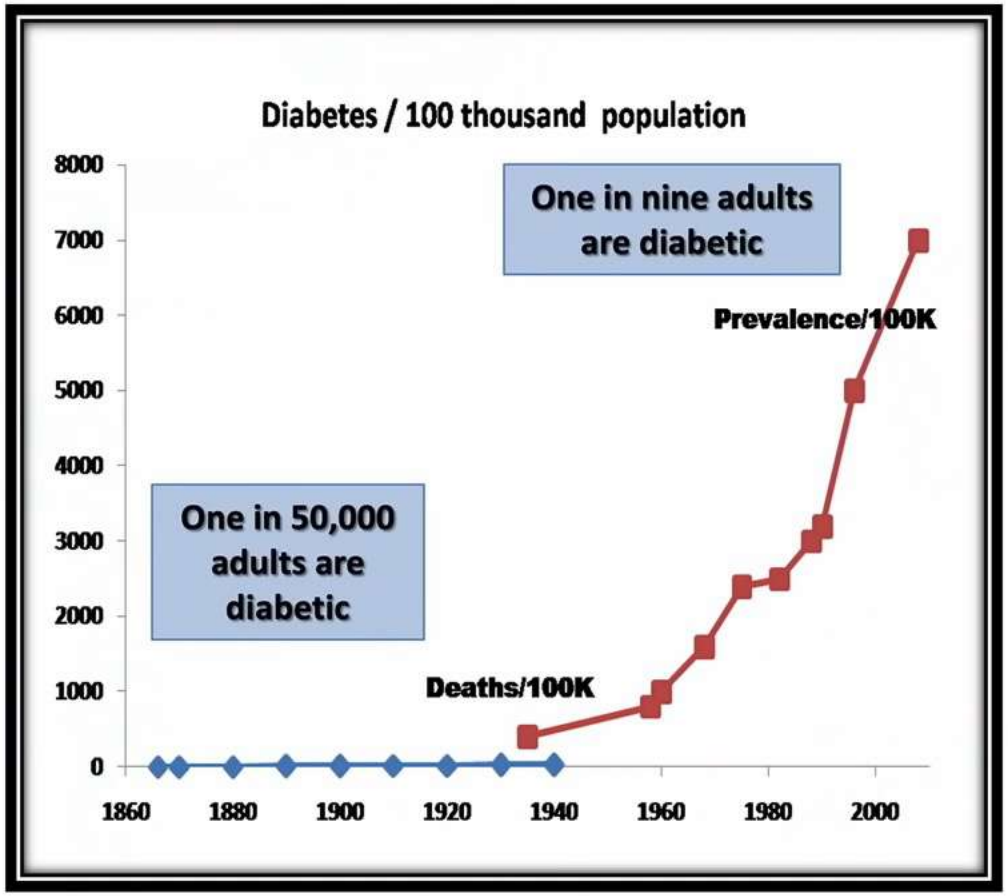
Dietary Goals

1. Raise consumption of carbohydrates until they constituted 55-60% of calories
2. Decrease fat consumption from approximately 40% to 30% of which no more than 1/3 from saturated fat



www.kidneylifescience.ca

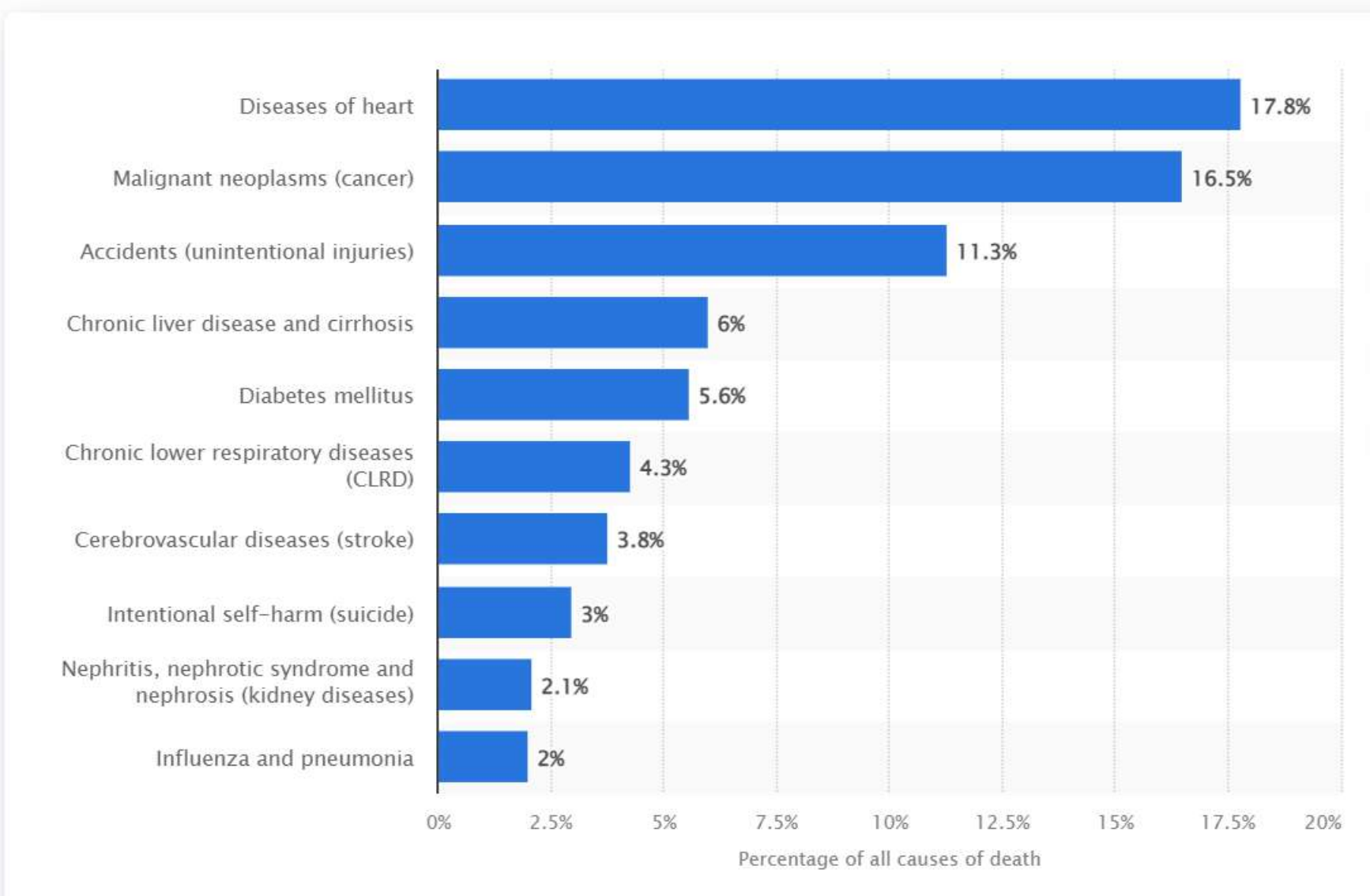
Obesity and Diabetes: The Twin Epidemics



Many proposed causes: Western diet and lack of exercise most favored

Johnson et al Am J Clin Nutr 2007

Year 2019


Diseases of heart
17.8%
Malignant neoplasms (cancer)
16.5%
Accidents (unintentional injuries)
11.3%
Chronic liver disease and cirrhosis
6%
Diabetes mellitus
5.6%
Chronic lower respiratory diseases (CLRD)
4.3%
Cerebrovascular diseases (stroke)
3.8%
Intentional self-harm (suicide)
3%
Nephritis, nephrotic syndrome and nephrosis (kidney diseases)
2.1%
Influenza and pneumonia
2%
0%
2.5%
5%
7.5%
10%
12.5%
15%
17.5%
20%
Percentage of all causes of death

openheart

Evidence from randomised controlled trials did not support the introduction of dietary fat guidelines in 1977 and 1983: a systematic review and meta-analysis

Zoë Harcombe,[1] Julien S Baker,[1] Stephen Mark Cooper,[2] Bruce Davies,[3] Nicholas Sculthorpe,[1] James J DiNicolantonio,[4] Fergal Grace[1]

To cite: Harcombe Z, Baker JS, Cooper SM, *et al*. Evidence from randomised controlled trials did not support the introduction of dietary fat guidelines in 1977 and 1983: a systematic review and meta-analysis. *Open Heart* 2015;**2**:e000196. doi:10.1136/openhrt-2014-000196

ABSTRACT

Objectives: National dietary guidelines were introduced in 1977 and 1983, by the US and UK governments, respectively, with the ambition of reducing coronary heart disease (CHD) by reducing fat intake. To date, no analysis of the evidence base for these recommendations has been undertaken. The present study examines the evidence from randomised controlled trials (RCTs) available to the US and UK regulatory committees at their respective points of implementation.

KEY MESSAGES

What is already known about this subject?

- Dietary recommendations were introduced in the US (1977) and in the UK (1983) to (1) reduce overall fat consumption to 30% of total energy intake and (2) reduce saturated fat consumption to 10% of total energy intake.

What does this study add?

- No randomised controlled trial (RCT) had tested

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[4]Saint Luke's Mid America Heart Institute, Kansas City, Missouri, USA

ABSTRACT

Objectives: National dietary guidelines were introduced in 1977 and 1983, by the US and UK governments, respectively, with the ambition of reducing coronary heart disease (CHD) by reducing fat intake. To date, no analysis of the evidence base for these recommendations has been undertaken. The present study examines the evidence from randomised controlled trials (RCTs) available to the US and UK regulatory committees at their respective points of implementation.

Methods: A systematic review and meta-analysis were undertaken of RCTs, published prior to 1983, which examined the relationship between dietary fat, serum cholesterol and the development of CHD.

Results: 2467 males participated in six dietary trials: five secondary prevention studies and one including healthy participants. There were 370 deaths from all-cause mortality in the intervention and control groups. The risk ratio (RR) from meta-analysis was 0.996 (95% CI 0.865 to 1.147). There were 207 and 216 deaths from CHD in the intervention and control groups, respectively. The RR was 0.989 (95% CI 0.784 to 1.247). There were no differences in all-cause mortality and non-significant differences in CHD mortality, resulting from the dietary interventions. The reductions in mean serum cholesterol levels were significantly higher in the intervention groups; this did not result in significant differences in CHD or all-cause mortality. Government dietary fat recommendations were untested in any trial prior to being introduced.

Conclusions: Dietary recommendations were introduced for 220 million US and 56 million UK citizens by 1983, in the absence of supporting evidence from RCTs.

KEY MESSAGES

What is already known about this subject?

- Dietary recommendations were introduced in the US (1977) and in the UK (1983) to (1) reduce overall fat consumption to 30% of total energy intake and (2) reduce saturated fat consumption to 10% of total energy intake.

What does this study add?

- No randomised controlled trial (RCT) had tested government dietary fat recommendations before their introduction. Recommendations were made for 276 million people following secondary studies of 2467 males, which reported identical all-cause mortality. RCT evidence did not support the introduction of dietary fat guidelines.

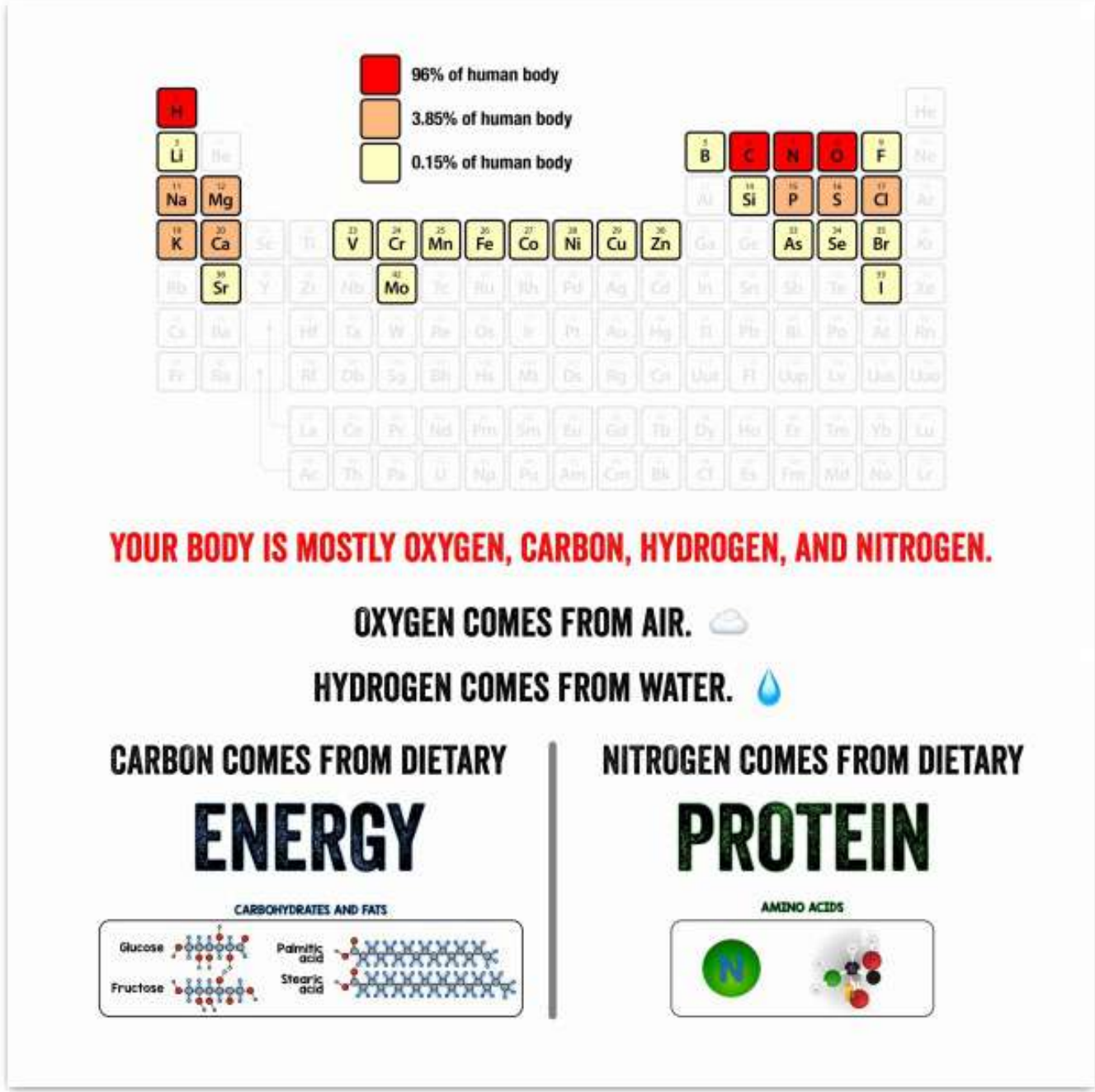
How might this impact on clinical practice?

- Clinicians may be more questioning of dietary guidelines, less accepting of low-fat advice (concomitantly high carbohydrate) and more engaged in nutritional discussions about the role of food in health.

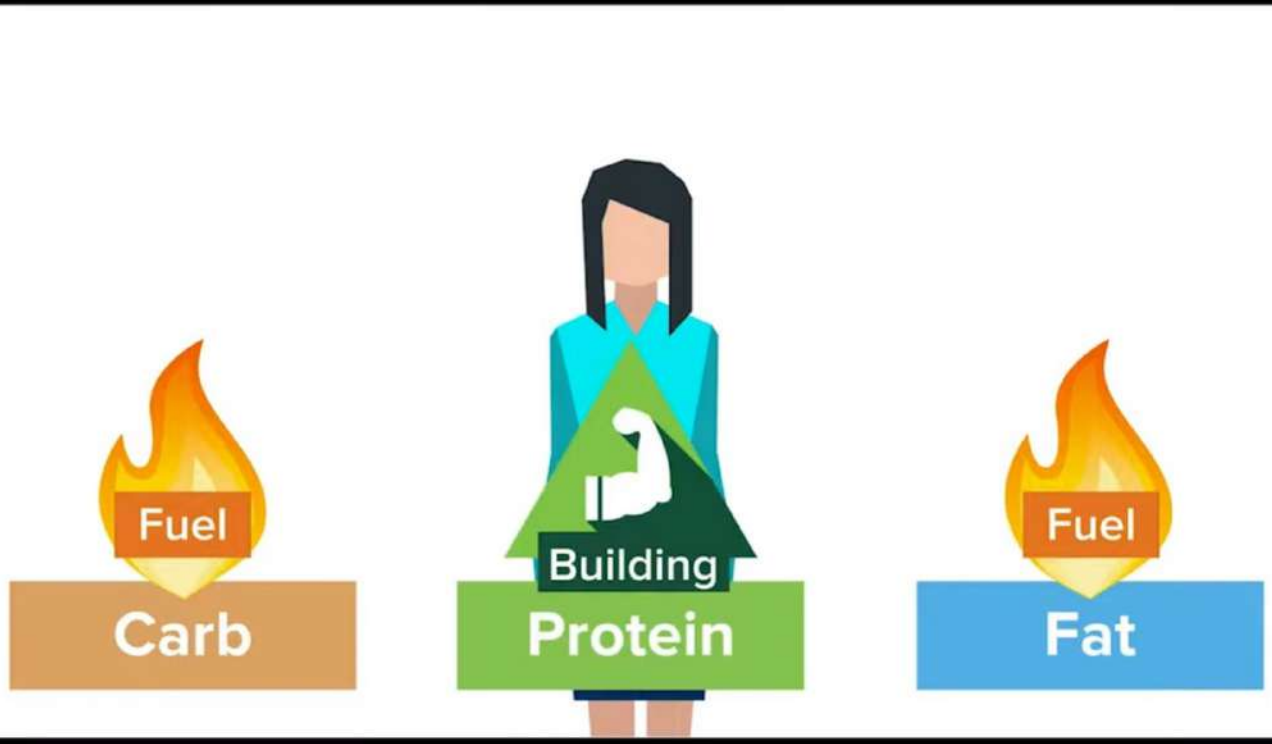
INTRODUCTION

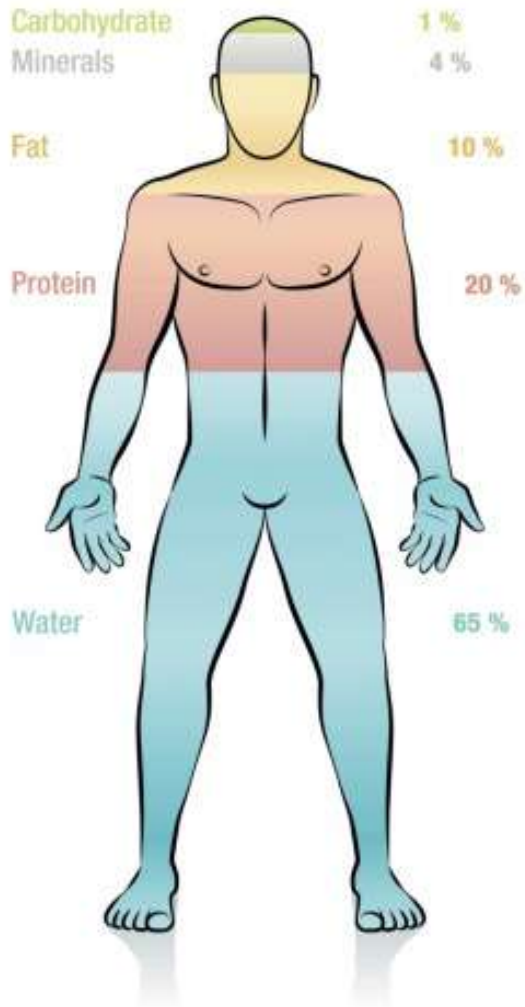
US public health dietary advice was advice issued by the National Advisory Committee on Nutritional Education in 1983.[2] The dietary recommendations in both cases focused on reducing dietary fat intake; specifically to (1) reduce overall fat consumption to 30% of total energy intake and (2) reduce saturated fat consumption to 10% of total energy intake.

The recommendations were an attempt to address the incidence of coronary heart disease (CHD). Both documents acknowledged that the evidence was not conclusive. Hegsted's introduction to the Dietary Goals for the US noted "there will undoubtedly be



Carbon, hydrogen, oxygen, and all of the ENERGY they contain comes from AIR, WATER, and SUNLIGHT. All of your nitrogen and other elements, necessary for PROTEIN, come from soil and the EARTH.

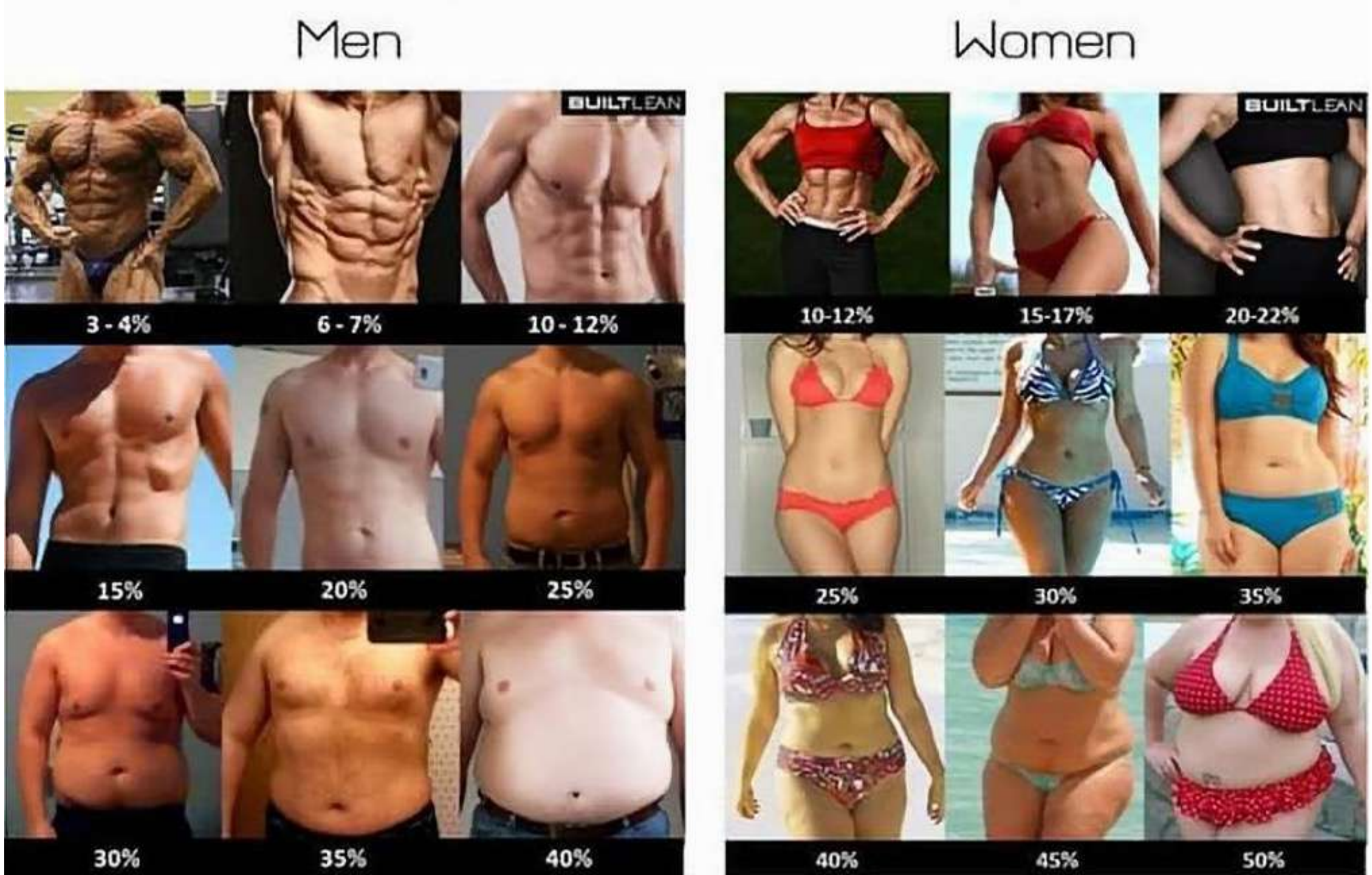

Fuel
Carb
Building
Protein
Fuel
Fat



ENERGY 10%
PROTEIN 20%

MUCH LIKE YOUR DIET, YOUR BODY HAS A PROTEIN TO ENERGY RATIO AS WELL— HIGHER IS BETTER.

[WILL BE LOW IF YOU ARE OVERWEIGHT OR UNDER-MUSCLED]


Men
Women
BUILTLEAN
3 - 4%
6 - 7%
10 - 12%
15%
20%
25%
30%
35%
40%
10-12%
15-17%
20-22%
25%
30%
35%
40%
45%
50%


30 ธ.ค. 2016

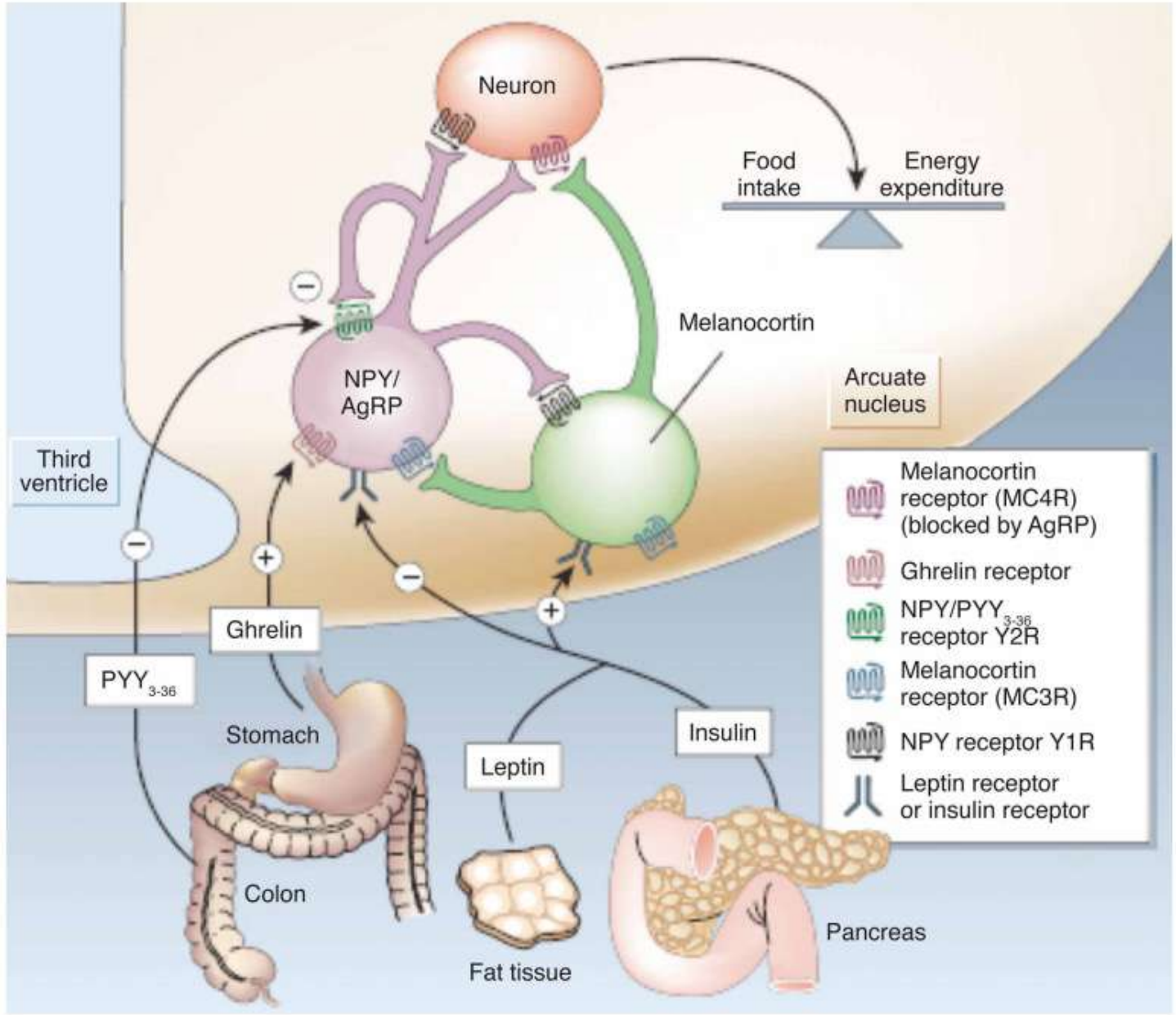
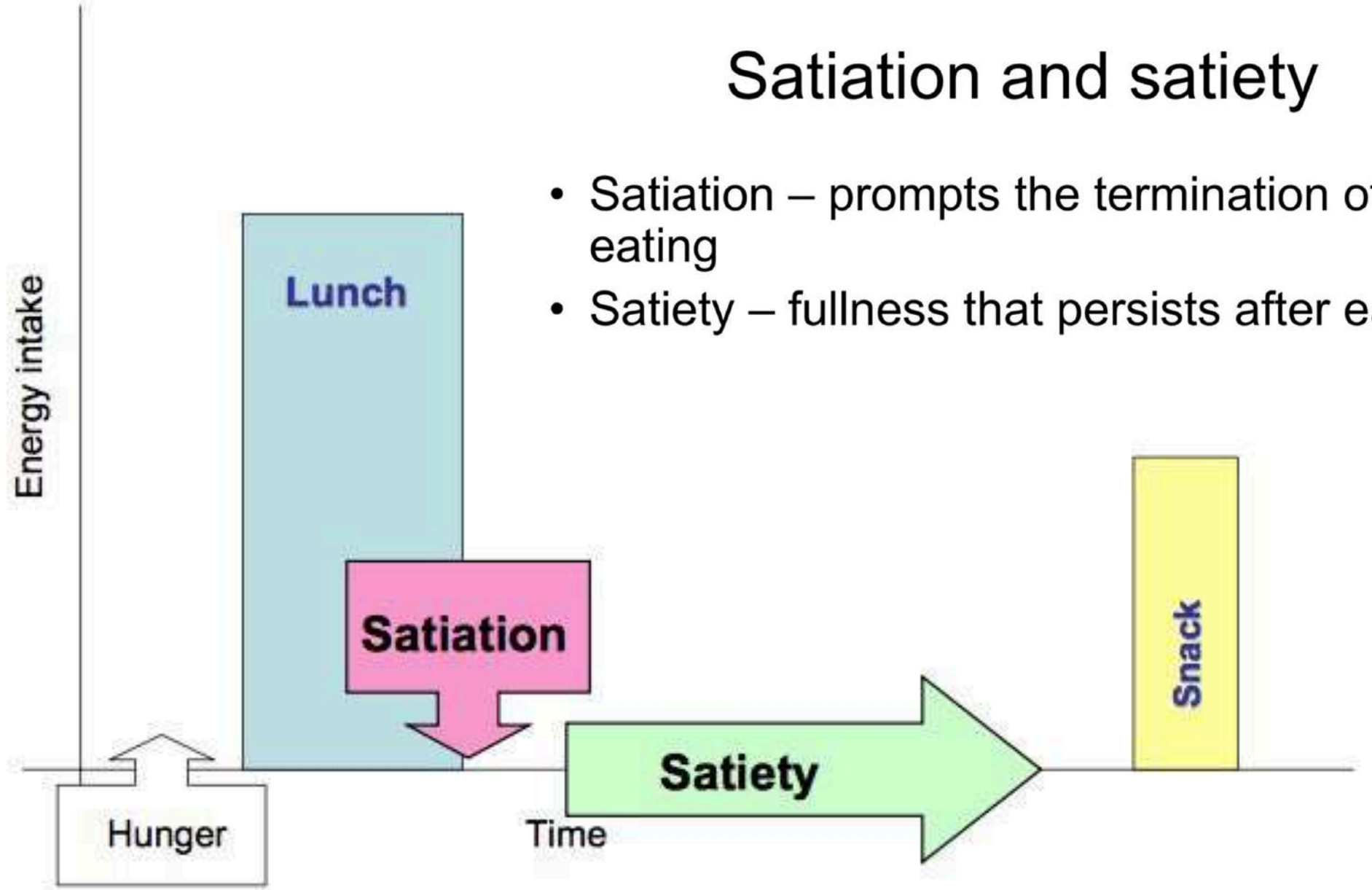


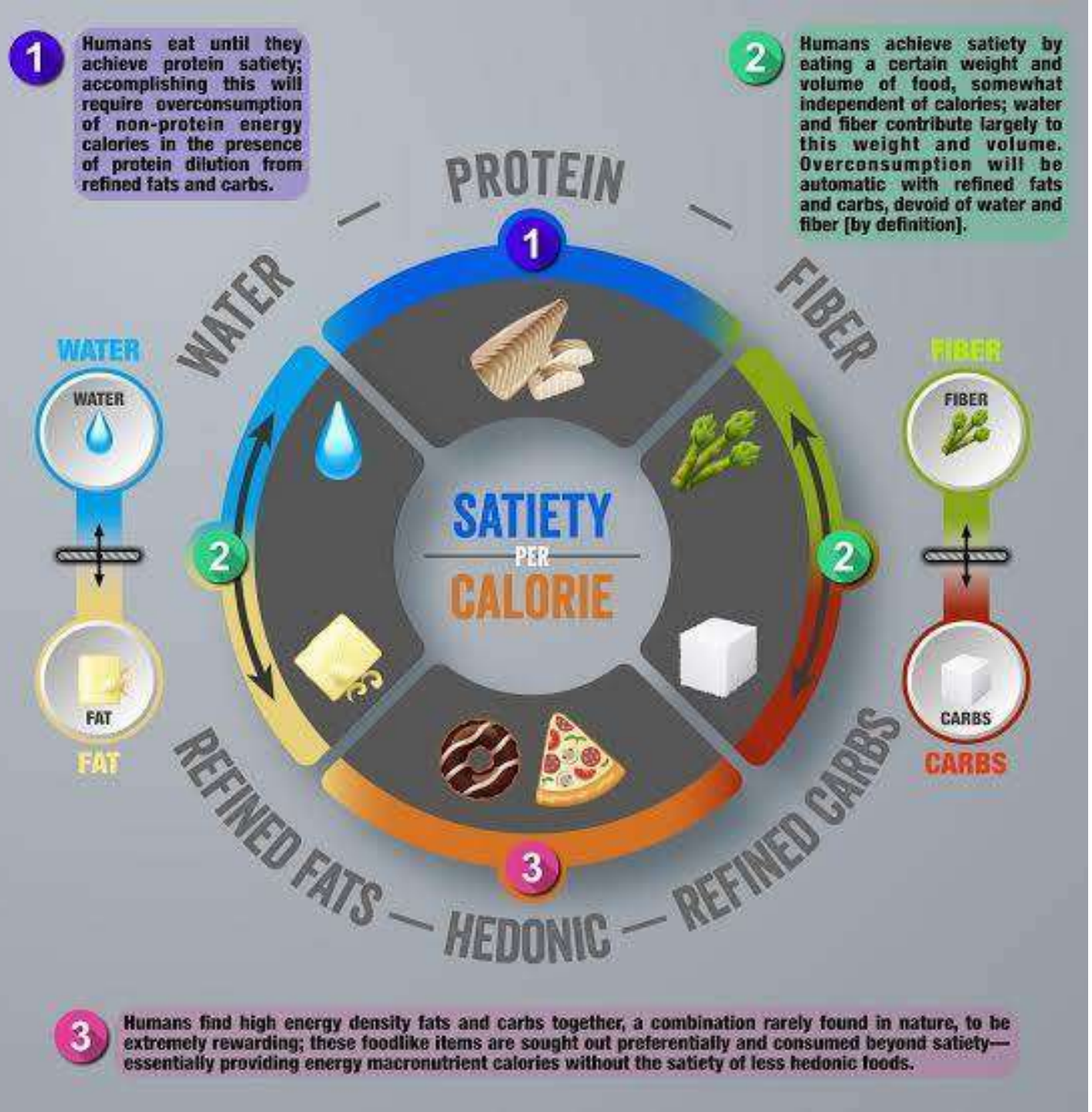
FIGURE 2 | Hypothalamic control of global energy balance. Appetite is regulated by a complex feed-back loop involving endocrine signals originated in peripheral tissues and intrahypothalamic peptides. Leptin and insulin inhibit the orexigenic NPY/AgRP neurons (purple) and stimulate the anorexigenic melanocortin neurons (green), resulting in a reduction of food intake. Ghrelin or $PYY_{3–36}$ activate or inhibit the NPY/AgRP neurons resulting in orexigenic or anorexigenic responses, respectively. Taken from Schwartz and Morton (2002). Reproduced with permission of the publisher.

What are satiation and satiety?



Satiation and satiety

- Satiation – prompts the termination of eating
- Satiety – fullness that persists after eating


1
Humans eat until they achieve protein satiety; accomplishing this will require overconsumption of non-protein energy calories in the presence of protein dilution from refined fats and carbs.
2
Humans achieve satiety by eating a certain weight and volume of food, somewhat independent of calories; water and fiber contribute largely to this weight and volume. Overconsumption will be automatic with refined fats and carbs, devoid of water and fiber [by definition].
PROTEIN
WATER
FIBER
REFINED FATS
HEDONIC
REFINED CARBS
WATER
WATER
FAT
FAT
FIBER
FIBER
CARBS
CARBS
SATIETY
PER
CALORIE
1
2
2
3
3
Humans find high energy density fats and carbs together, a combination rarely found in nature, to be extremely rewarding; these foodlike items are sought out preferentially and consumed beyond satiety—essentially providing energy macronutrient calories without the satiety of less hedonic foods.

HIGHEST and LOWEST

highest ad libitum energy intake

protein

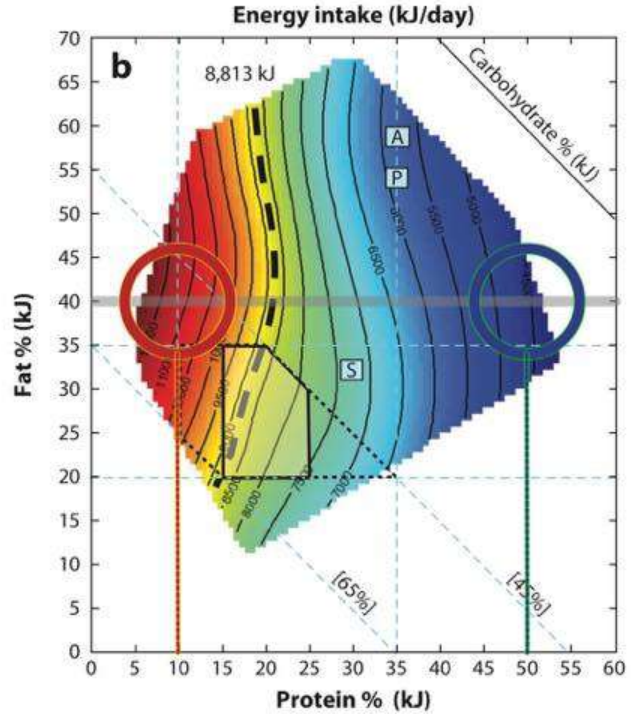
10%

fat

40%

carbohydrate

50%



lowest ad libitum energy intake

protein

50%

fat

40%

carbohydrate

10%

Combining fats and carbohydrates

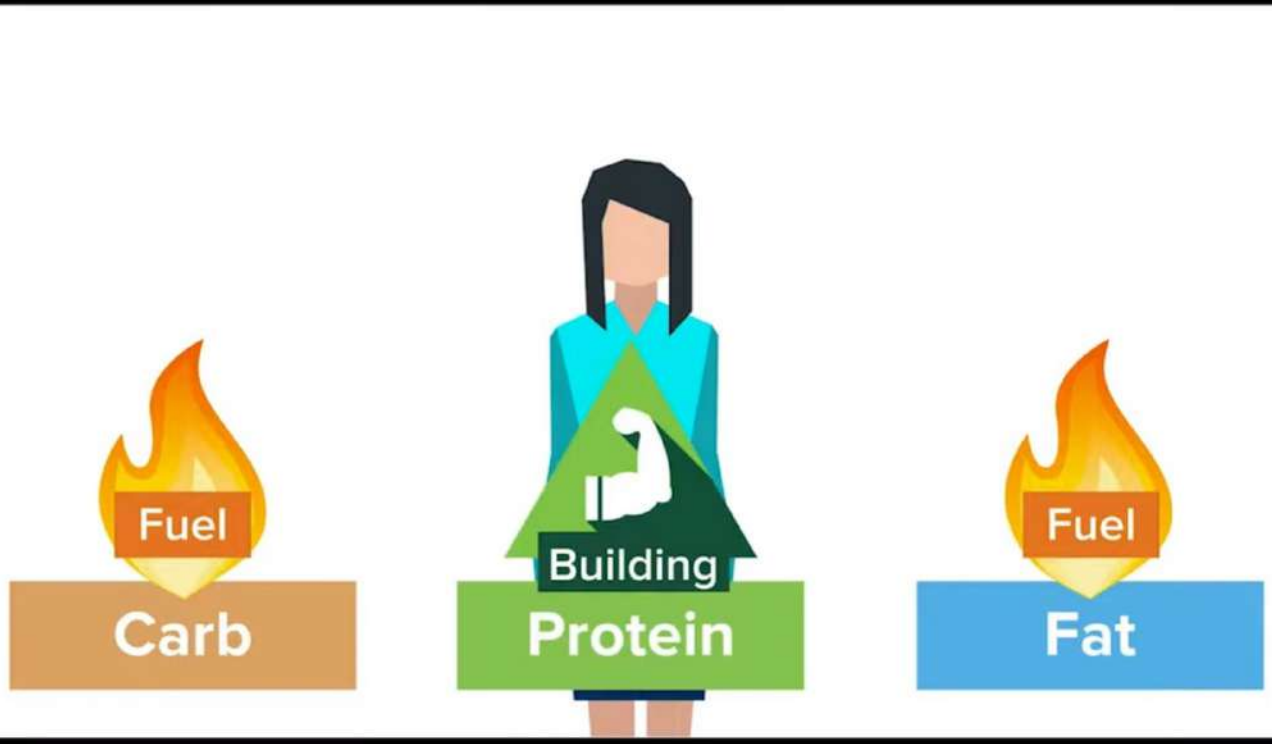
Food companies know these ‘comfort foods’ release dopamine In addictive centers of the brain.

DR. STEVEN
YOUR PLACE FOR ANSWERS

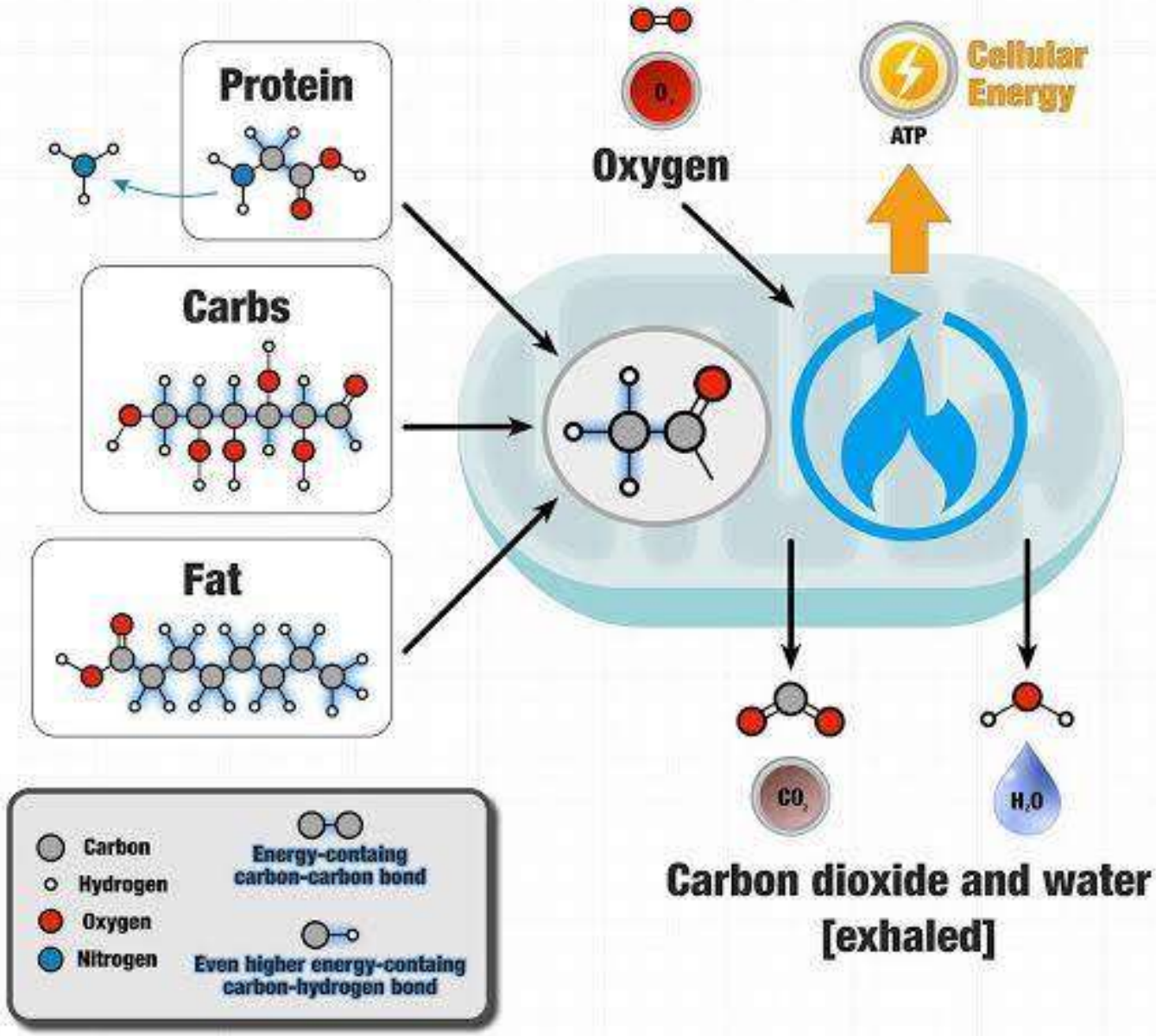
BEWARE THE TRIFECTA

HIGH CARB • HIGH FAT • HIGH ENERGY DENSITY

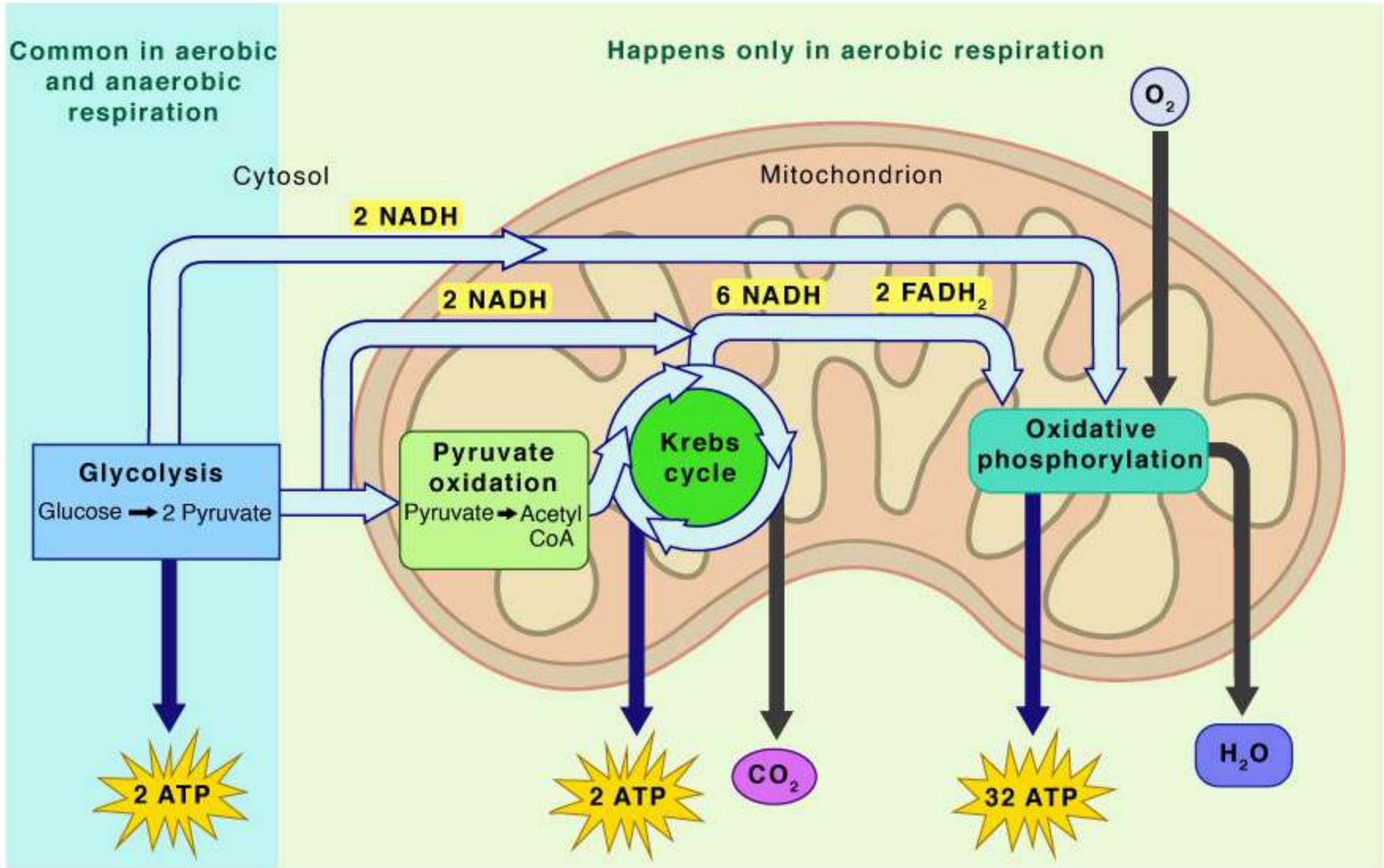
Treat these foods with a LOT of respect, because they are extremely powerful!


Fuel
Carb
Building
Protein
Fuel
Fat

Mitochondria oxidize all macronutrient carbons for energy the same way.

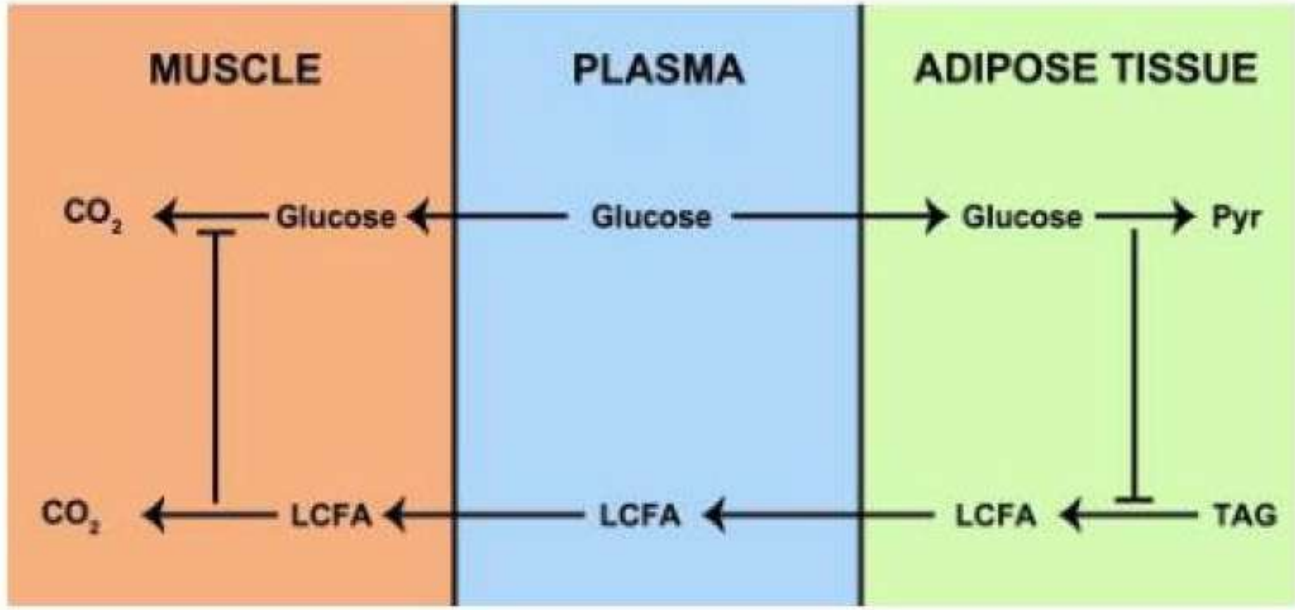


Cellular Respiration



The glucose-fatty acid (Randle) cycle

- The "cycle" also describes the control of fuel selection through the dynamic interactions between circulating concentrations of glucose and fatty acids in coordination with hormones.
- *Inhibition of glucose utilization by fatty acids is a form of glucose intolerance that resembles, or may lead to, insulin resistance.*

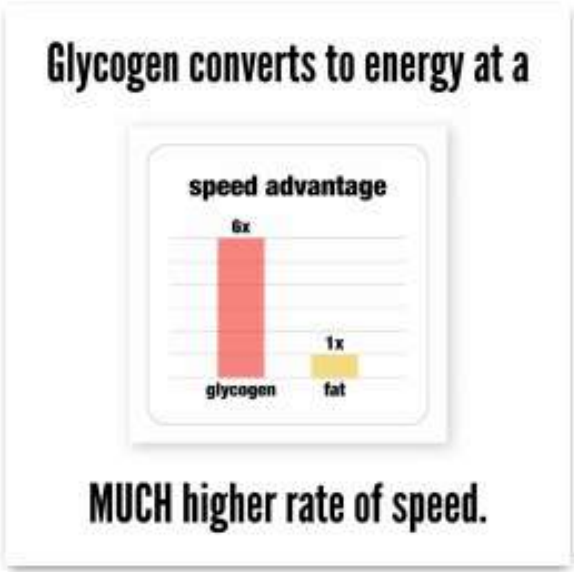


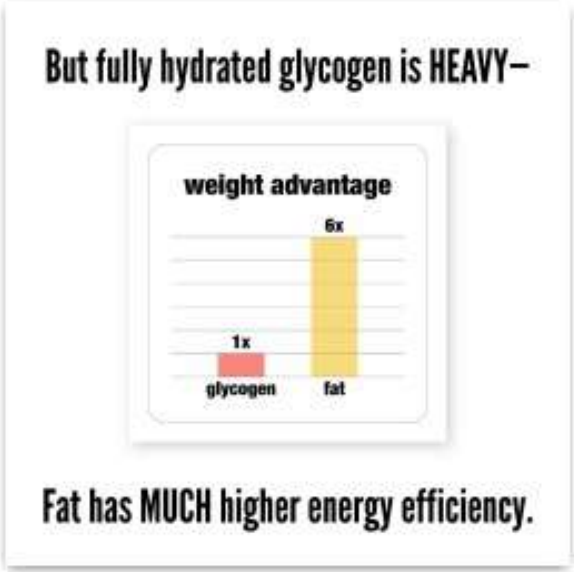
TWO COMPARTMENT SYSTEM

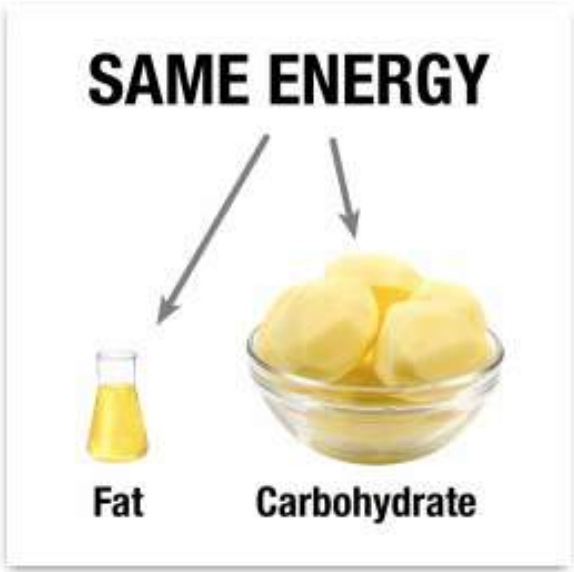
So you have two completely separate energy storage compartments in your body. Glucose (carbohydrate), which is water-soluble, is stored as glycogen (just chains of glucose) in your liver and your muscles. Fat, which is NOT water-soluble, is stored in your adipocytes. Fat storage is MUCH larger, and your body prefers to carry only about 1% of your energy as glycogen. What gives?

It turns out that glucose, from glycogen, is FAST. You can convert glucose into energy SIX TIMES faster compared to fat. So why don't we only use glycogen to store energy? Because glycogen is HEAVY! Glycogen is 'fully hydrated' (this is why we call them carboHYDRATES), which means it has a lot of water attached to it. And that water weighs a lot! So glycogen is about six times heavier than the same amount of energy stored as fat.

Glycogen, which is just chains of glucose molecules, is a sort of 'human starch' and is identical to the starch in potatoes. The fat in the lipid droplets of your adipocytes is very similar to olive oil. Note the photo below of the same amount of stored energy as olive oil versus potatoes. At six times the size and weight, you can see why your body doesn't want to carry around all your energy as glycogen. In fact, because of the weight efficiency, your body carries 100 times more fat than glycogen.

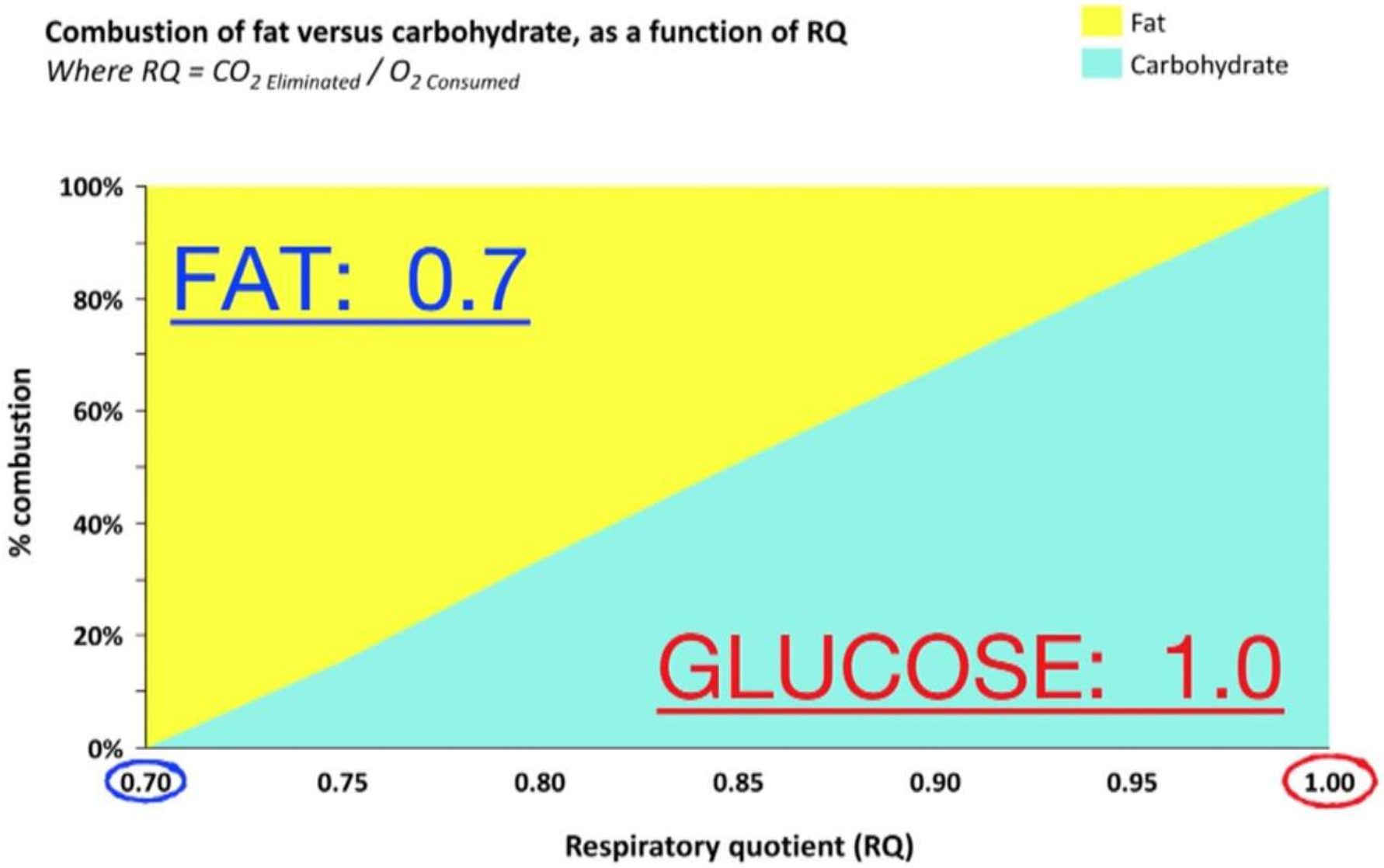


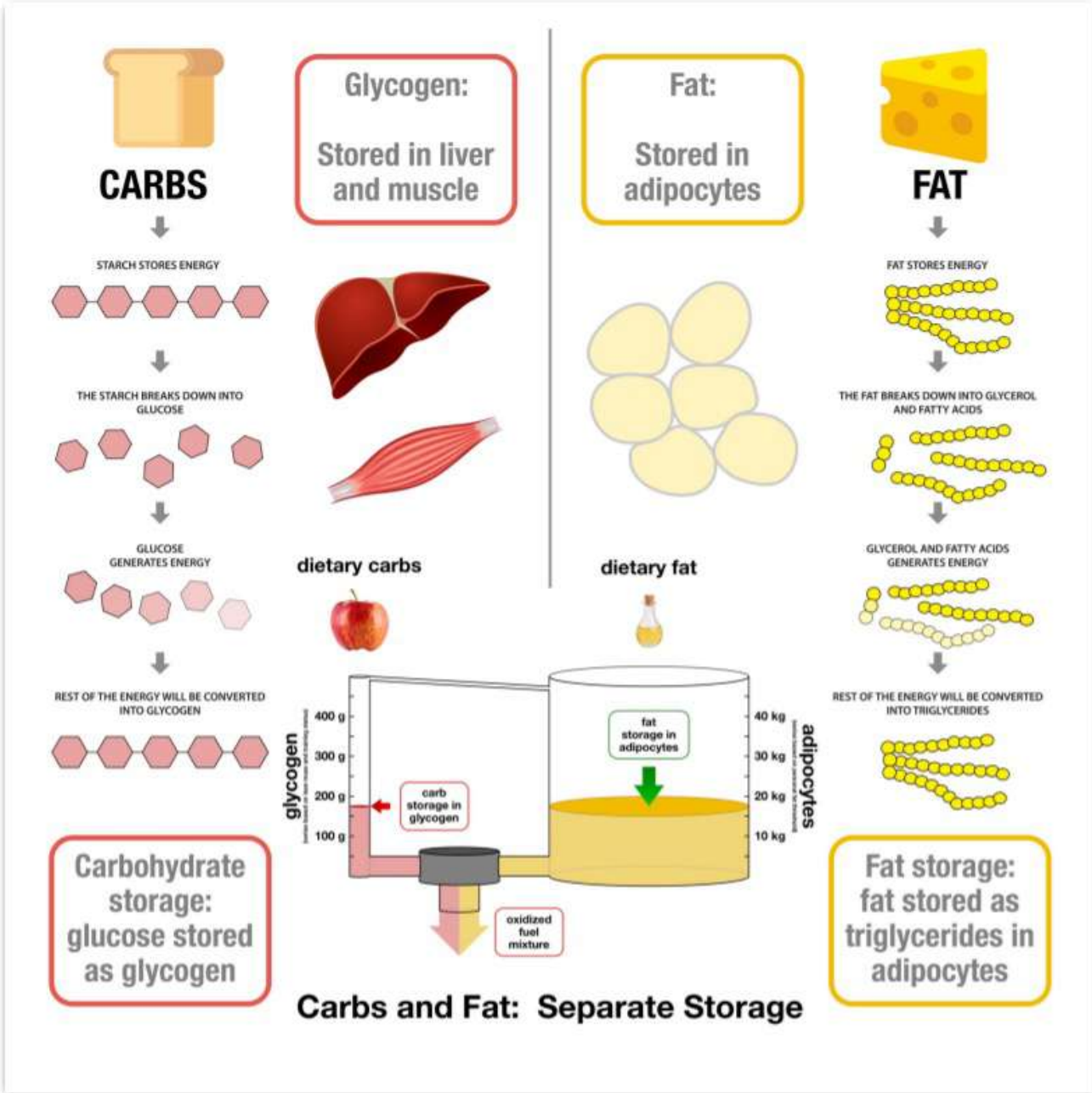




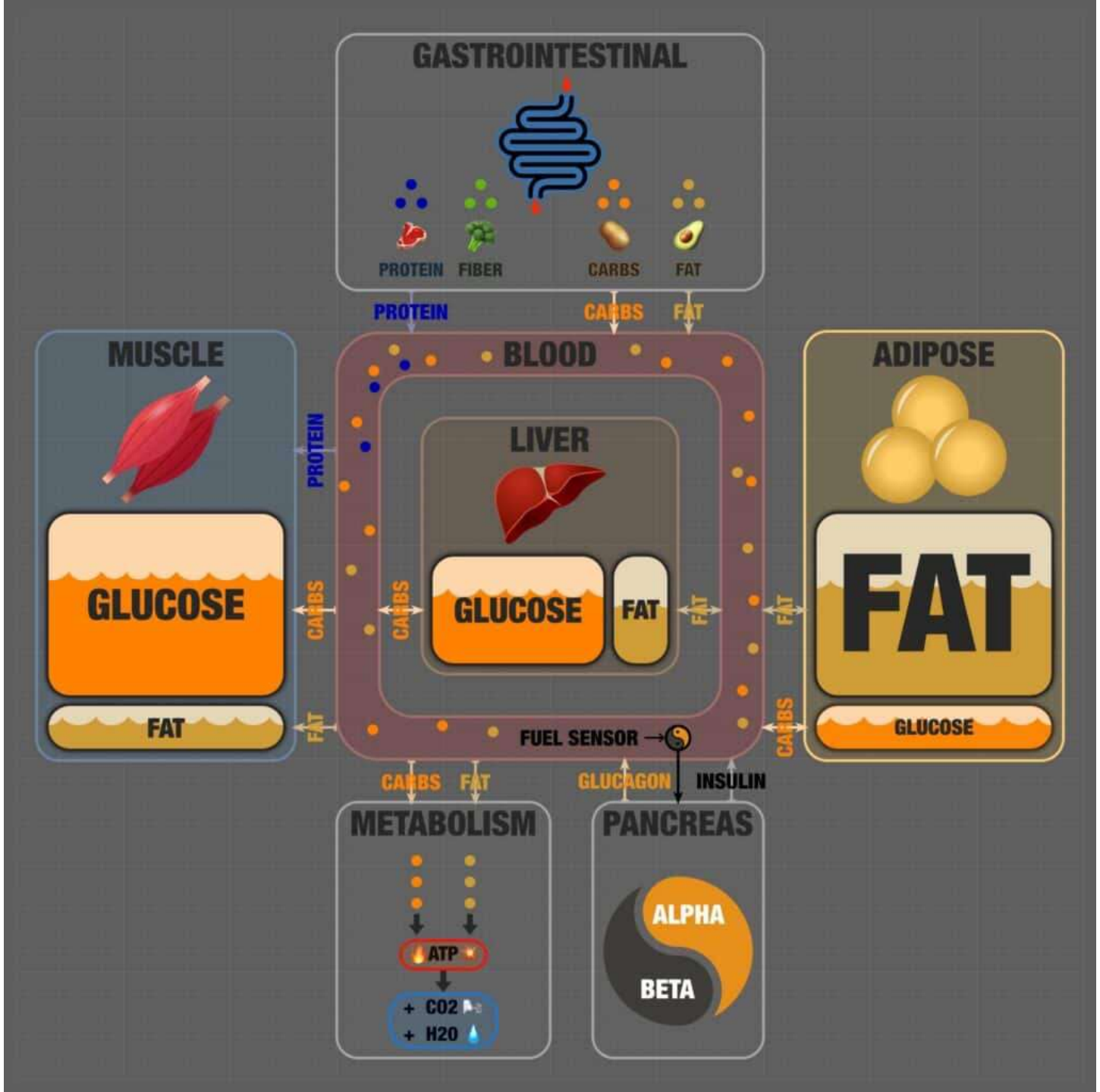
Combustion of fat versus carbohydrate, as a function of RQ

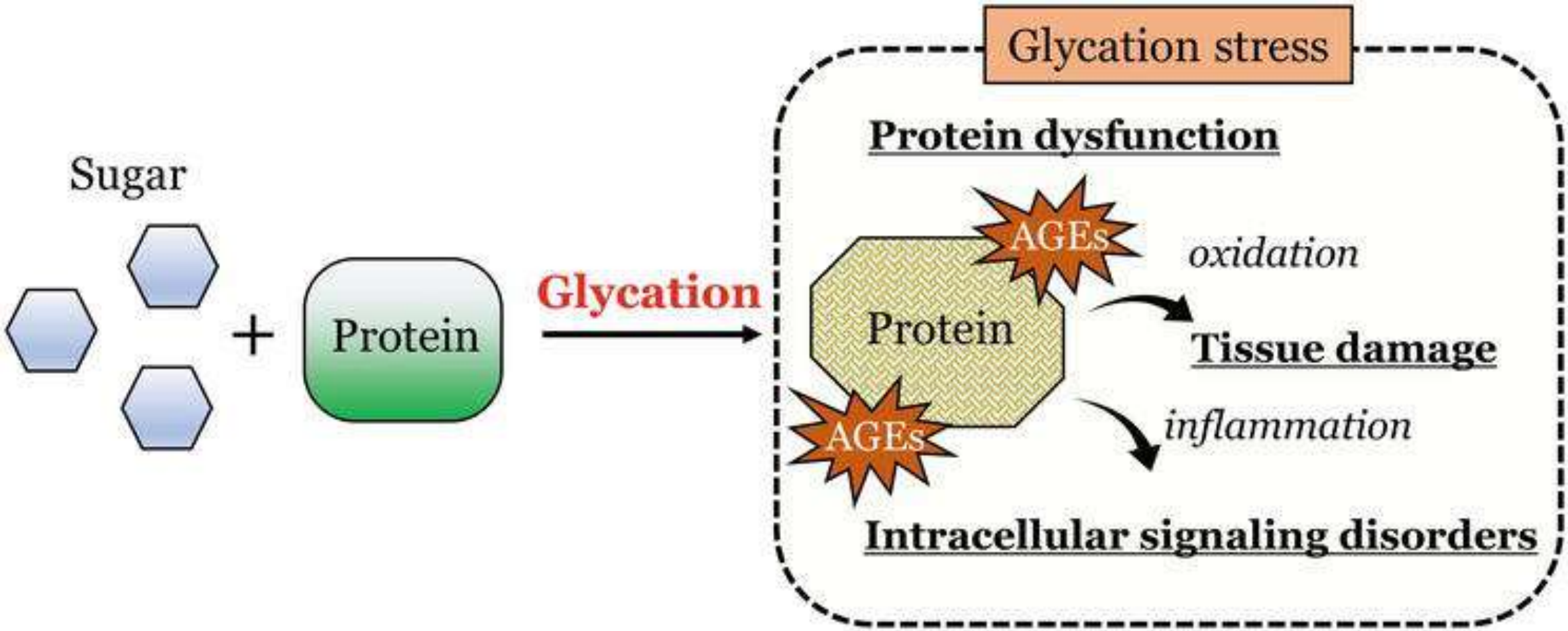
Where RQ = $CO_{2\ Eliminated}$ / $O_{2\ Consumed}$





All non-fiber carbs are broken down into simple sugars like glucose and stored as glycogen. All fats are broken down into fatty acids, then stored as triglycerides in fat cells (adipocytes).


GASTROINTESTINAL
PROTEIN
FIBER
CARBS
FAT
PROTEIN
CARBS
FAT
MUSCLE
BLOOD
ADIPOSE
LIVER
PROTEIN
GLUCOSE
CARBS
CARBS
GLUCOSE
FAT
FAT
FAT
FAT
FAT
FUEL SENSOR
CARBS
GLUCOSE
CARBS
FAT
GLUCAGON
INSULIN
METABOLISM
PANCREAS
ATP
+ CO2
+ H2O
ALPHA
BETA


Sugar
Protein
Glycation
Glycation stress
Protein dysfunction
AGEs
Protein
AGEs
oxidation
Tissue damage
inflammation
Intracellular signaling disorders

ต้องใช้น้ำตาลเท่าไหร่?

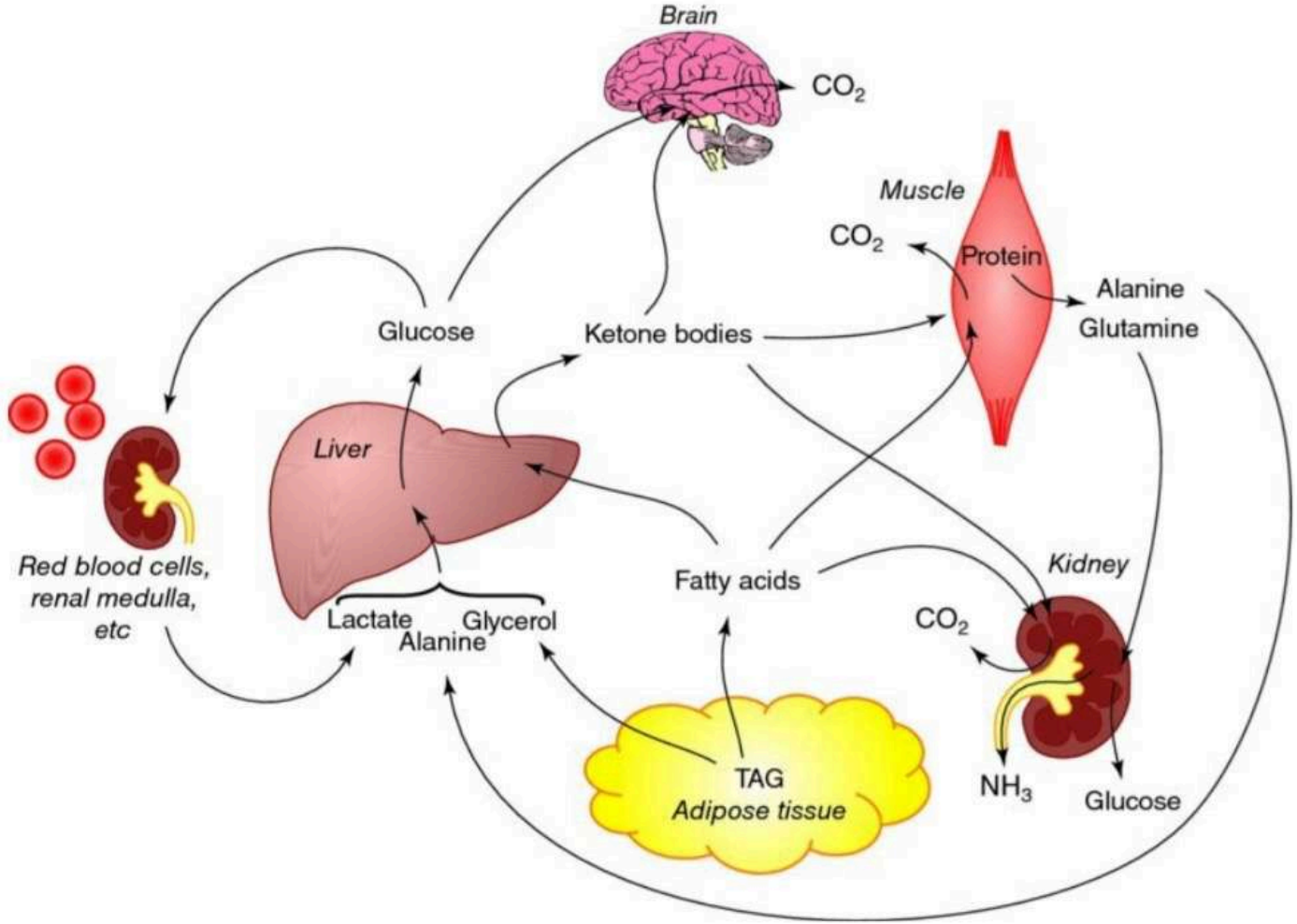
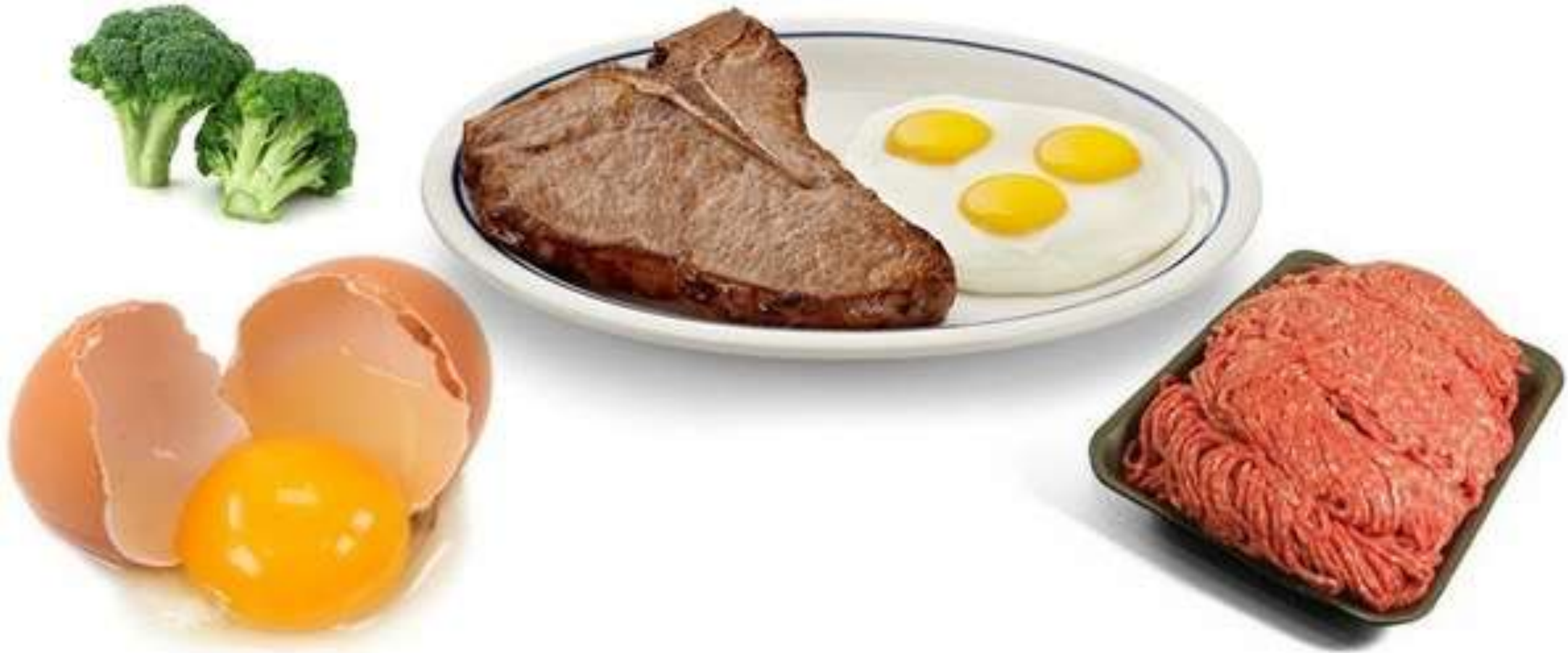


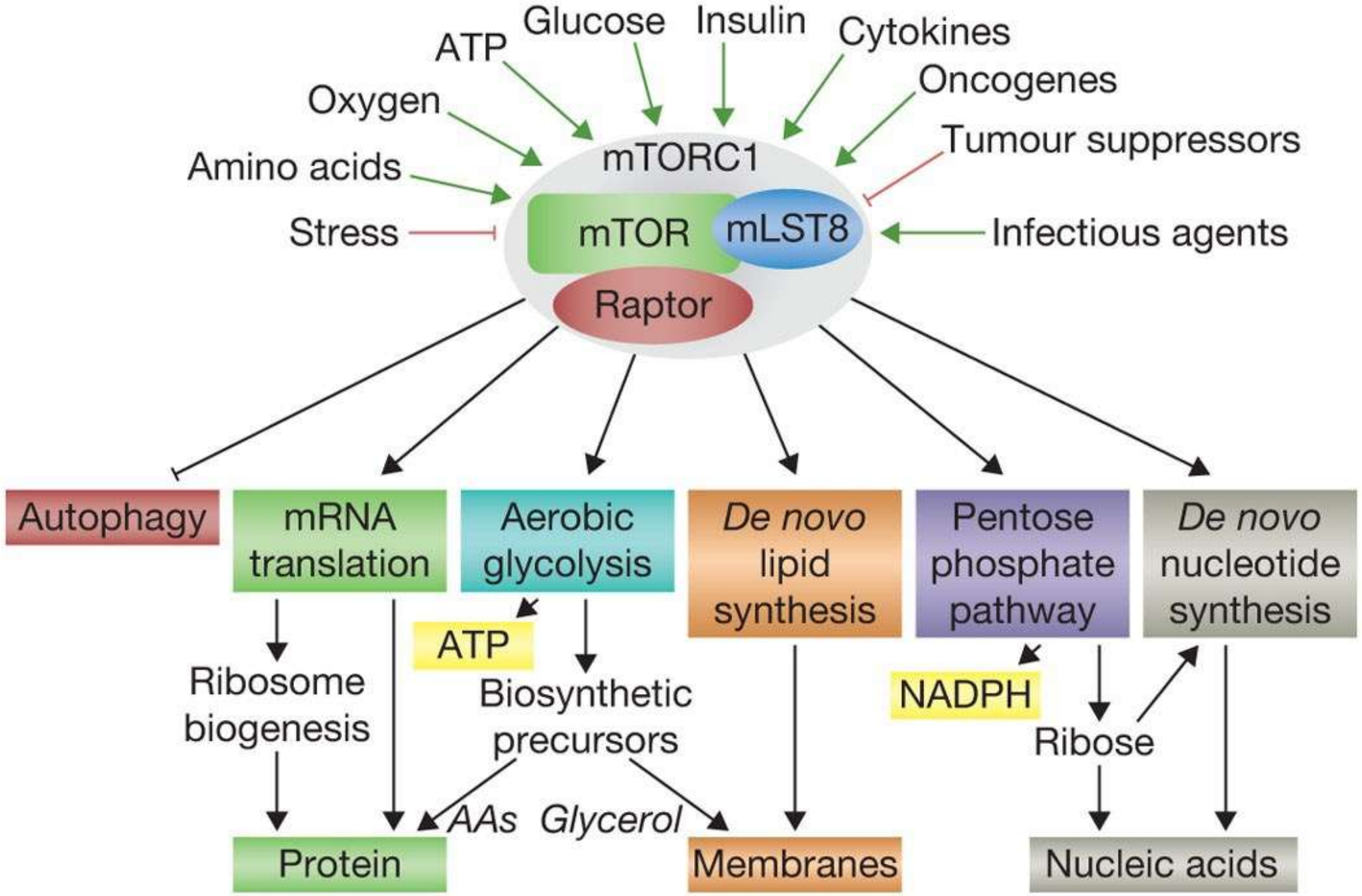
Figure 9.6 Major fuel flows in prolonged starvation. Protein (especially that in muscle) and glycerol (from lipolysis of triacylglycerol in adipose tissue) are the only long-term sources of glucose. The complete oxidation of glucose is decreased by the production of ketone bodies, which serve as an alternative fuel, for example, for the brain. Those tissues that cannot oxidise ketone bodies or non-esterified fatty acids and must therefore use glucose (e.g. red blood cells, renal medulla) produce lactate, which is 'recycled' in gluconeogenesis. The major source of fuel for oxidation is thus adipose tissue triacylglycerol (TAG), providing fuel both in the form of non-esterified fatty acids and (via the liver) ketone bodies.

There are NO essential carbohydrates!

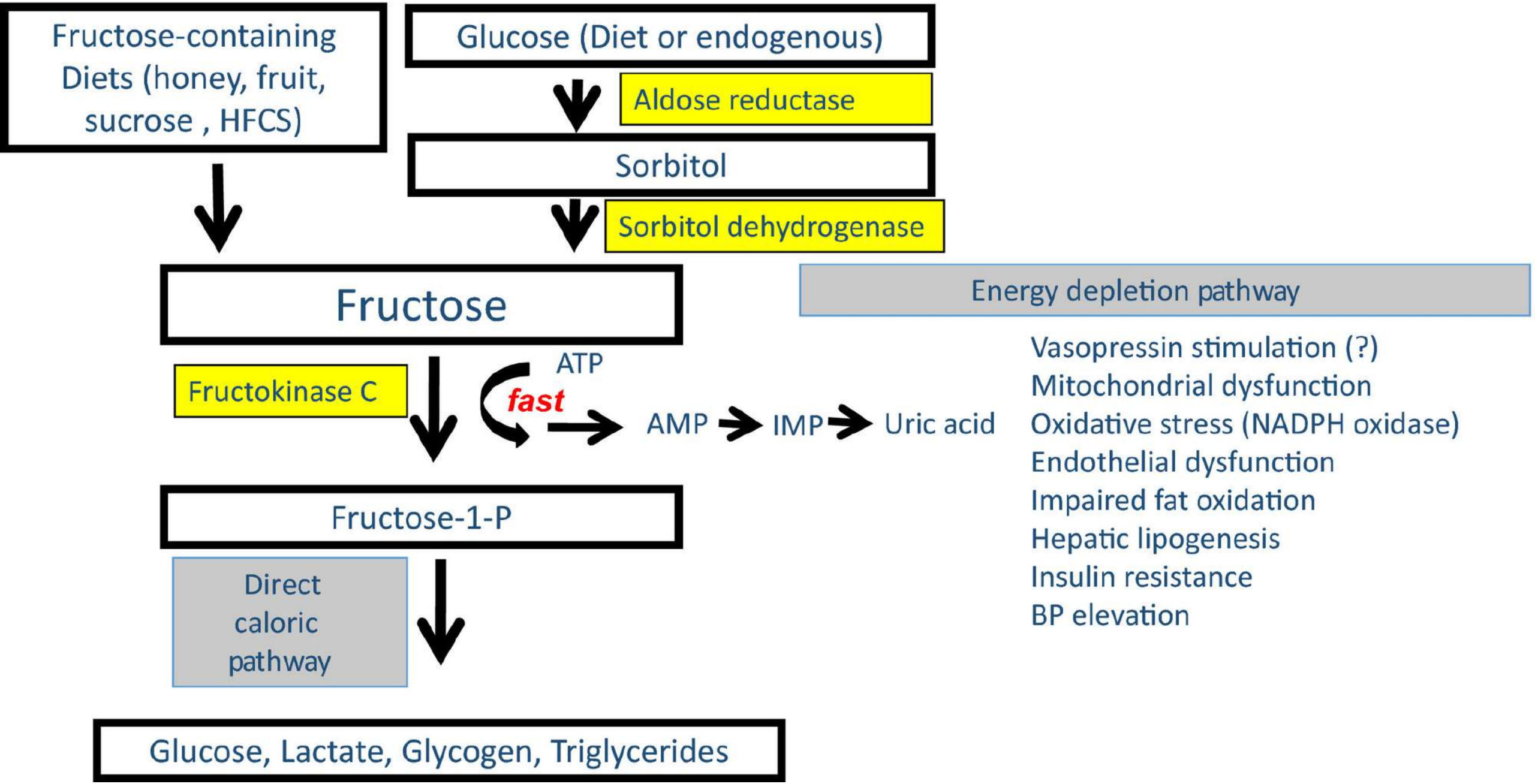
So, why do you "need" to eat carbs?

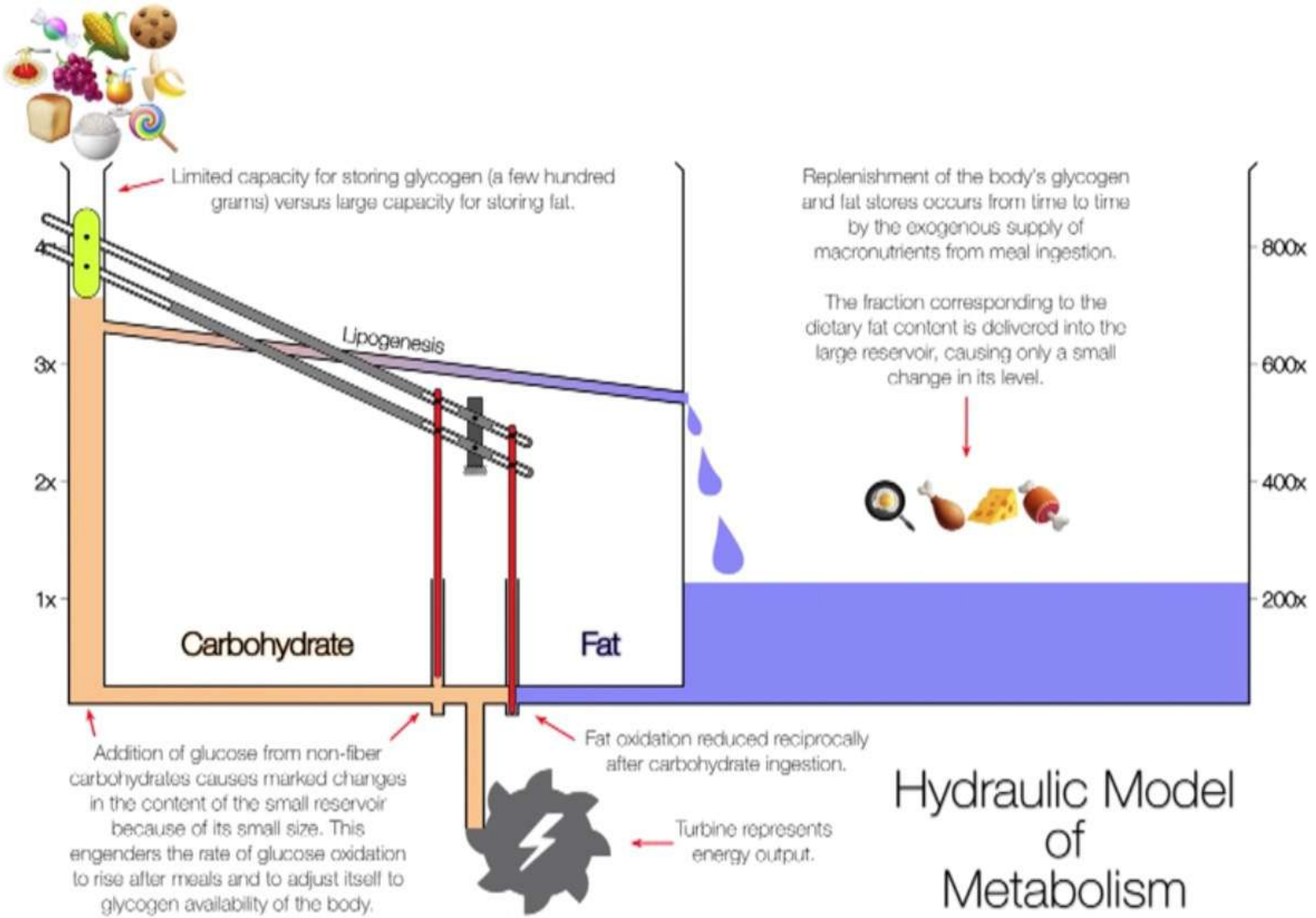


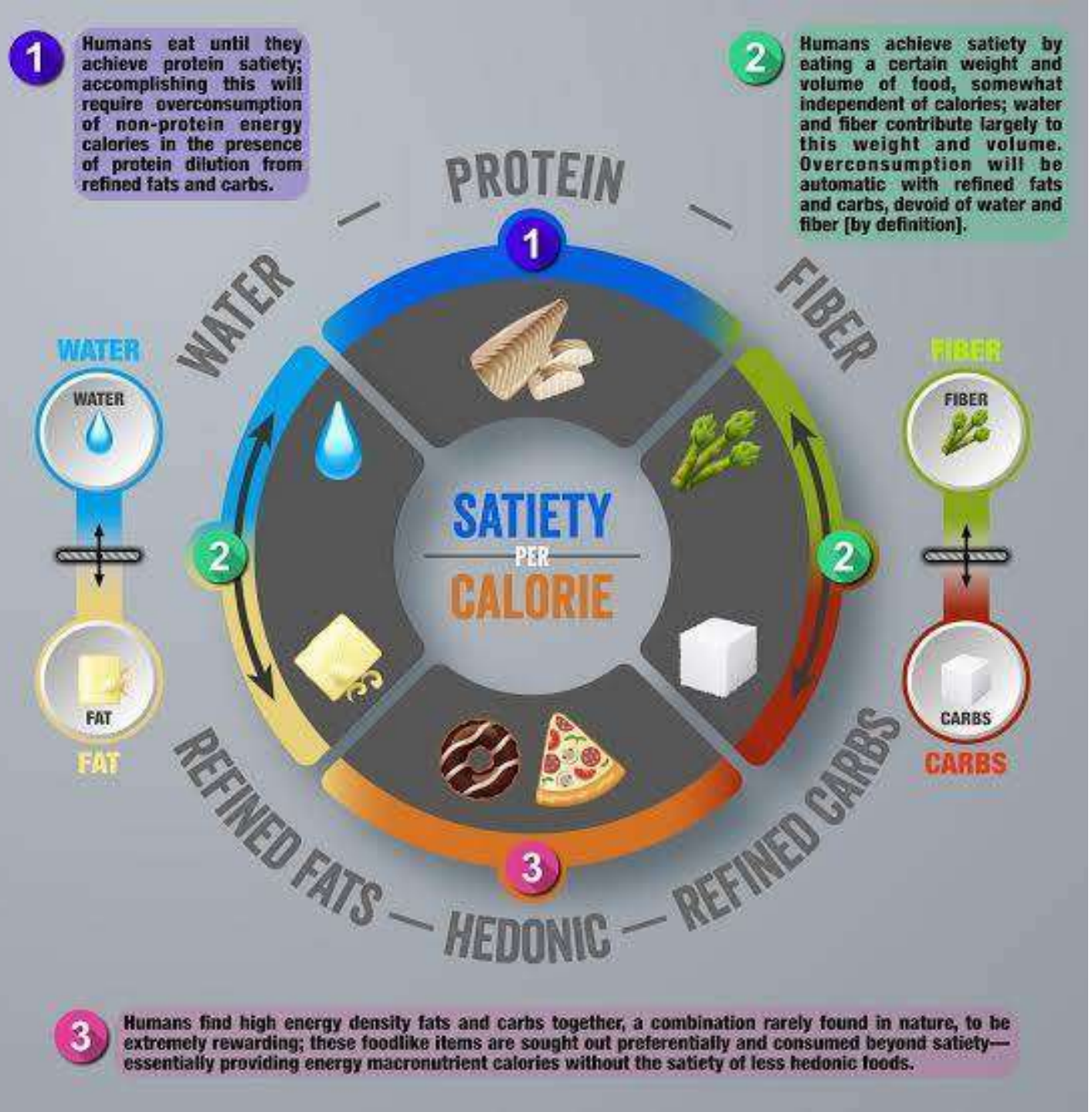
What happens when we eat ?



Anabolic cell growth and proliferation


Fructose-containing Diets (honey, fruit, sucrose , HFCS)
Glucose (Diet or endogenous)
Aldose reductase
Sorbitol
Sorbitol dehydrogenase
Fructose
Energy depletion pathway
Fructokinase C
ATP
fast
AMP
IMP
Uric acid
Vasopressin stimulation (?)
Mitochondrial dysfunction
Oxidative stress (NADPH oxidase)
Endothelial dysfunction
Impaired fat oxidation
Hepatic lipogenesis
Insulin resistance
BP elevation
Fructose-1-P
Direct caloric pathway
Glucose, Lactate, Glycogen, Triglycerides


Limited capacity for storing glycogen (a few hundred grams) versus large capacity for storing fat.
Replenishment of the body's glycogen and fat stores occurs from time to time by the exogenous supply of macronutrients from meal ingestion.
The fraction corresponding to the dietary fat content is delivered into the large reservoir, causing only a small change in its level.
Lipogenesis
4x
3x
2x
1x
800x
600x
400x
200x
Carbohydrate
Fat
Addition of glucose from non-fiber carbohydrates causes marked changes in the content of the small reservoir because of its small size. This engenders the rate of glucose oxidation to rise after meals and to adjust itself to glycogen availability of the body.
Fat oxidation reduced reciprocally after carbohydrate ingestion.
Turbine represents energy output.
Hydraulic Model of Metabolism


1
Humans eat until they achieve protein satiety; accomplishing this will require overconsumption of non-protein energy calories in the presence of protein dilution from refined fats and carbs.
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Humans achieve satiety by eating a certain weight and volume of food, somewhat independent of calories; water and fiber contribute largely to this weight and volume. Overconsumption will be automatic with refined fats and carbs, devoid of water and fiber [by definition].
PROTEIN
WATER
FIBER
REFINED FATS
HEDONIC
REFINED CARBS
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WATER
FAT
FAT
FIBER
FIBER
CARBS
CARBS
SATIETY
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CALORIE
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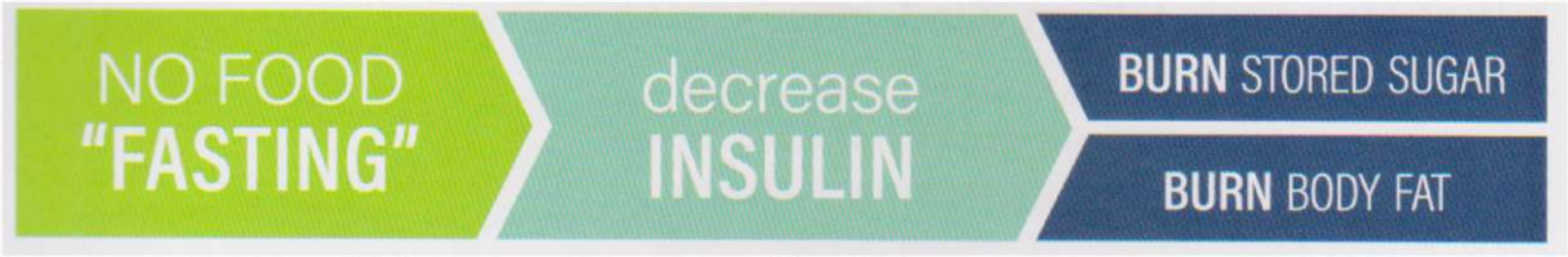
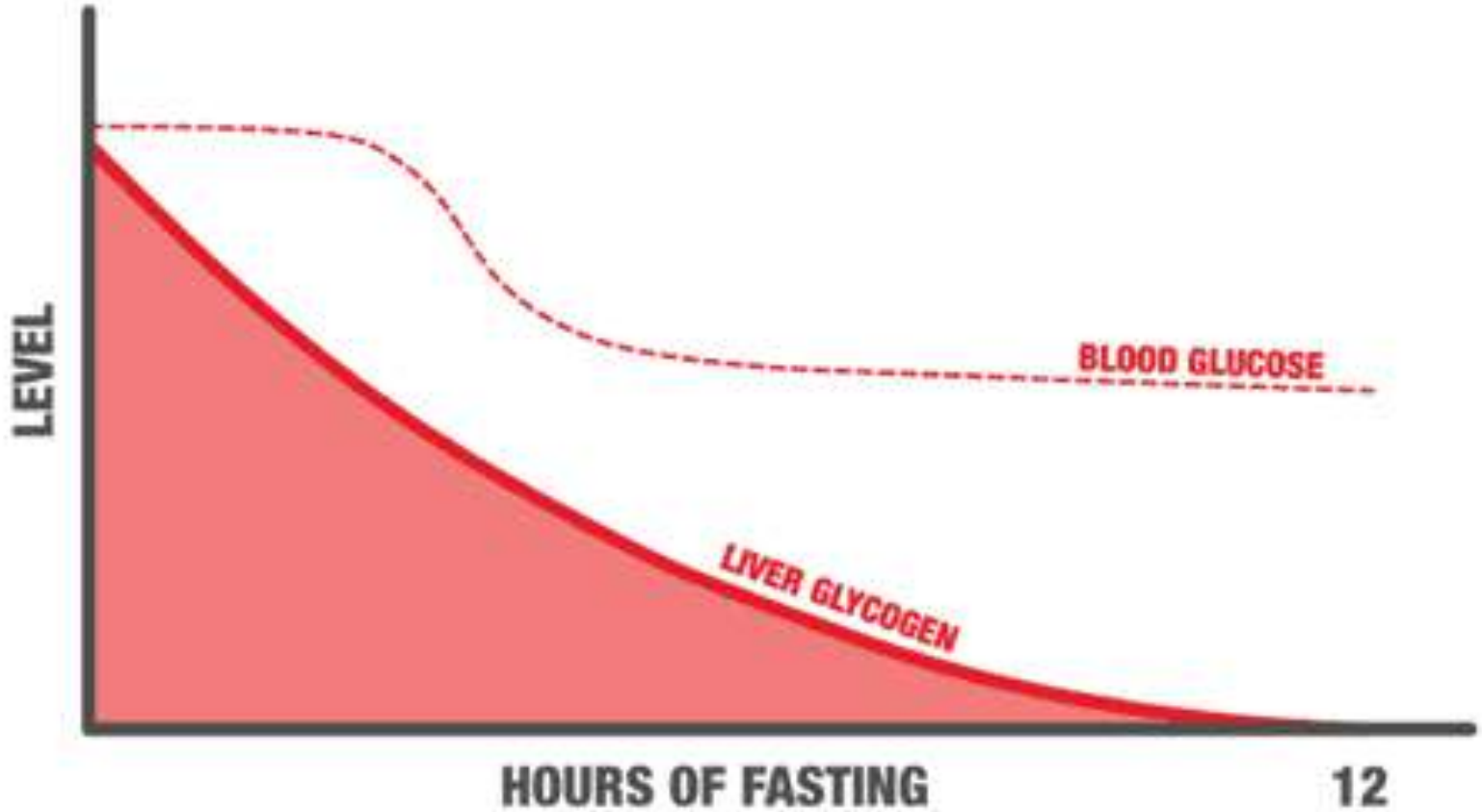
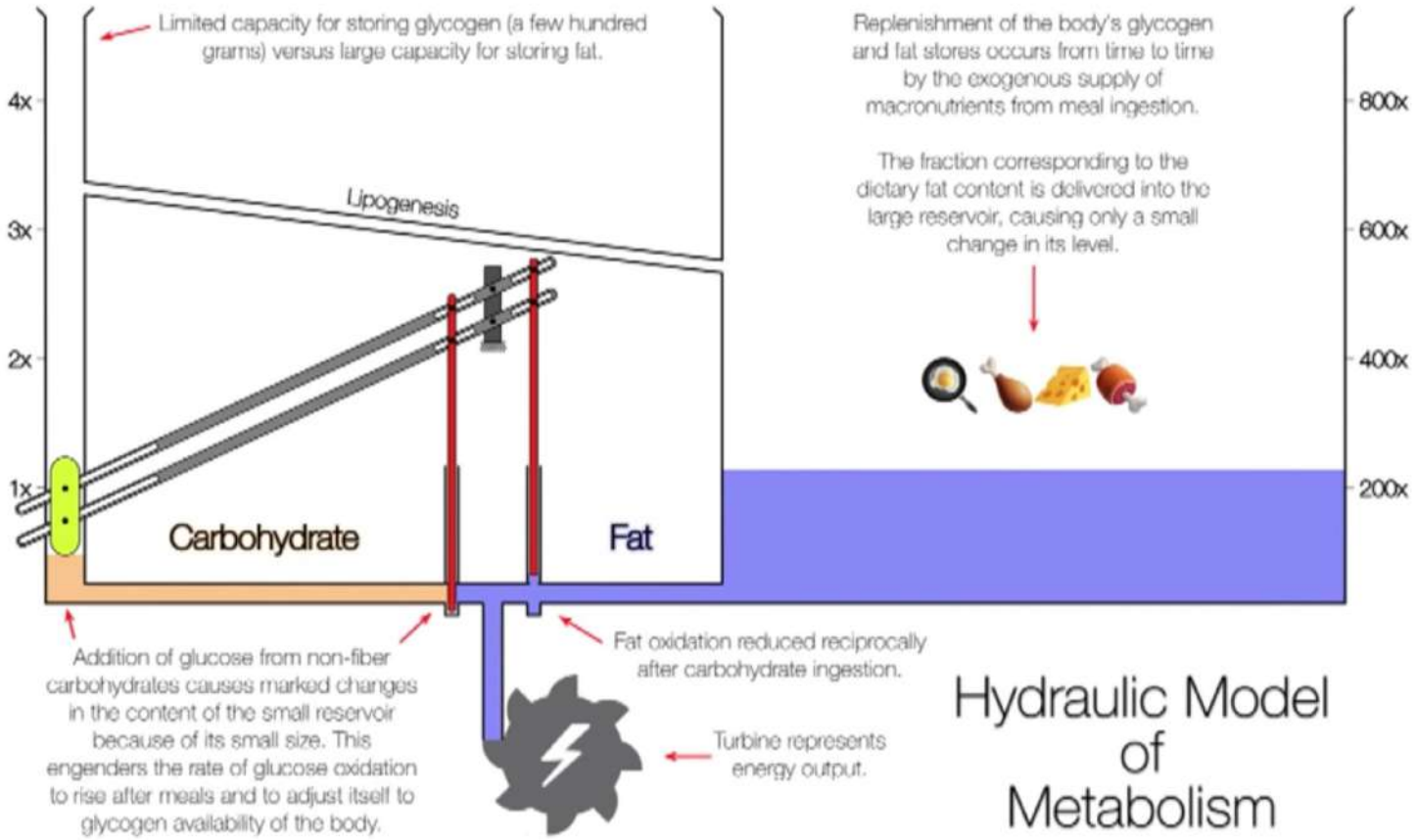
Humans find high energy density fats and carbs together, a combination rarely found in nature, to be extremely rewarding; these foodlike items are sought out preferentially and consumed beyond satiety—essentially providing energy macronutrient calories without the satiety of less hedonic foods.

What happens when we fast ?




LEVEL
BLOOD GLUCOSE
LIVER GLYCOGEN
HOURS OF FASTING
12


Limited capacity for storing glycogen (a few hundred grams) versus large capacity for storing fat.
4x
3x
2x
1x
Lipogenesis
Carbohydrate
Fat
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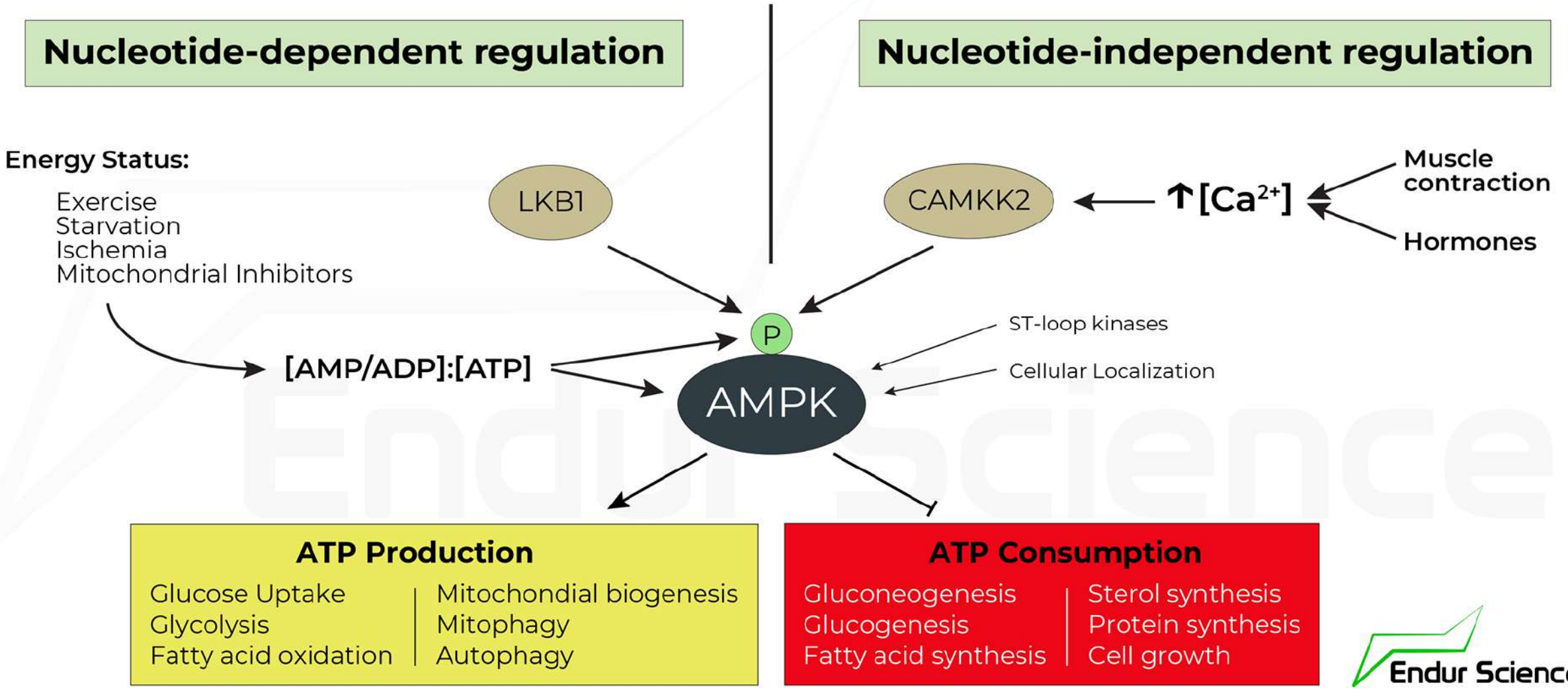
Fat oxidation reduced reciprocally after carbohydrate ingestion.
Turbine represents energy output.
Replenishment of the body's glycogen and fat stores occurs from time to time by the exogenous supply of macronutrients from meal ingestion.
The fraction corresponding to the dietary fat content is delivered into the large reservoir, causing only a small change in its level.
800x
600x
400x
200x
Hydraulic Model of Metabolism

AMPK, an energy stress sensor

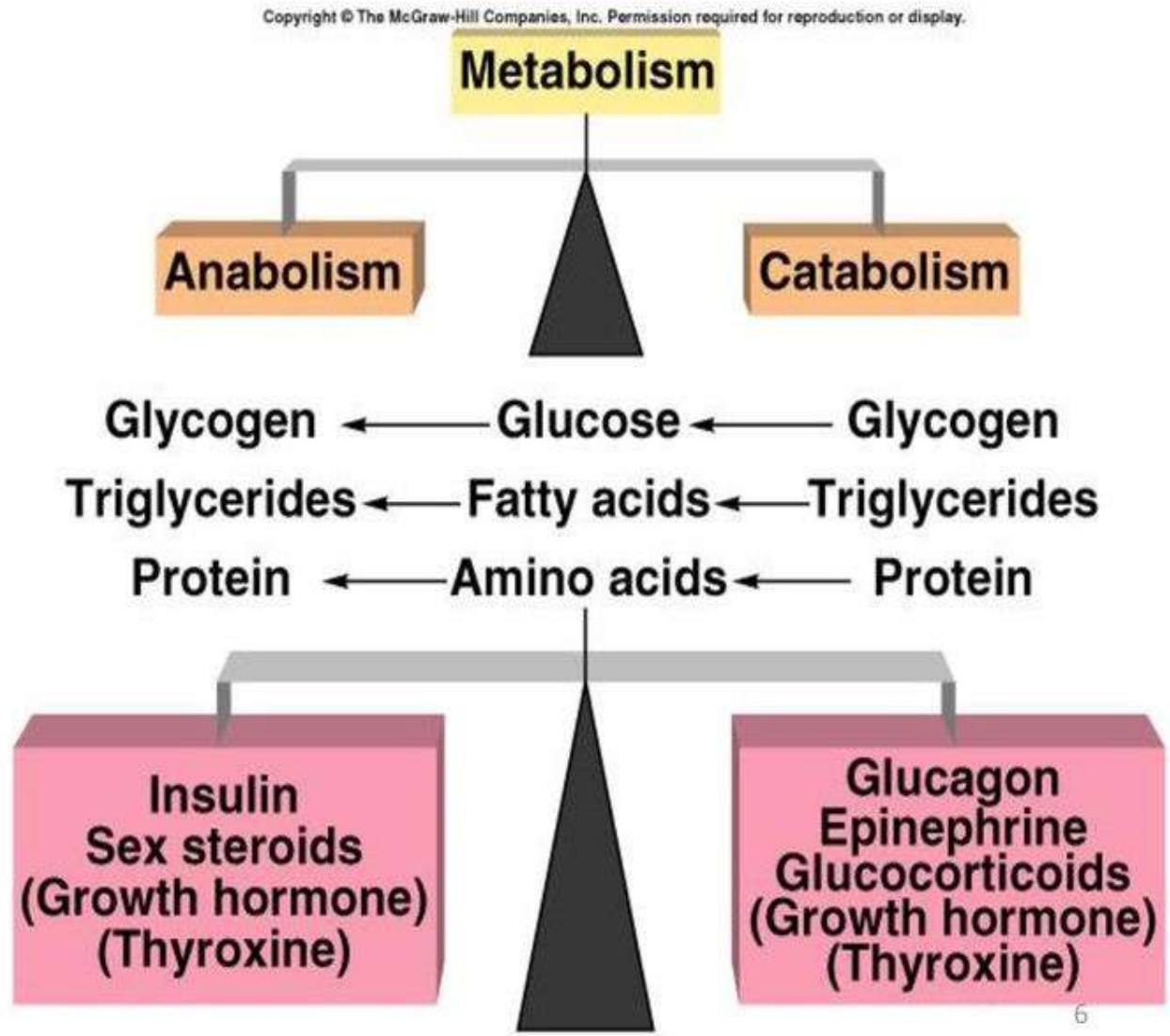


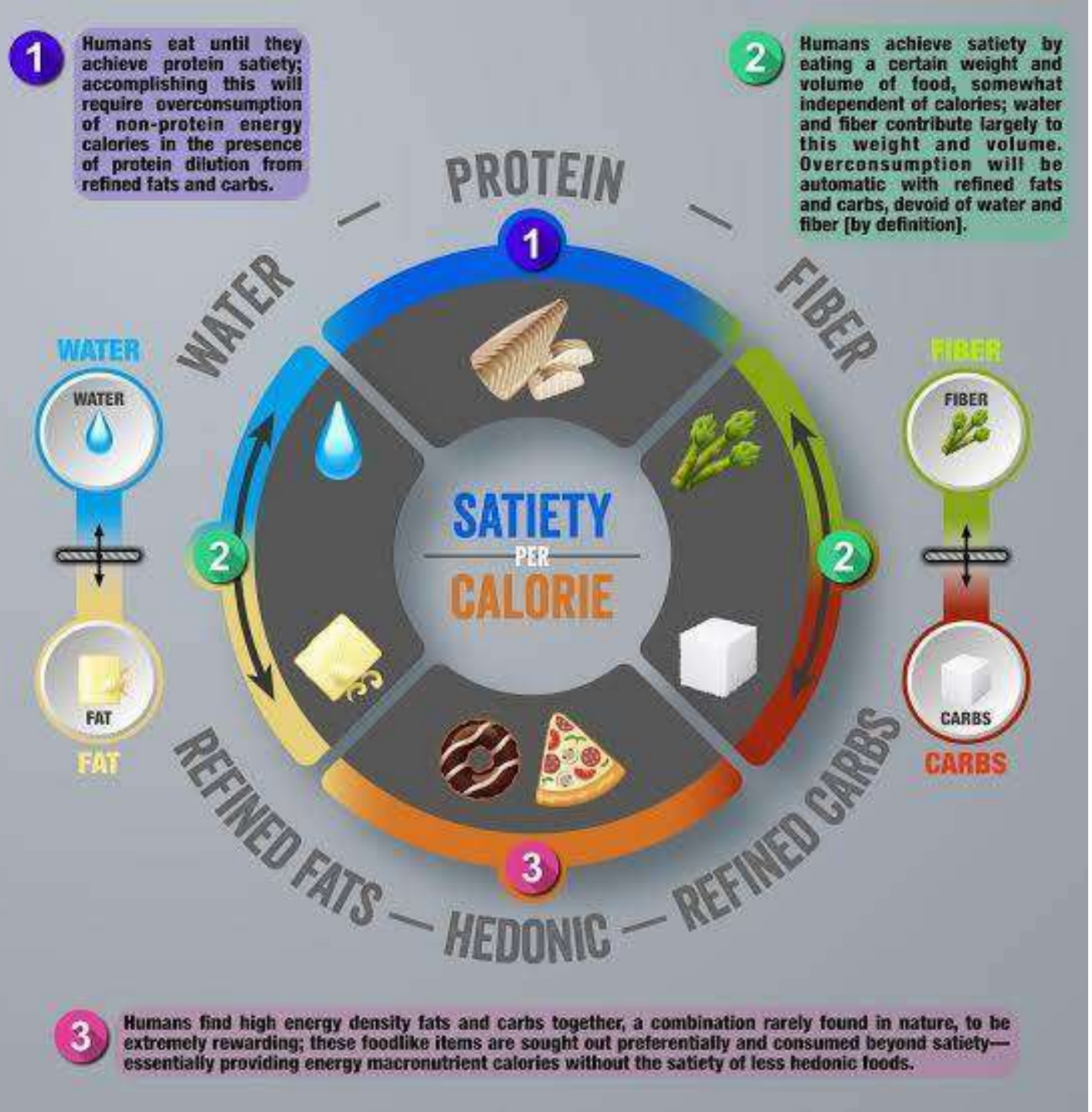
Endur Science

Adapted from: Garcia, Daniel, and Reuben J. Shaw. 2017. "AMPK: Mechanisms of Cellular Energy Sensing and Restoration of Metabolic Balance." Molecular Cell 66 (6): 789–800.

Balance Between Anabolism and Catabolism

- The rate of deposit and withdrawal of energy substrates, and the conversion of 1 type of energy substrate into another; are regulated by hormones.

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FIBER
FIBER
CARBS
CARBS
SATIETY
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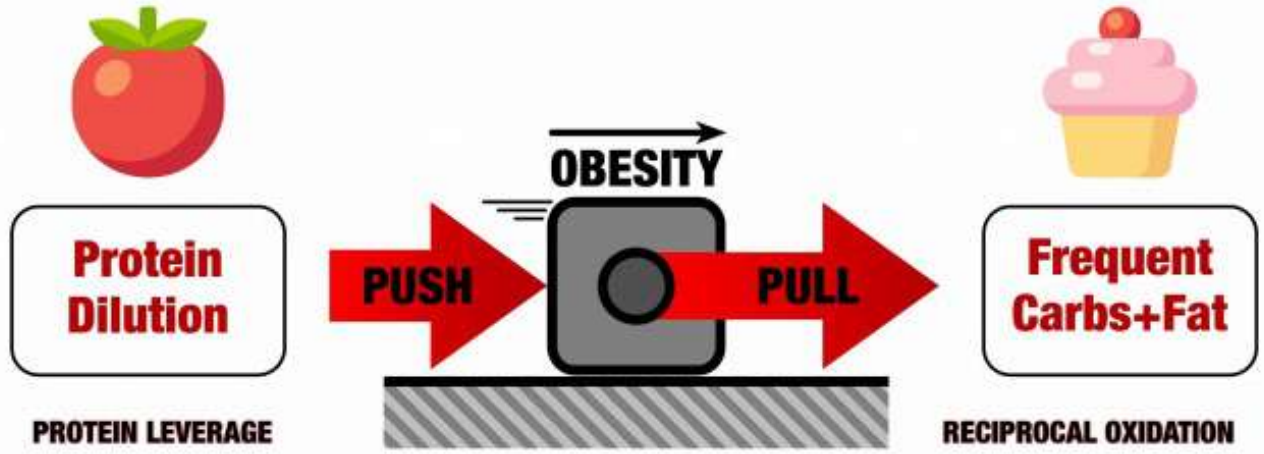
3
Humans find high energy density fats and carbs together, a combination rarely found in nature, to be extremely rewarding; these foodlike items are sought out preferentially and consumed beyond satiety—essentially providing energy macronutrient calories without the satiety of less hedonic foods.

Obesity is simultaneously pushed forward by the dilution of protein and minerals, and pulled forward by the frequent co-ingestion of carbohydrates and fat.



Dilution of protein and minerals with carbohydrates and/or fats requires overingestion of this non-protein energy to achieve the same protein satiety.

Carbs displace fat oxidation and fat passively accumulates. Subsequent glucose dependence and inadequate fat adaptation drives hunger.

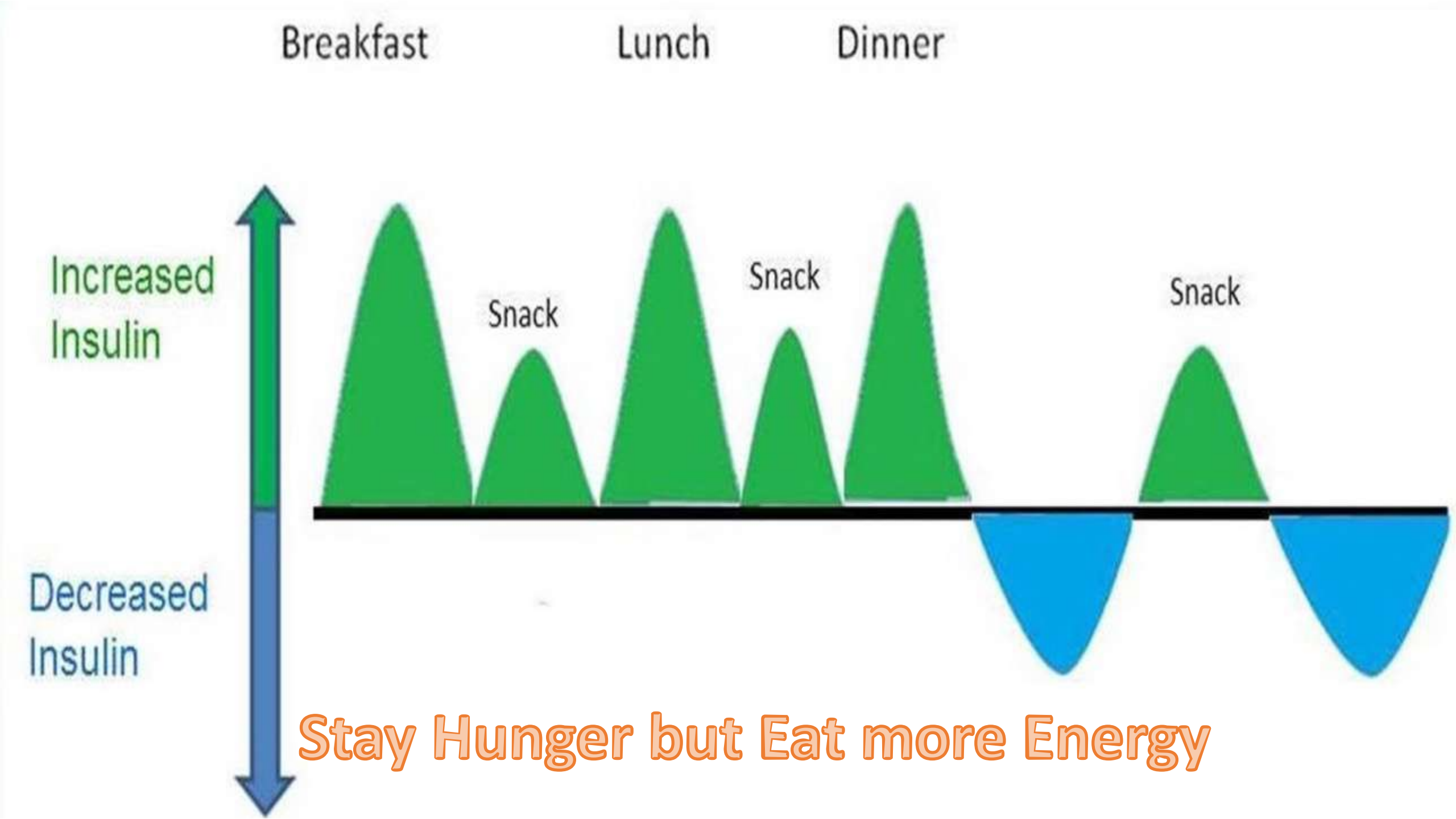
Potential Solutions:

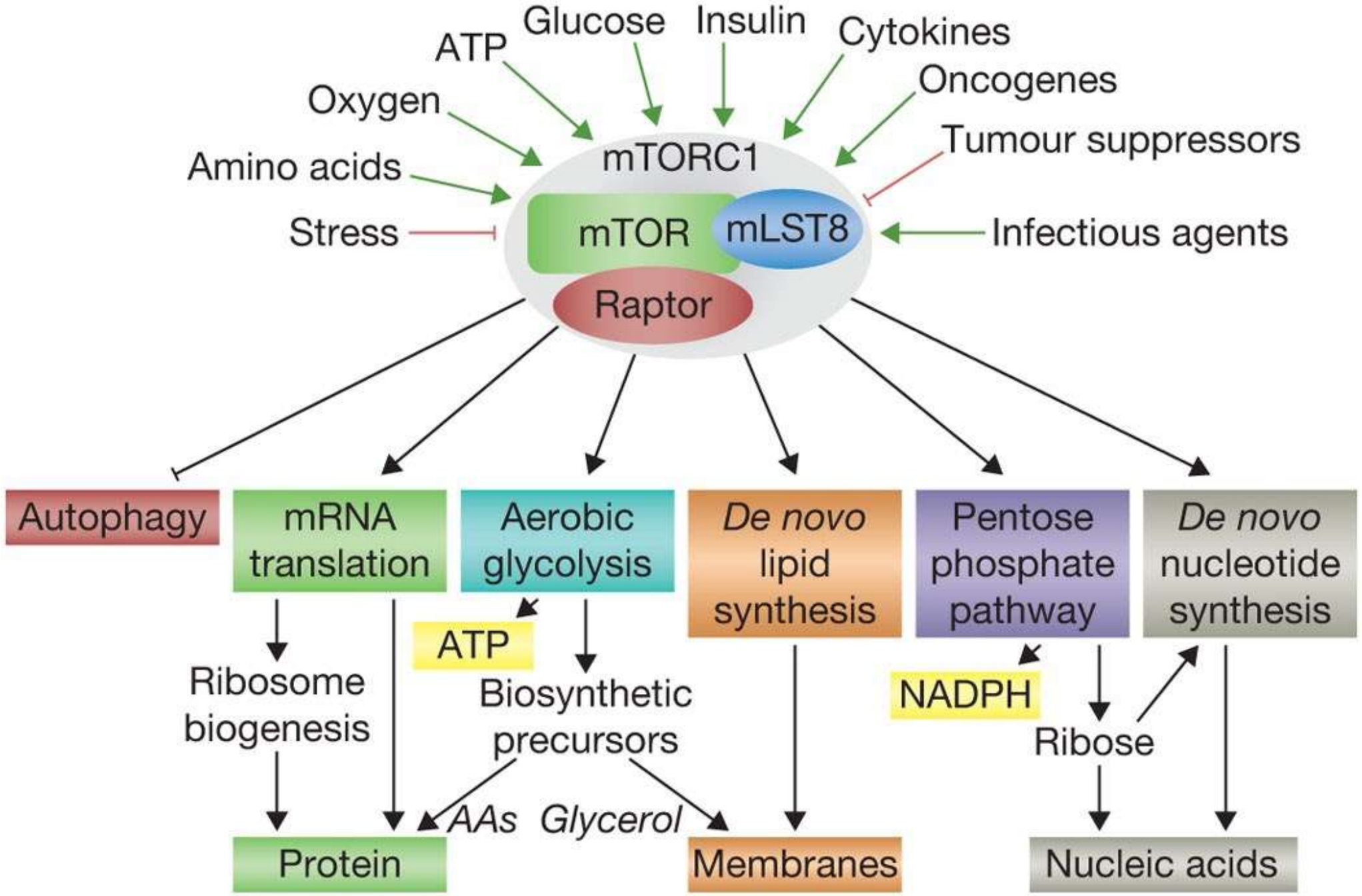
1. Low carbohydrate diet.
2. Low fat diet.
3. High protein diet.
4. Avoid refined carbohydrates and fats with any whole foods diet.

Potential Solutions:

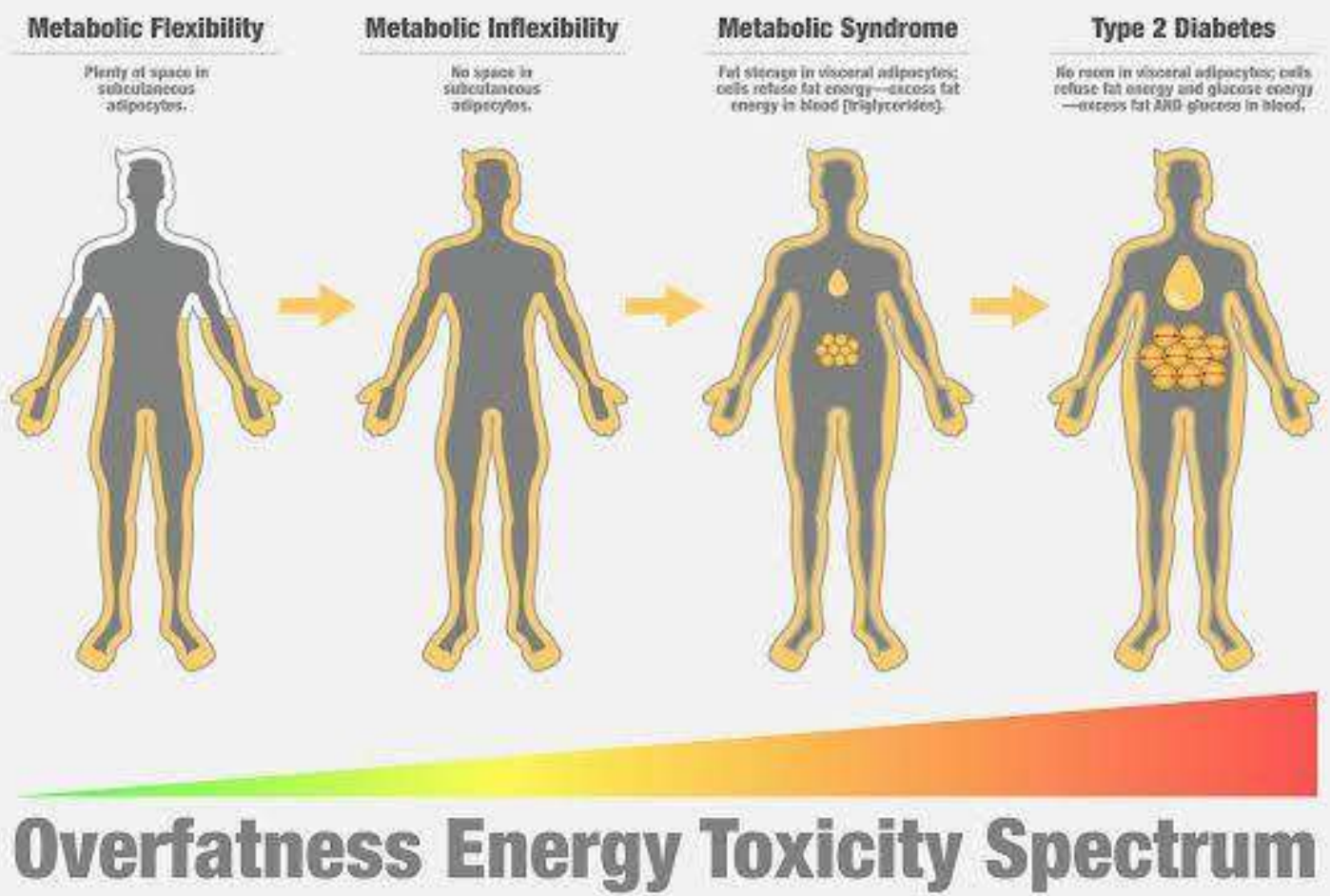
1. Low carbohydrate diet.
2. Low fat diet.
3. High protein diet.
4. Any whole foods diet.
5. Eat less frequently (intermittent).

Nutrient hunger from protein dilution and the addictive nature of carbs and fats together both drive obesity.


Breakfast
Lunch
Dinner
Increased Insulin
Decreased Insulin
Snack
Snack
Snack
Stay Hunger but Eat more Energy



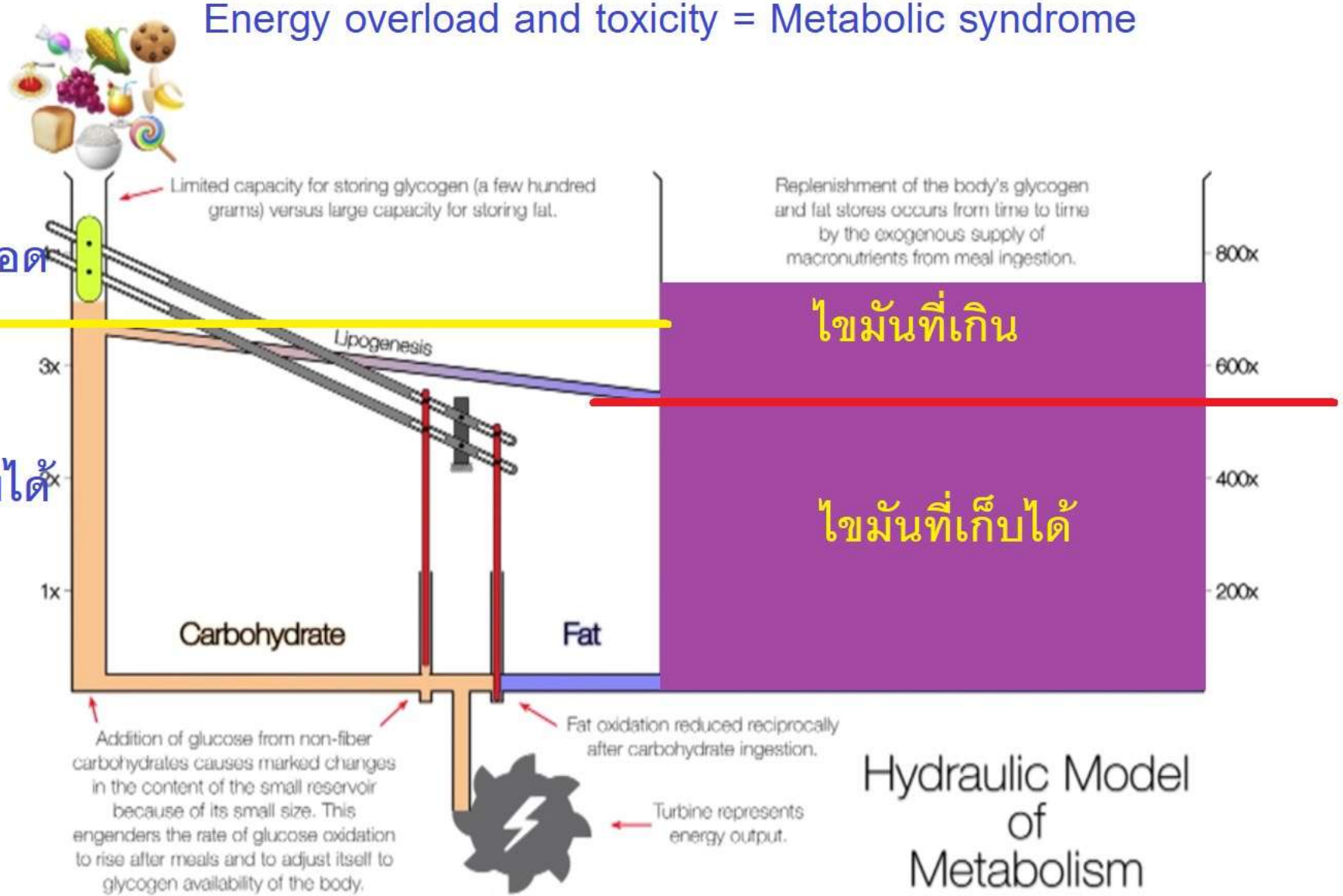
Anabolic cell growth and proliferation


Metabolic Flexibility
Plenty of space in subcutaneous adipocytes.
Metabolic Inflexibility
No space in subcutaneous adipocytes.
Metabolic Syndrome
Fat storage in visceral adipocytes; cells refuse fat energy—excess fat energy in blood (triglycerides).
Type 2 Diabetes
No room in visceral adipocytes; cells refuse fat energy and glucose energy —excess fat AND glucose in blood.
Overfatness Energy Toxicity Spectrum
THE P:E DIET

Energy overload and toxicity = Metabolic syndrome

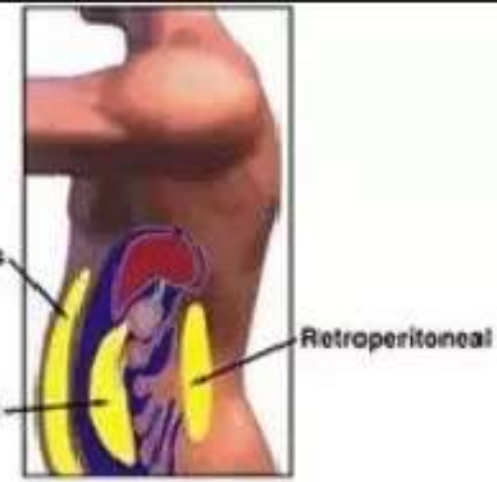
Glucose ที่เกิน
และสูงขึ้นในเลือด

ไกลโคเจนที่เก็บได้



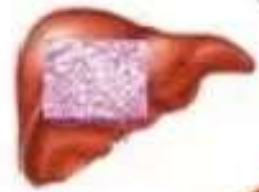
Cr.Ted Naiman,MD Modified by @DietDoctorThailand

Excess calories
(increased intake +/-
reduced energy expenditure)



Subcutaneous stores overwhelmed
(genes, ethnicity, ageing)

FAT
'Spill over'



Hepatic lipid accumulation

muscle

pancreatic beta cell

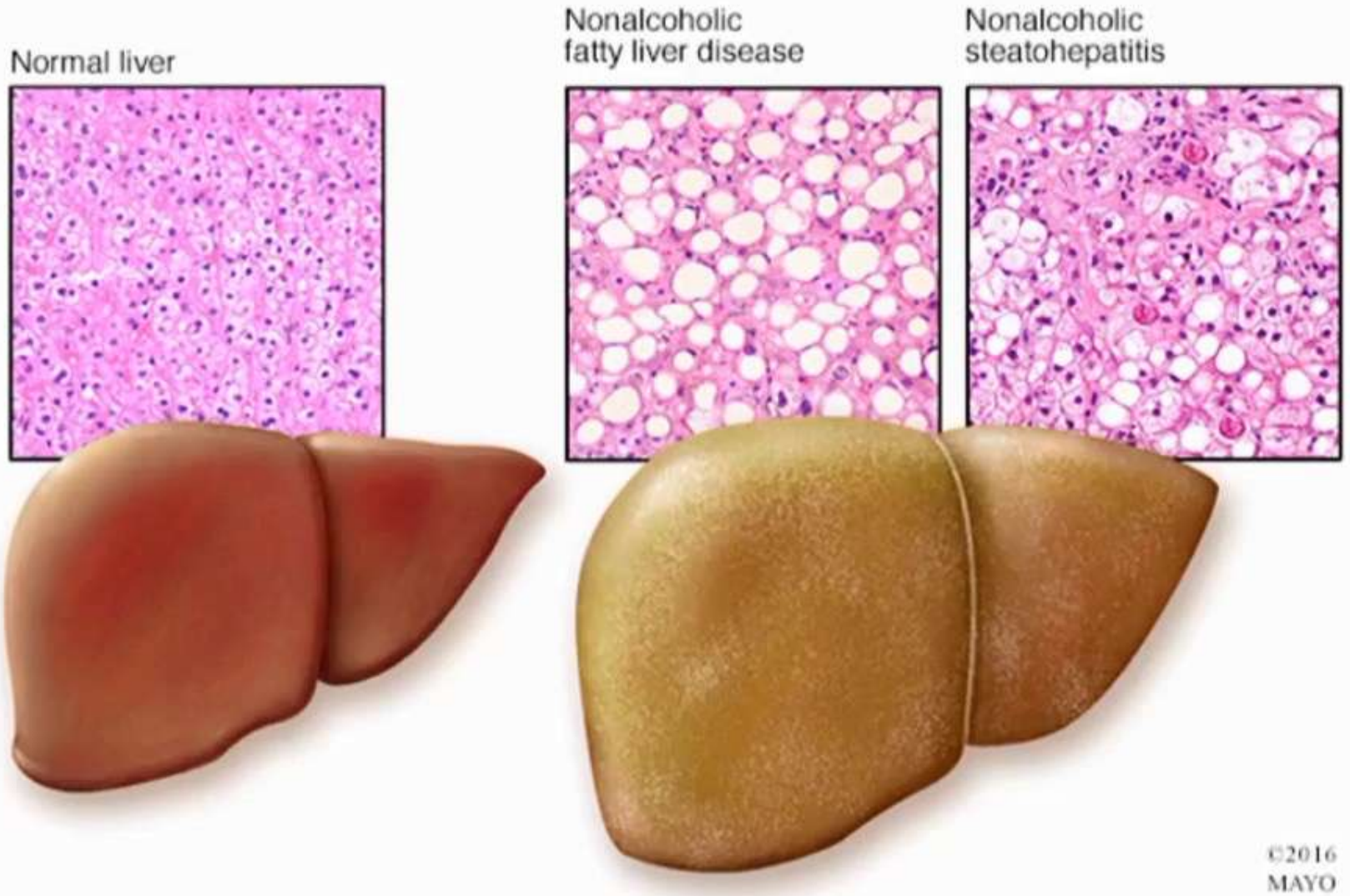
Perivascular fat ⇒
Endothelial dysfunction (altered blood flow)

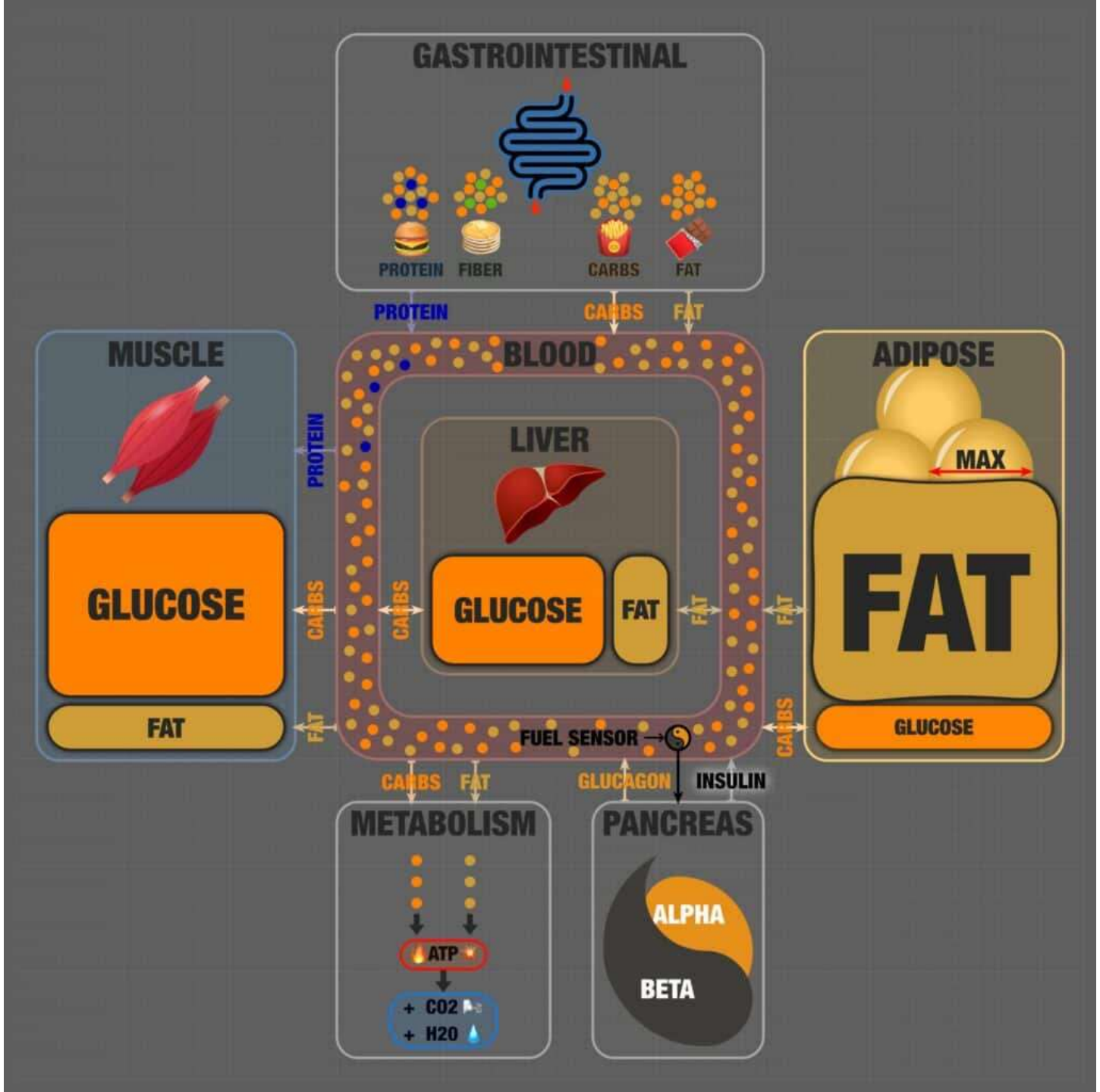
Insulin resistance

β cell dysfunction?

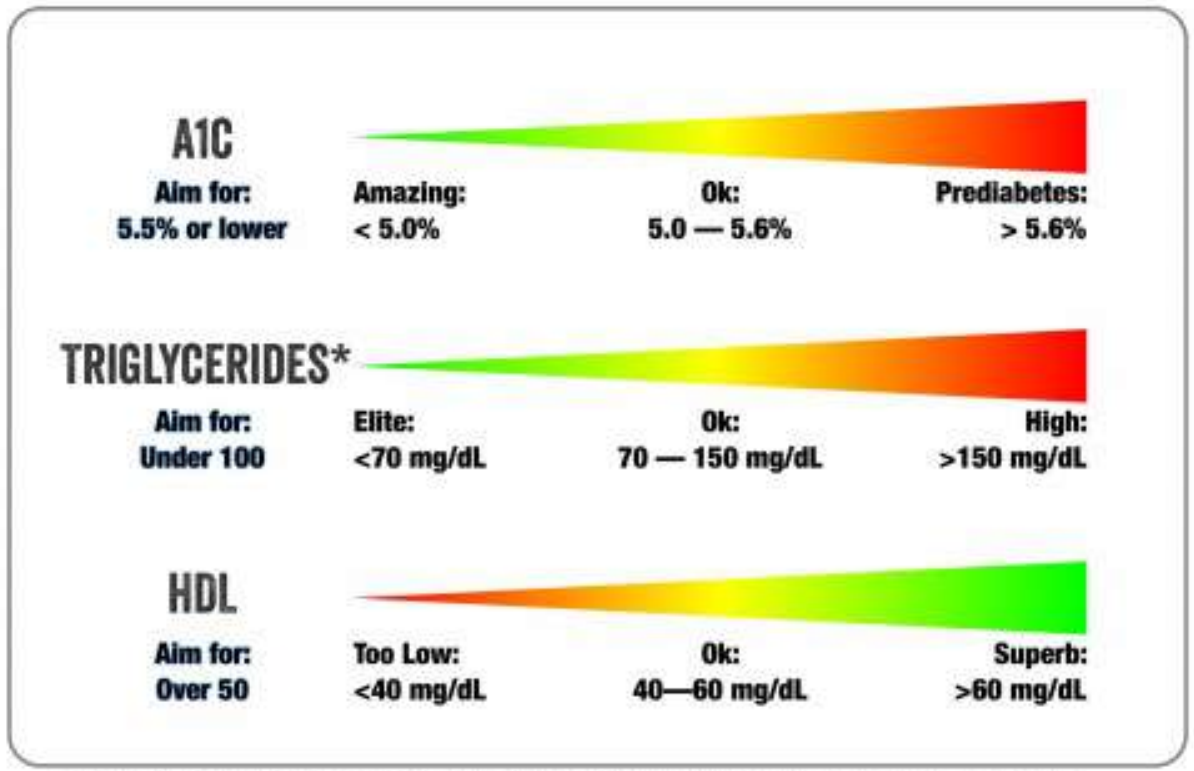
Hyperglycaemia

What causes fatty liver?




GASTROINTESTINAL
PROTEIN
FIBER
CARBS
FAT
PROTEIN
CARBS
FAT
MUSCLE
BLOOD
ADIPOSE
LIVER
MAX
PROTEIN
GLUCOSE
FAT
CARBS
CARBS
GLUCOSE
FAT
FAT
FAT
FAT
GLUCOSE
FAT
FUEL SENSOR
CARBS
CARBS
FAT
GLUCAGON
INSULIN
METABOLISM
PANCREAS
ATP
+ CO2
+ H2O
ALPHA
BETA

Top 3 basic lab tests:



*This is for FASTING triglycerides. Draw blood after consuming no calories for 9–12 hours.

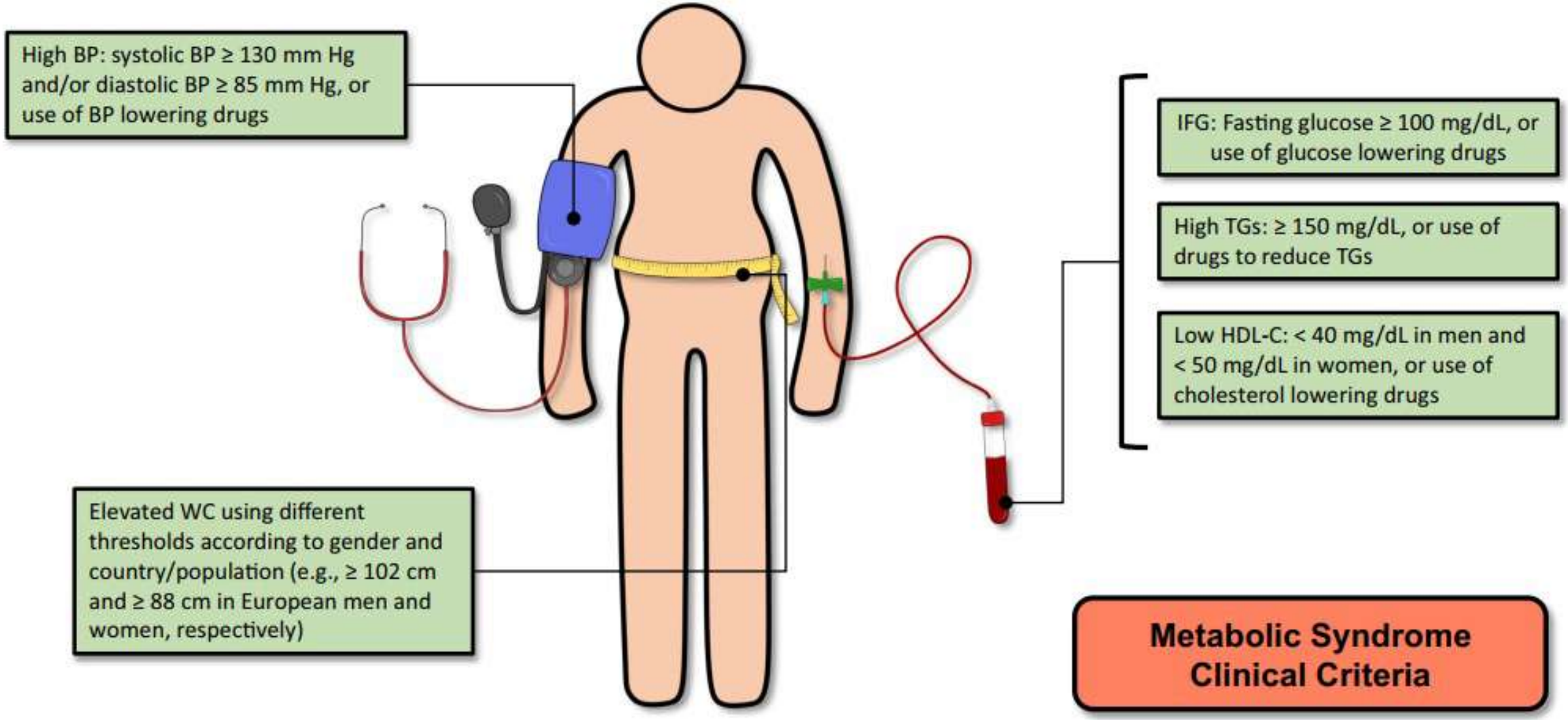


FIGURE 1 Metabolic syndrome diagnostic criteria. As reported by the International Diabetes Federation—American Heart Association/National Heart, Lung, and Blood Institute Joint Interim Statement definition.[15] BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; IFG, impaired fasting glucose; TGs, triglycerides; WC, waist circumference

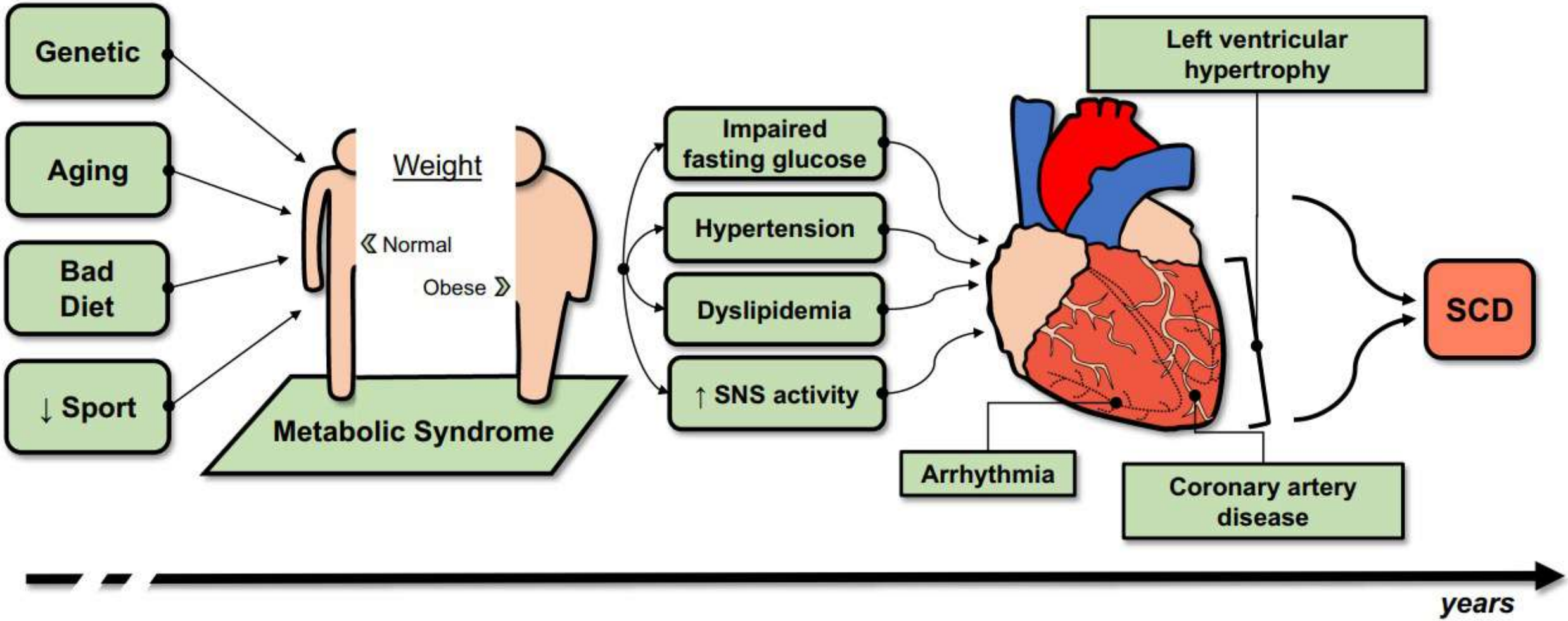
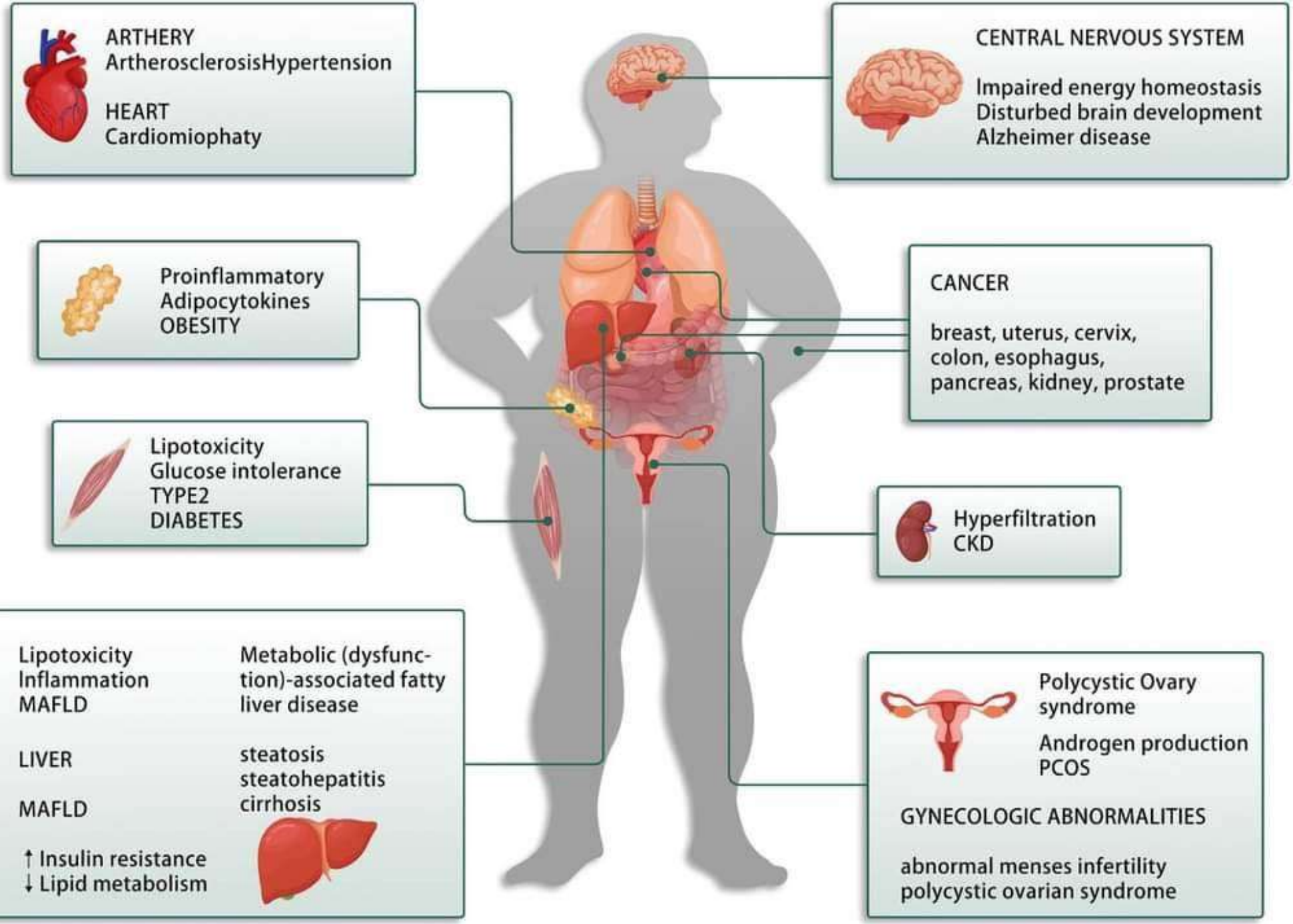
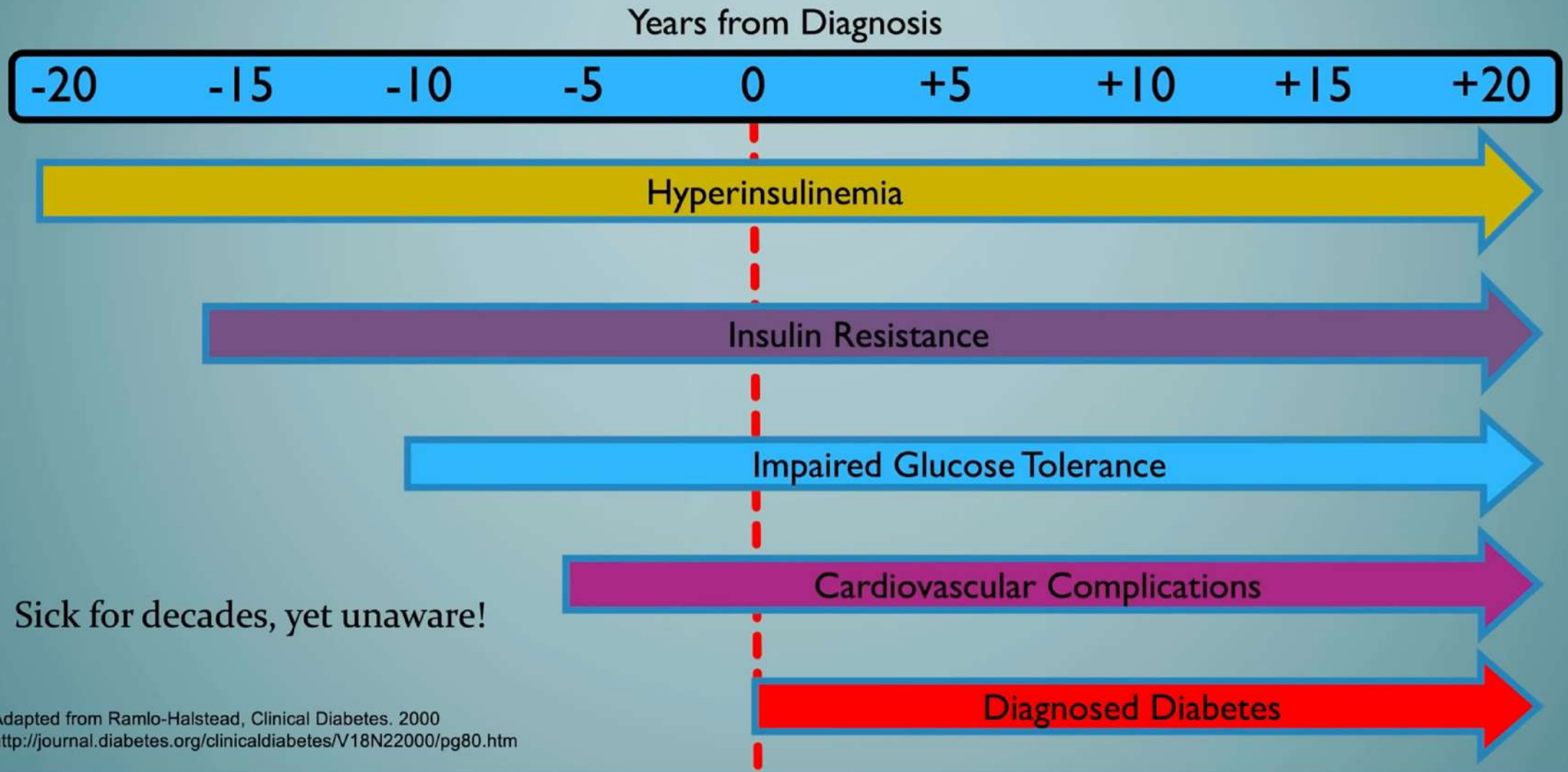


FIGURE 2 The detrimental effects of metabolic syndrome on the heart. Genetic determinants, but also ageing, diet and reduced physical activity concur in the development of MetS. Such individuals might appear lean even when 'metabolically obese'. Singularly and taken together, IFG, HTN, DysL and increased sympathetic activity are key elements in MetS pathophysiology with damaging effects on the heart favouring the development of CHD, LVH and AR, eventually causing SCD. However, such associations remain under-investigated, and the main underlying mechanisms are still poorly understood. SCD, sudden cardiac death; SNS, sympathetic nervous system


ARTHERY
ArtherosclerosisHypertension
HEART
Cardiomiophaty
CENTRAL NERVOUS SYSTEM
Impaired energy homeostasis
Disturbed brain development
Alzheimer disease
Proinflammatory
Adipocytokines
OBESITY
CANCER
breast, uterus, cervix,
colon, esophagus,
pancreas, kidney, prostate
Lipotoxicity
Glucose intolerance
TYPE2
DIABETES
Hyperfiltration
CKD
Lipotoxicity
Inflammation
MAFLD
Metabolic (dysfunc-
tion)-associated fatty
liver disease
LIVER
steatosis
steatohepatitis
cirrhosis
MAFLD
↑ Insulin resistance
↓ Lipid metabolism
Polycystic Ovary
syndrome
Androgen production
PCOS
GYNECOLOGIC ABNORMALITIES
abnormal menses infertility
polycystic ovarian syndrome

Natural History of Type 2 Diabetes



Sick for decades, yet unaware!

Adapted from Ramlo-Halstead, Clinical Diabetes. 2000
http://journal.diabetes.org/clinicaldiabetes/V18N22000/pg80.htm

Two Phases of Type 2 Diabetes

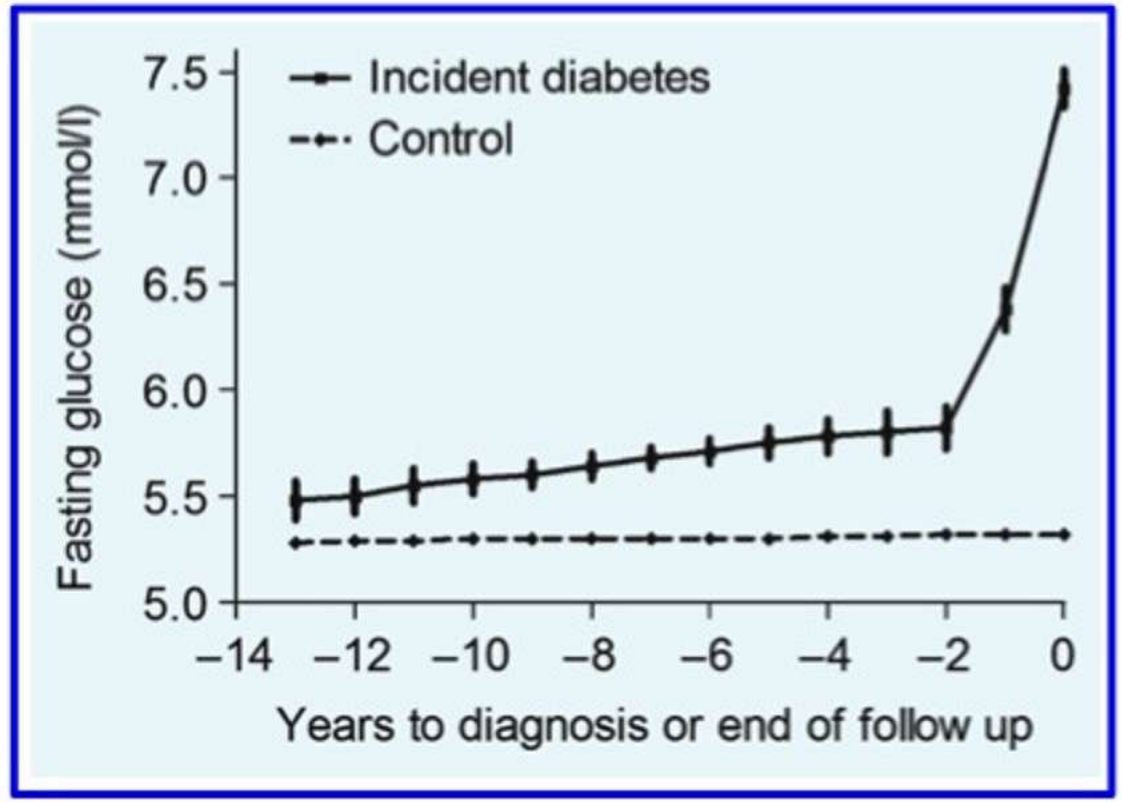
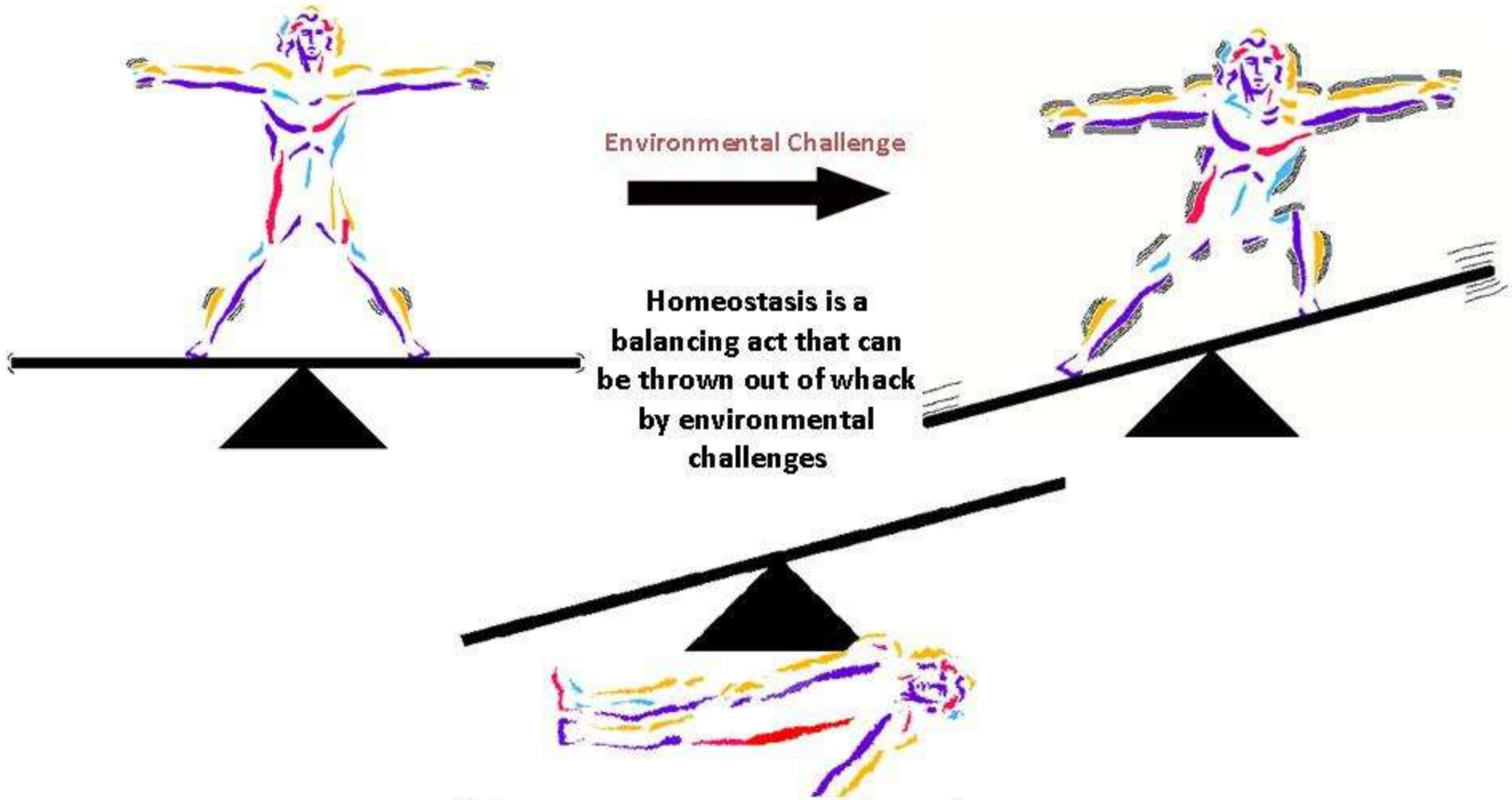
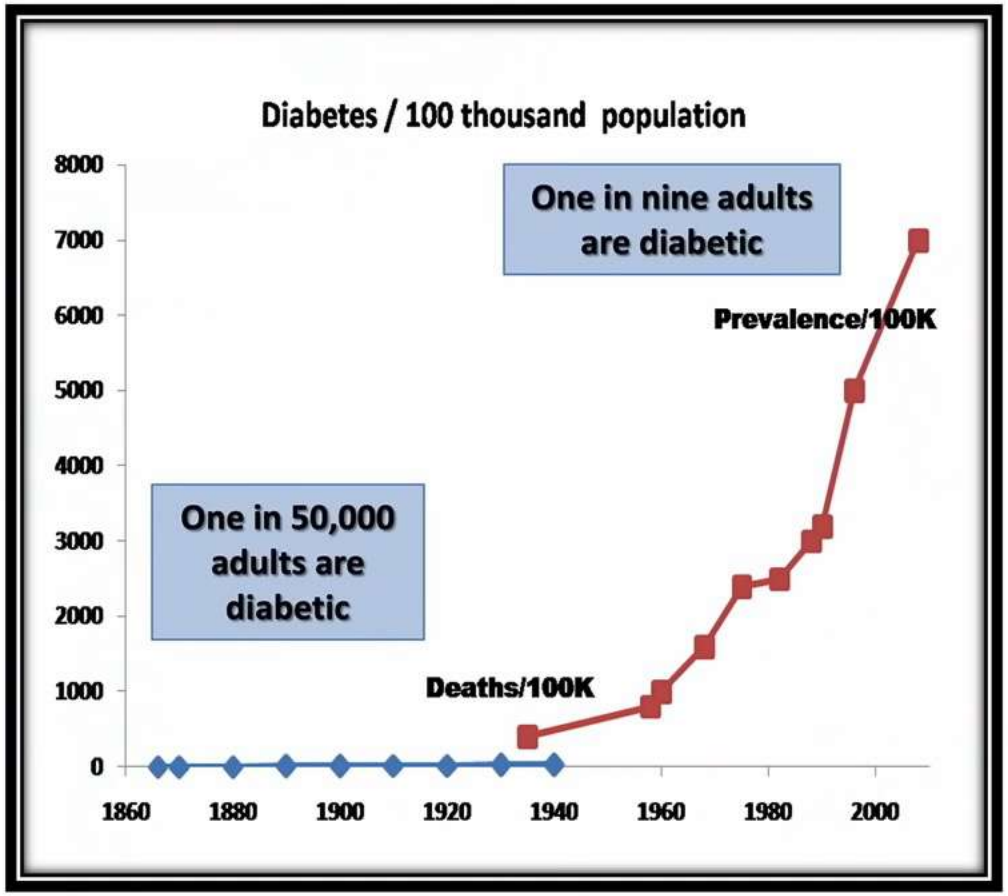


FIGURE 1 Change in fasting plasma glucose during the 13 years prior to onset of Type 2 diabetes. These data from the Whitehall II study demonstrate the elevation of plasma glucose within the normal range

Trajectories of glycaemia, insulin sensitivity, and insulin secretion before diagnosis of type 2 diabetes: an analysis from the Whitehall II study. Lancet 2009; 373: 2215–2221.


Environmental Challenge
Homeostasis is a balancing act that can be thrown out of whack by environmental challenges
If the system cannot restore balance, it can lead to death! (Credit: tollecausam.com)

Obesity and Diabetes: The Twin Epidemics



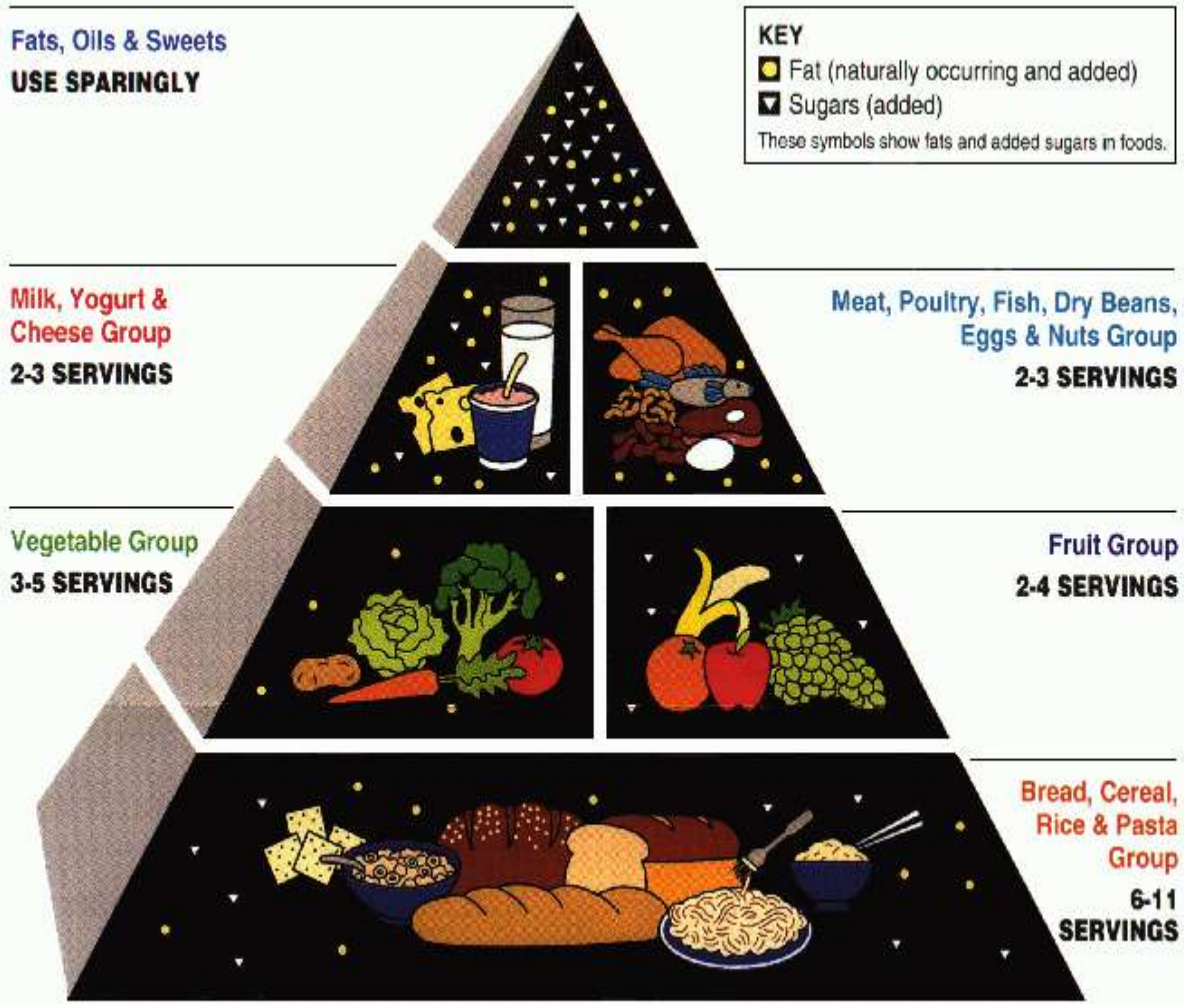
Many proposed causes: Western diet and lack of exercise most favored

Johnson et al Am J Clin Nutr 2007

Dietary Goals For the United States 1977

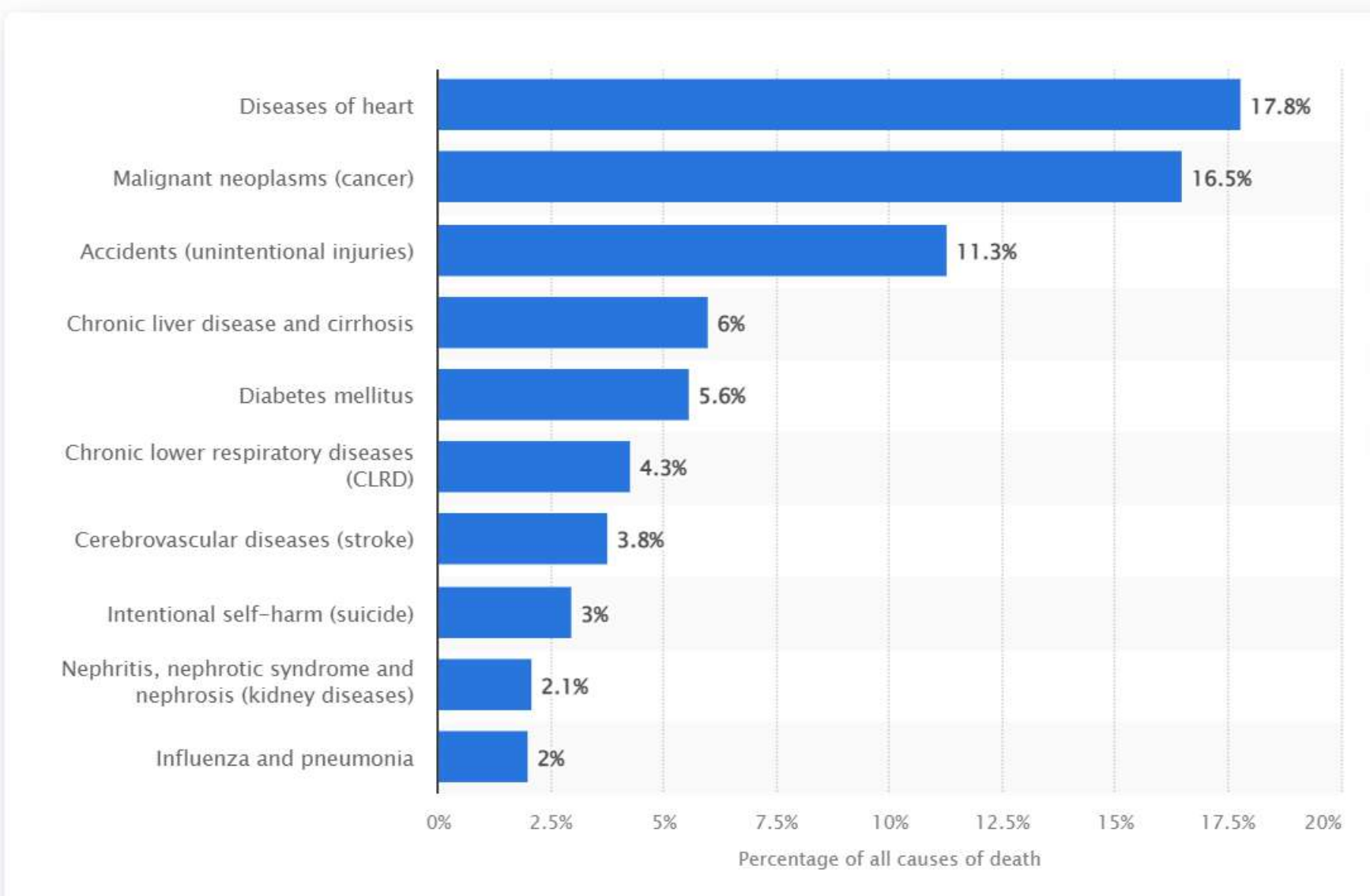
Dietary Goals

1. Raise consumption of carbohydrates until they constituted 55-60% of calories
2. Decrease fat consumption from approximately 40% to 30% of which no more than 1/3 from saturated fat



www.kidneylifescience.ca

Year 2019


Diseases of heart
17.8%
Malignant neoplasms (cancer)
16.5%
Accidents (unintentional injuries)
11.3%
Chronic liver disease and cirrhosis
6%
Diabetes mellitus
5.6%
Chronic lower respiratory diseases (CLRD)
4.3%
Cerebrovascular diseases (stroke)
3.8%
Intentional self-harm (suicide)
3%
Nephritis, nephrotic syndrome and nephrosis (kidney diseases)
2.1%
Influenza and pneumonia
2%
0%
2.5%
5%
7.5%
10%
12.5%
15%
17.5%
20%
Percentage of all causes of death

openheart

Evidence from randomised controlled trials did not support the introduction of dietary fat guidelines in 1977 and 1983: a systematic review and meta-analysis

Zoë Harcombe,[1] Julien S Baker,[1] Stephen Mark Cooper,[2] Bruce Davies,[3] Nicholas Sculthorpe,[1] James J DiNicolantonio,[4] Fergal Grace[1]

To cite: Harcombe Z, Baker JS, Cooper SM, *et al*. Evidence from randomised controlled trials did not support the introduction of dietary fat guidelines in 1977 and 1983: a systematic review and meta-analysis. *Open Heart* 2015;**2**:e000196. doi:10.1136/openhrt-2014-000196

ABSTRACT

Objectives: National dietary guidelines were introduced in 1977 and 1983, by the US and UK governments, respectively, with the ambition of reducing coronary heart disease (CHD) by reducing fat intake. To date, no analysis of the evidence base for these recommendations has been undertaken. The present study examines the evidence from randomised controlled trials (RCTs) available to the US and UK regulatory committees at their respective points of implementation.

KEY MESSAGES

What is already known about this subject?

- Dietary recommendations were introduced in the US (1977) and in the UK (1983) to (1) reduce overall fat consumption to 30% of total energy intake and (2) reduce saturated fat consumption to 10% of total energy intake.

What does this study add?

- No randomised controlled trial (RCT) had tested

To cite: Harcombe Z, Baker JS, Cooper SM, *et al*. Evidence from randomised controlled trials did not support the introduction of dietary fat guidelines in 1977 and 1983: a systematic review and meta-analysis. *Open Heart* 2015;**2**:e000196. doi:10.1136/openhrt-2014-000196

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[4]Saint Luke's Mid America Heart Institute, Kansas City, Missouri, USA

ABSTRACT

Objectives: National dietary guidelines were introduced in 1977 and 1983, by the US and UK governments, respectively, with the ambition of reducing coronary heart disease (CHD) by reducing fat intake. To date, no analysis of the evidence base for these recommendations has been undertaken. The present study examines the evidence from randomised controlled trials (RCTs) available to the US and UK regulatory committees at their respective points of implementation.

Methods: A systematic review and meta-analysis were undertaken of RCTs, published prior to 1983, which examined the relationship between dietary fat, serum cholesterol and the development of CHD.

Results: 2467 males participated in six dietary trials: five secondary prevention studies and one including healthy participants. There were 370 deaths from all-cause mortality in the intervention and control groups. The risk ratio (RR) from meta-analysis was 0.996 (95% CI 0.865 to 1.147). There were 207 and 216 deaths from CHD in the intervention and control groups, respectively. The RR was 0.989 (95% CI 0.784 to 1.247). There were no differences in all-cause mortality and non-significant differences in CHD mortality, resulting from the dietary interventions. The reductions in mean serum cholesterol levels were significantly higher in the intervention groups; this did not result in significant differences in CHD or all-cause mortality. Government dietary fat recommendations were untested in any trial prior to being introduced.

Conclusions: Dietary recommendations were introduced for 220 million US and 56 million UK citizens by 1983, in the absence of supporting evidence from RCTs.

KEY MESSAGES

What is already known about this subject?

- Dietary recommendations were introduced in the US (1977) and in the UK (1983) to (1) reduce overall fat consumption to 30% of total energy intake and (2) reduce saturated fat consumption to 10% of total energy intake.

What does this study add?

- No randomised controlled trial (RCT) had tested government dietary fat recommendations before their introduction. Recommendations were made for 276 million people following secondary studies of 2467 males, which reported identical all-cause mortality. RCT evidence did not support the introduction of dietary fat guidelines.

How might this impact on clinical practice?

- Clinicians may be more questioning of dietary guidelines, less accepting of low-fat advice (concomitantly high carbohydrate) and more engaged in nutritional discussions about the role of food in health.

INTRODUCTION

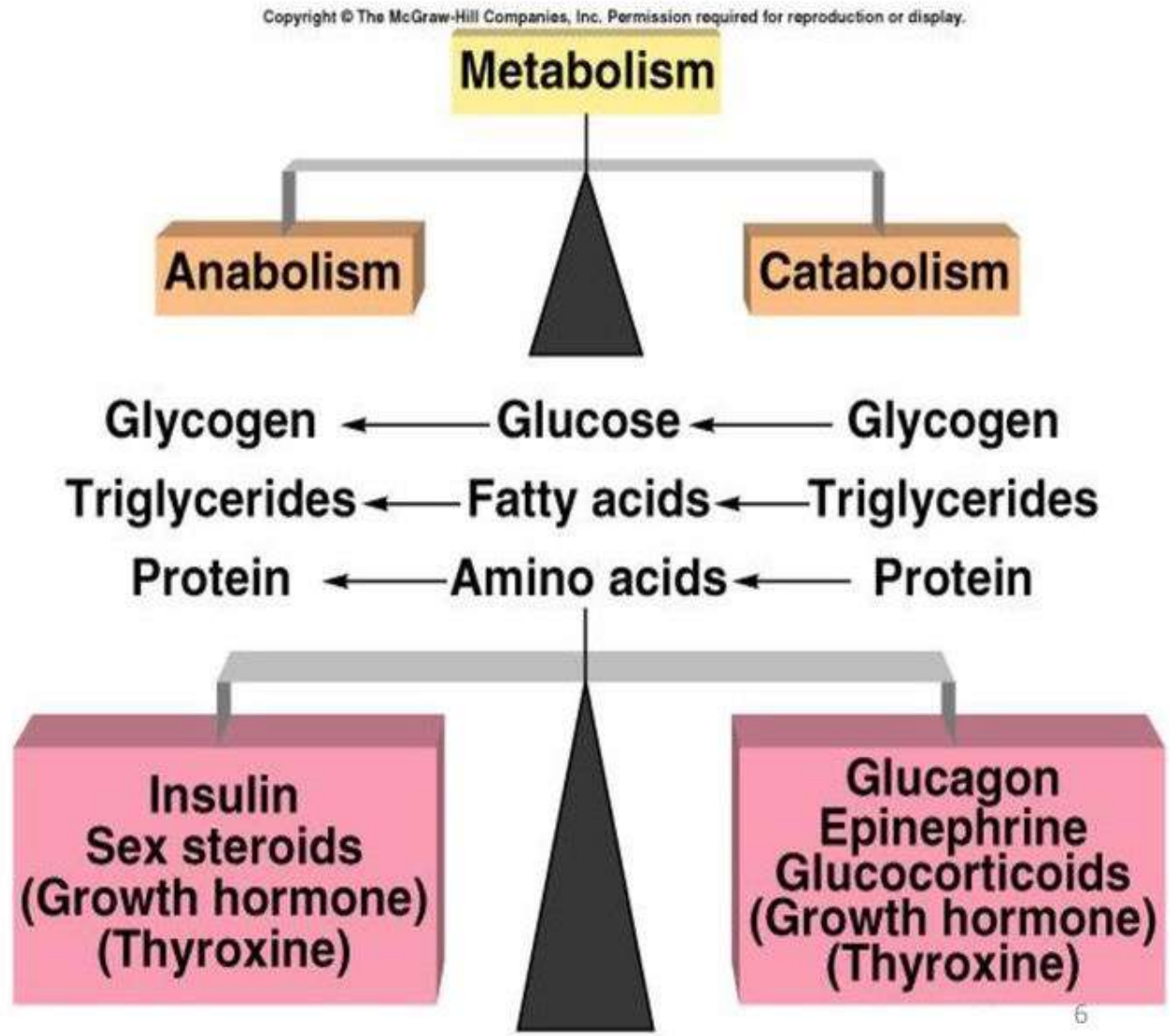
US public health dietary advice was advice issued by the National Advisory Committee on Nutritional Education in 1983.[2] The dietary recommendations in both cases focused on reducing dietary fat intake; specifically to (1) reduce overall fat consumption to 30% of total energy intake and (2) reduce saturated fat consumption to 10% of total energy intake.

The recommendations were an attempt to address the incidence of coronary heart disease (CHD). Both documents acknowledged that the evidence was not conclusive. Hegsted's introduction to the Dietary Goals for the US noted "there will undoubtedly be


How can we
make the change?

Balance Between Anabolism and Catabolism

- The rate of deposit and withdrawal of energy substrates, and the conversion of 1 type of energy substrate into another; are regulated by hormones.



Target Protein

One gram daily per pound of DESIRED body weight, from properly raised animals.

Limit Carbs

Unlimited fiber from green vegetables, but limit net carbs to less than about fifty grams daily.

Balance Fat

If you have too much fat in your body, skip high fat foods and eat only fats from eggs and lean meat.

SPECIFIC ADAPTATION TO IMPOSED DEMAND

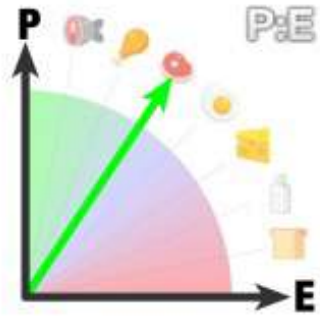
Adaptability. Humans are amazing survival machines, which is why we live in all climates, eating all diets. Your body will always adapt to meet the demands you impose upon it.

If you want your body to be better at burning fat, you only have to do one thing:

Eat fewer carbohydrates.

If you want your body to be better at burning its own stored body fat, you only have to do two things:

Eat fewer carbohydrates, and then eat less fat.



1. MAXIMIZE SATIETY.

TARGET PROTEIN AND MINERALS FOR HIGHEST NUTRIENT DENSITY.

2. MAXIMIZE FAT ADAPTATION.

USE INTERMITTENT FASTING WITH LOW CARBOHYDRATE FREQUENCY.

3. AVOID THE TRIFECTA.

HIGH CARB + HIGH FAT + HIGH ENERGY DENSITY = OVEREATING.

The three pillars of The P:E Diet.

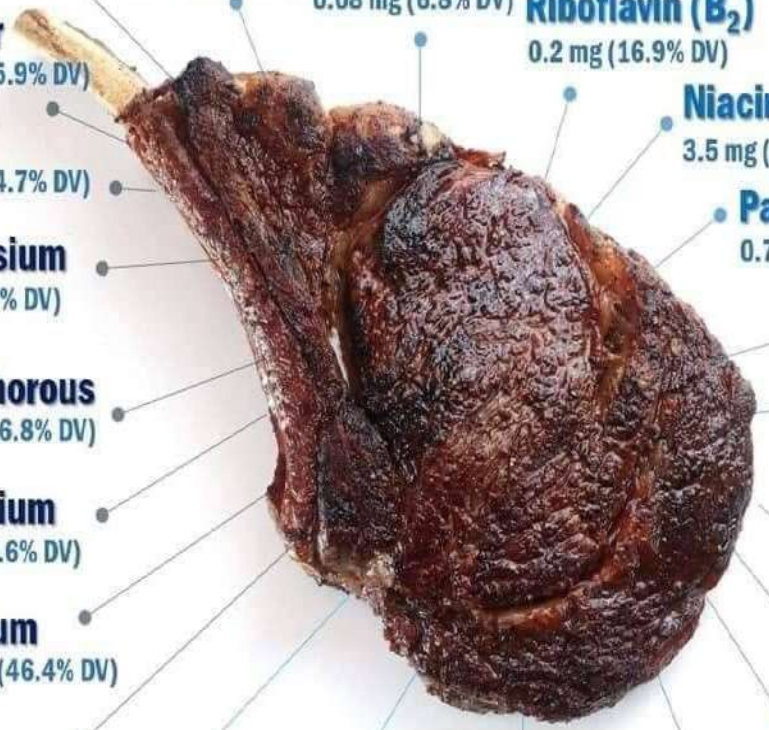



Nutrition Facts

THE RIBEYE

per 100 gram serving
approx. 3.5 ounces

nutritionwithjudy



ESSENTIAL MINERALS

- **Calcium** 6 mg (0.5% DV)
- **Copper** 0.1 mg (15.9% DV)
- **Iron** 2.6 mg (14.7% DV)
- **Magnesium** 24 mg (5.7% DV)
- **Phosphorous** 210 mg (16.8% DV)
- **Potassium** 357 mg (7.6% DV)
- **Selenium** 25.5 mcg (46.4% DV)
- **Zinc** 7.8 mg (70.9% DV)

ESSENTIAL VITAMINS

- **Vitamin A** 2 mcg (0.2% DV)
- **Thiamin (B_1)** 0.08 mg (6.8% DV)
- **Riboflavin (B_2)** 0.2 mg (16.9% DV)
- **Niacin (B_3)** 3.5 mg (21.9% DV)
- **Pantothenic Acid (B_5)** 0.7 mg (13.2% DV)
- **Vitamin B_6** 0.4 mg (22.9% DV)
- **Folate (B_9)** 3 mcg (0.8% DV)
- **Vitamin B_{12}** 3.2 mcg (134% DV)
- **Vitamin D** 0.1 mcg (0.5% DV)
- **Vitamin E** 0.2 mg (1.3% DV)
- **Choline** 74.9 mg (13.6% DV)
- **Omega 3** 10 mg
- **Omega 6** 240 mg
- **Vitamin K** 1.5 mg (1.3% DV)

RDAs are based off the Recommended Daily Allowance. Per the USDA, ribeye is missing Biotin (B_7) (dairy, liver, salmon, yolk), Chromium (eggs, fish, liver), Manganese (bone broth, egg), and Molybdenum (eggs, liver).

Source: USDA database

www.nutritionwithjudy.com

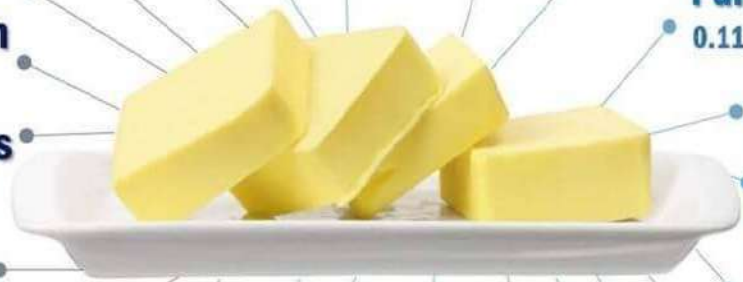
nwj

Nutrition Facts

BUTTER

per 100 gram serving
approx. 3.5 ounces

nutritionwithjudy



ESSENTIAL MINERALS

- **Calcium** 24 mg (1.9% DV)
- **Iron** 0.02 mg (0.1% DV)
- **Manganese** 0.6 mg (28% DV)
- **Molybdenum** trace
- **Phosphorous** 24 mg (1.9% DV)
- **Potassium** 24 mg (0.5% DV)
- **Selenium** 0.05 mcg (0.09% DV)
- **Zinc** 0.005 mg (0.05% DV)

ESSENTIAL VITAMINS

- **Vitamin A** 684 mcg (76% DV)
- **Thiamin (B_1)** 0.005 mg (0.4% DV)
- **Riboflavin (B_2)** 0.03 mg (2.6% DV)
- **Niacin (B_3)** 0.04 mg (0.3% DV)
- **Pantothenic Acid (B_5)** 0.11 mg (2.2% DV)
- **Vitamin B_6** 0.003mg (0.2% DV)
- **Biotin (B_7)** 94.3mcg (314.3% DV)
- **Folate (B_9)** 3 mcg (0.8% DV)
- **Vitamin B_{12}** 0.2 mg (8.3% DV)
- **Vitamin D** 14 mg (70% DV)
- **Choline** 18.8 mg (3.4% DV)
- **Omega 3** 315 mg
- **Omega 6** 2,728 mg
- **Vitamin K** 7 mg (5.8% DV)
- **Vitamin E** 2.3 mg (15.5% DV)

RDAs are based off the Recommended Daily Allowance. Per the USDA, butter is missing Vitamin C (salmon, oysters, and pork belly), Chromium (eggs, fish, liver), Copper (beef), and Manganese (bone broth, egg).

Source: USDA database

www.nutritionwithjudy.com

nwj


AFE´& BISTR
Polo RL


CMU
RUN
40M
STAFF


42.195 ไม่ต้องกินเจลเลยครับ
กินแต่น้ำแร่ เกลือแร่เม็ด
17:51


HbA1C 6% + ยา


HbA1c 5.6% ไม่มียา


65 ปี...Carnivore Diet

Steve Curtin

1 วัน ·

She's been carnivore for over 60 years.


38
46
48
50
52
53
56

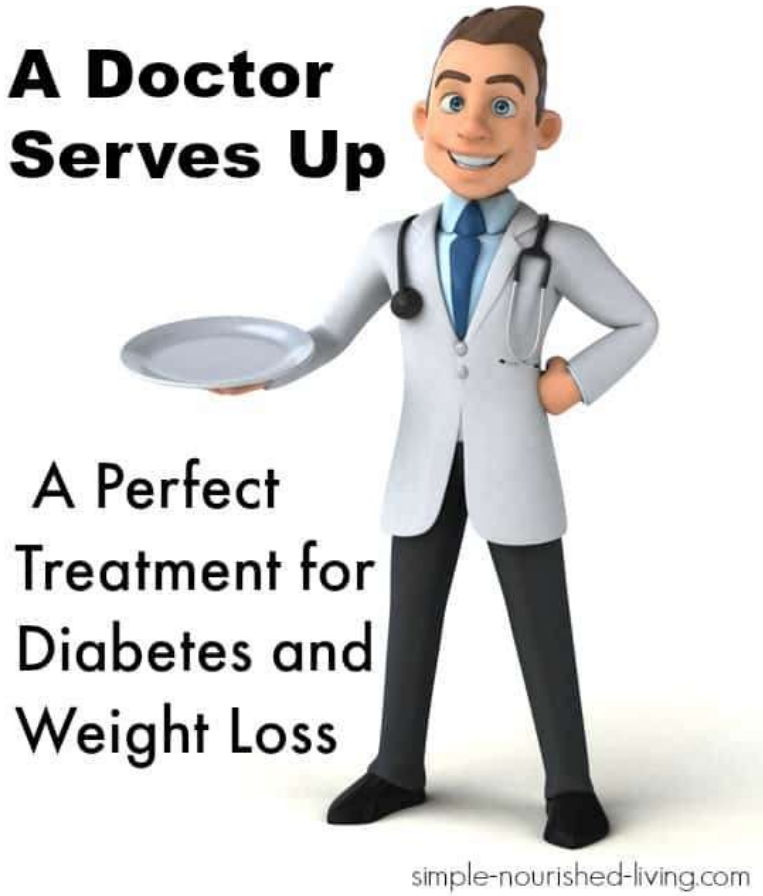

38ปี
55ปี

Them: wow you seem happier are you taking something?

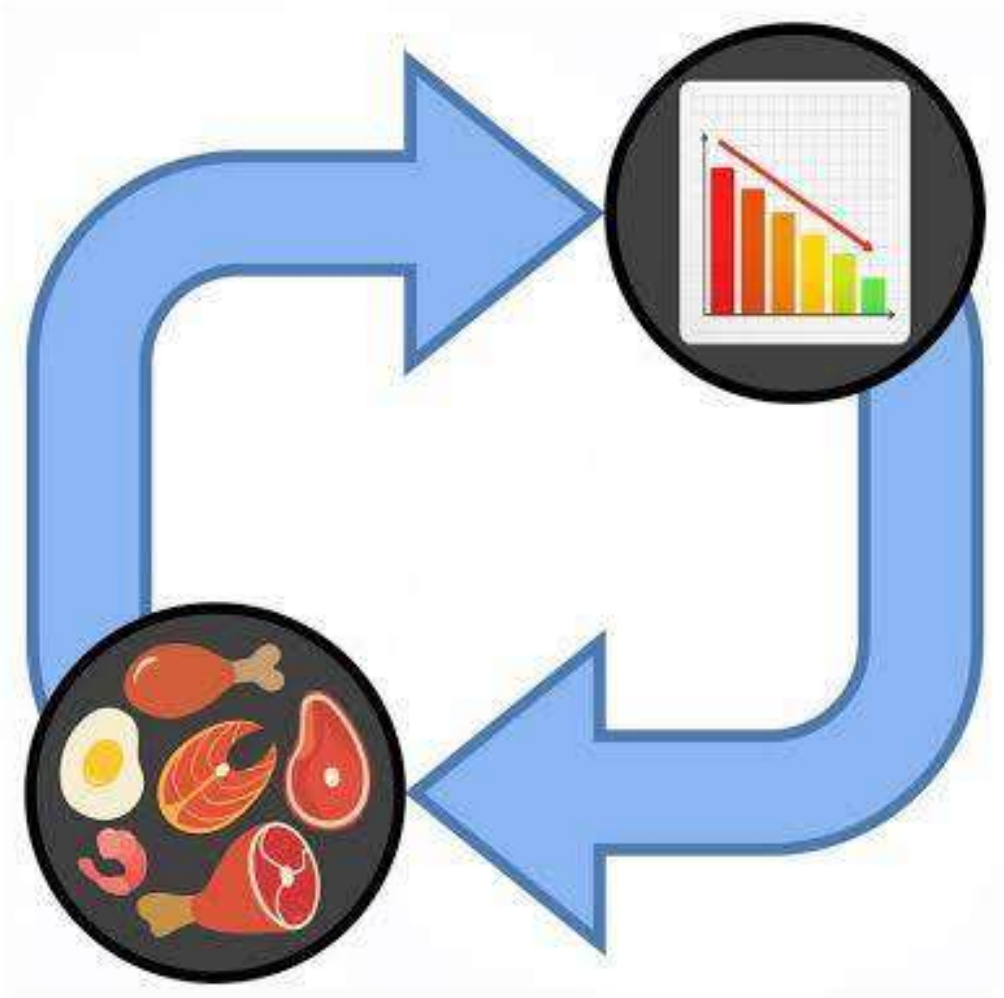
Me:

When to eat determines how persistent insulin is.

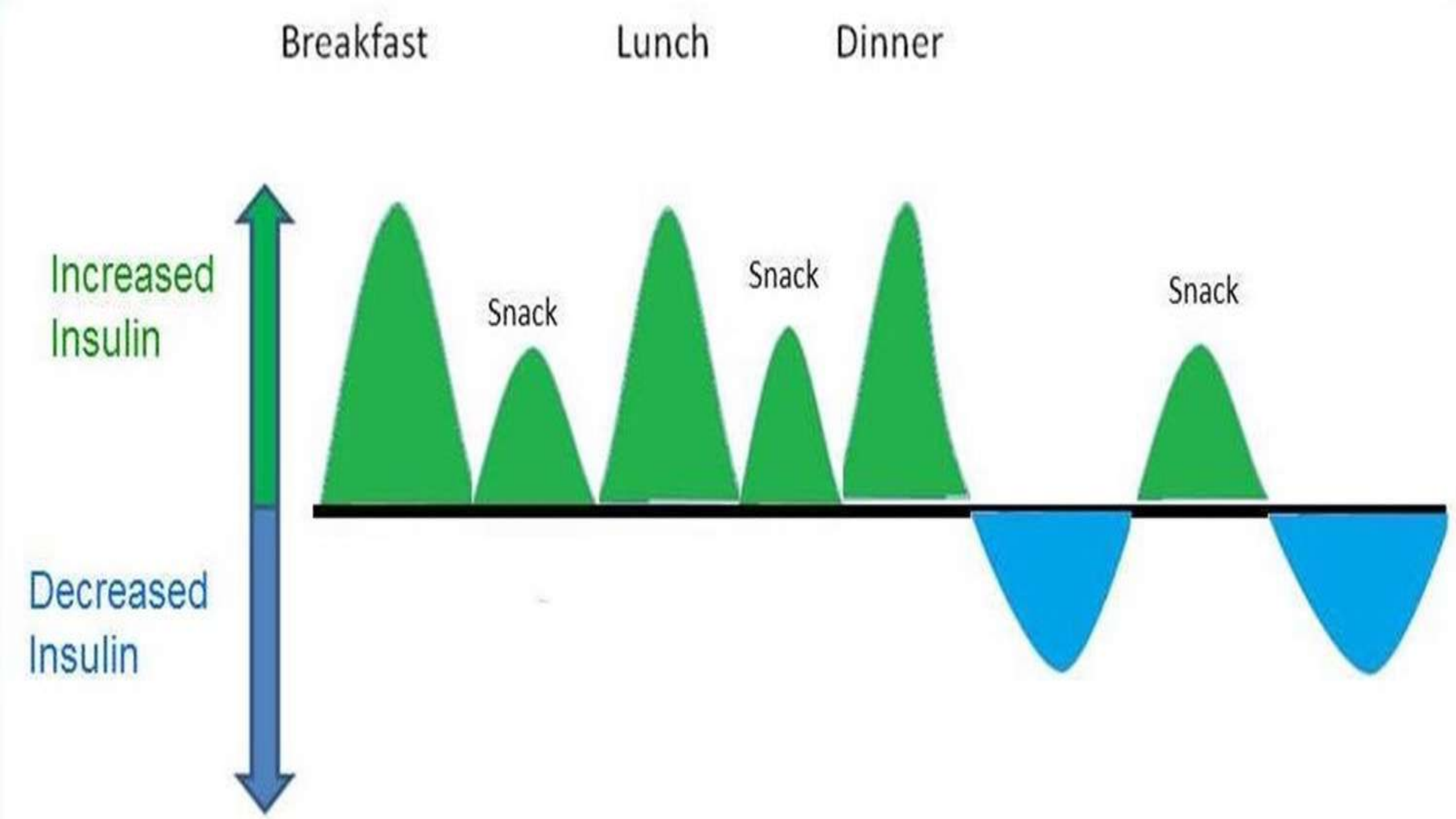
Intermittent fasting.

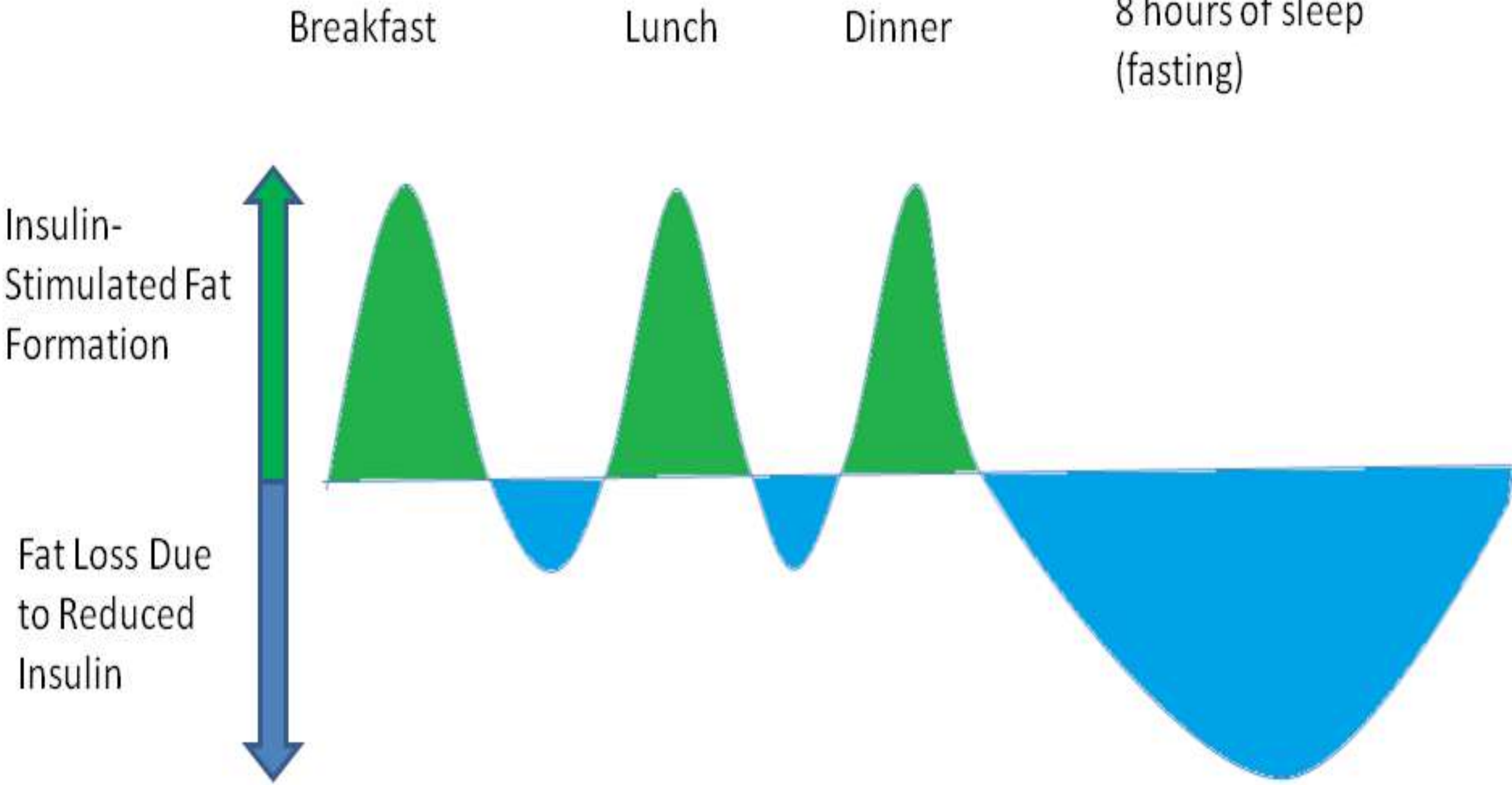


1. Don’t eat unless you are actually hungry.

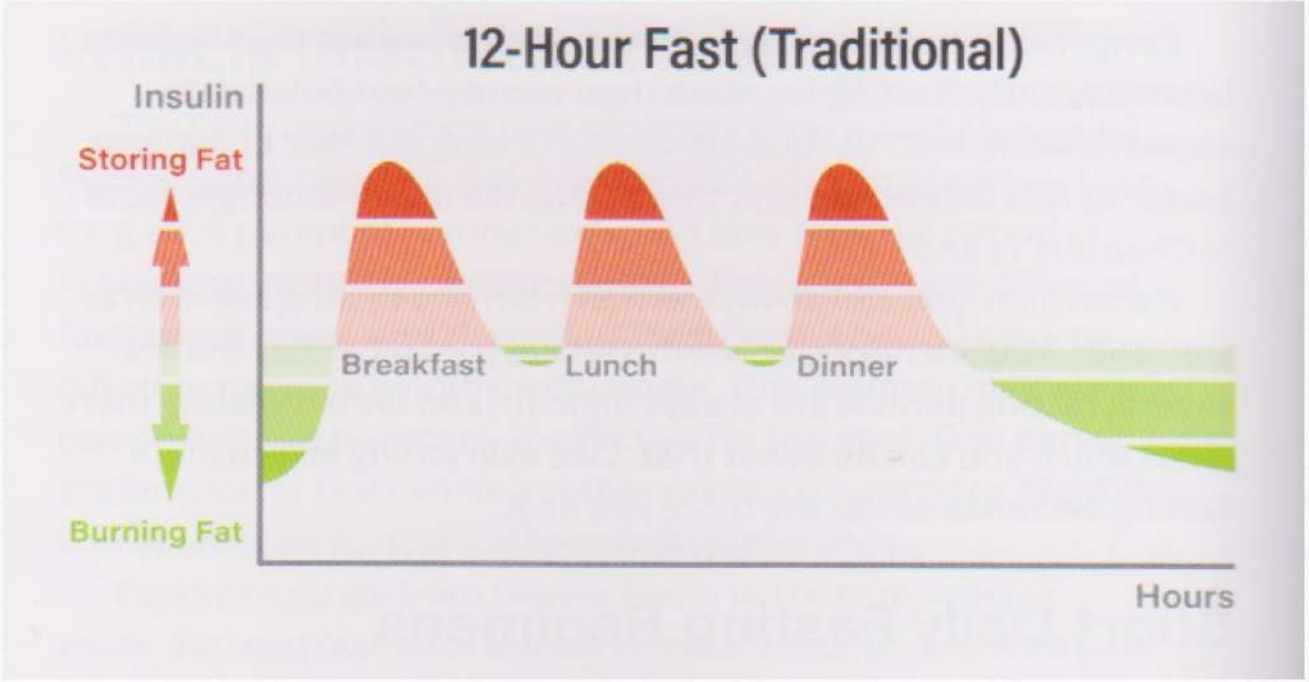


2. If you are actually hungry, eat protein.

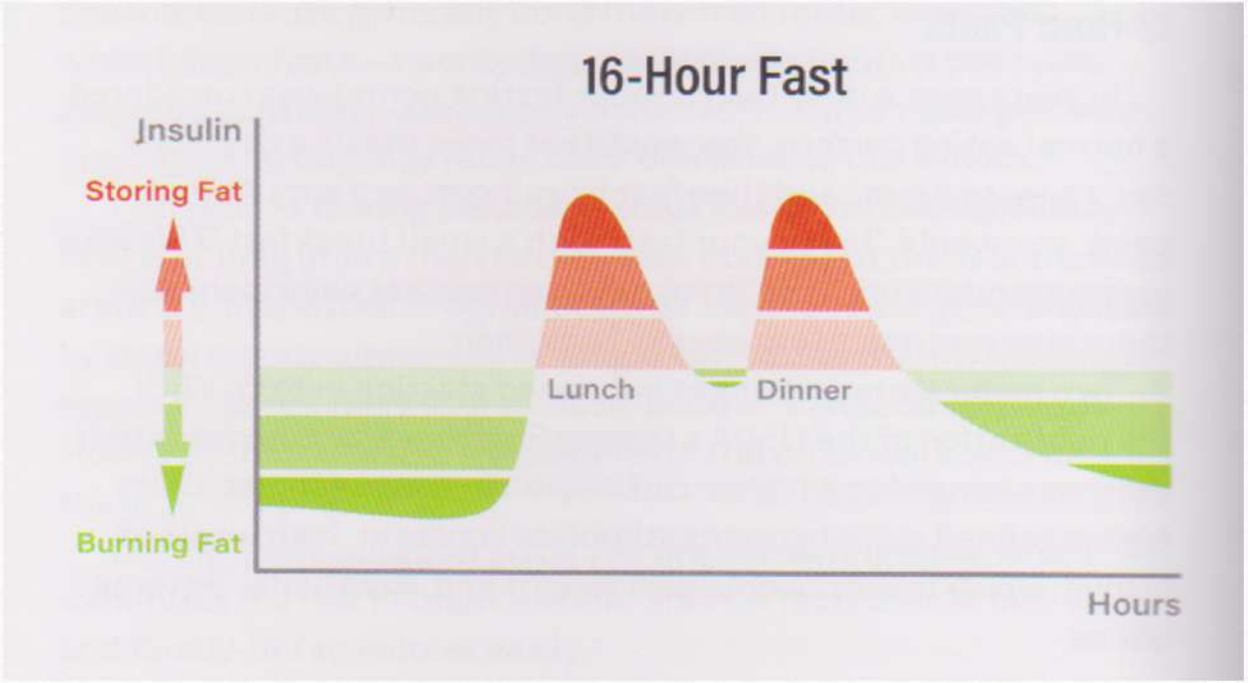

Breakfast
Lunch
Dinner
Increased Insulin
Decreased Insulin
Snack
Snack
Snack


Breakfast
Lunch
Dinner
8 hours of sleep (fasting)
Insulin-Stimulated Fat Formation
Fat Loss Due to Reduced Insulin

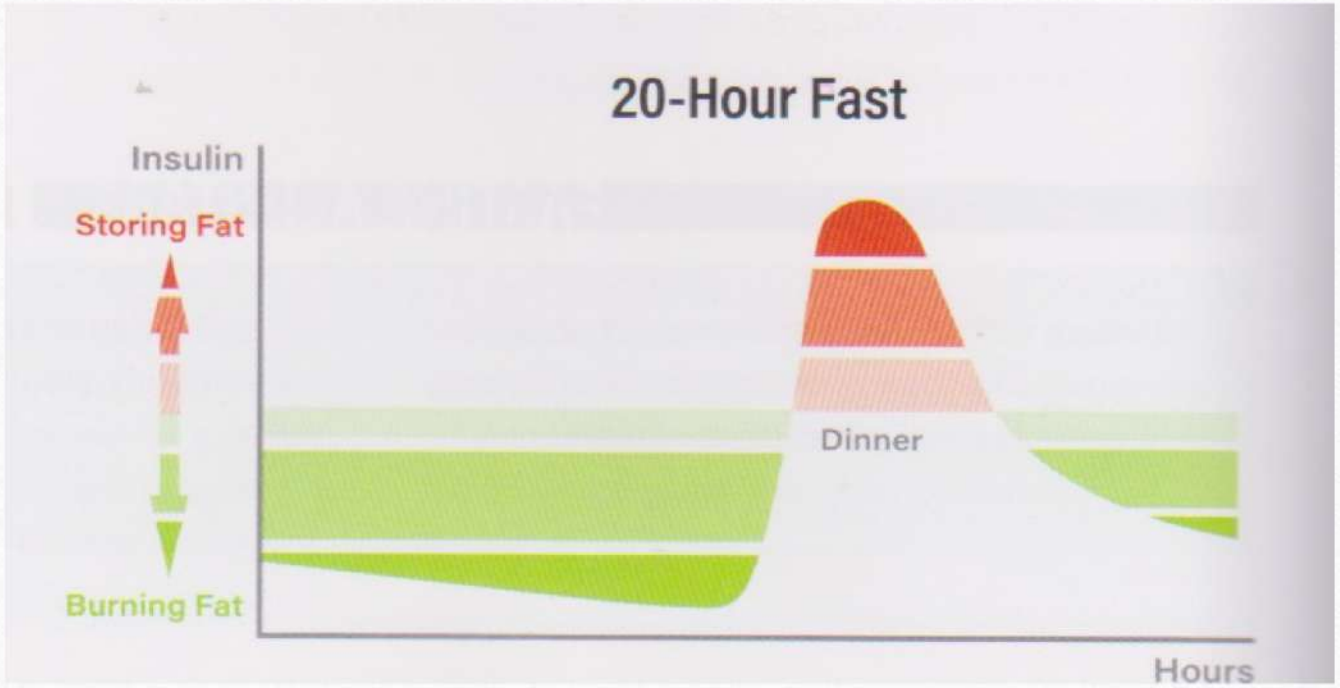
Short Daily Fasting Regimens

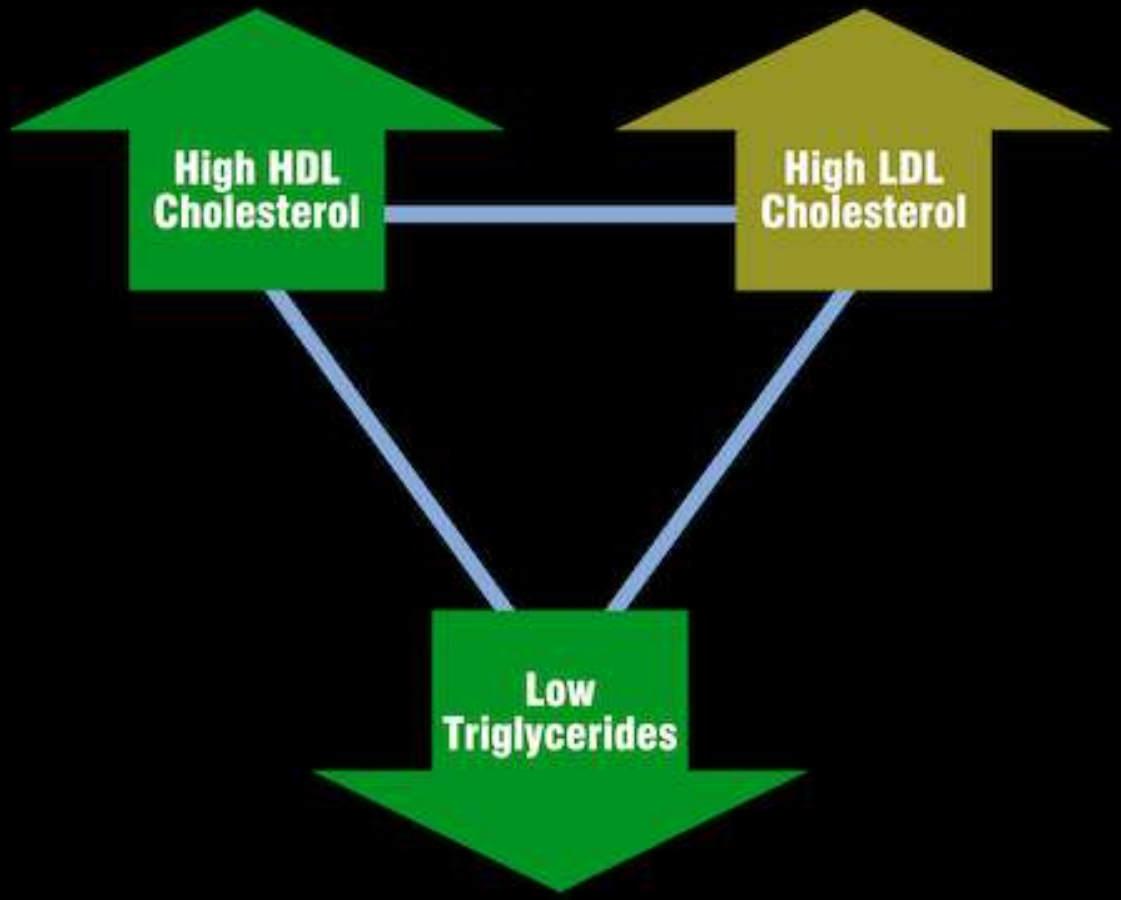


Short Daily Fasting Regimens

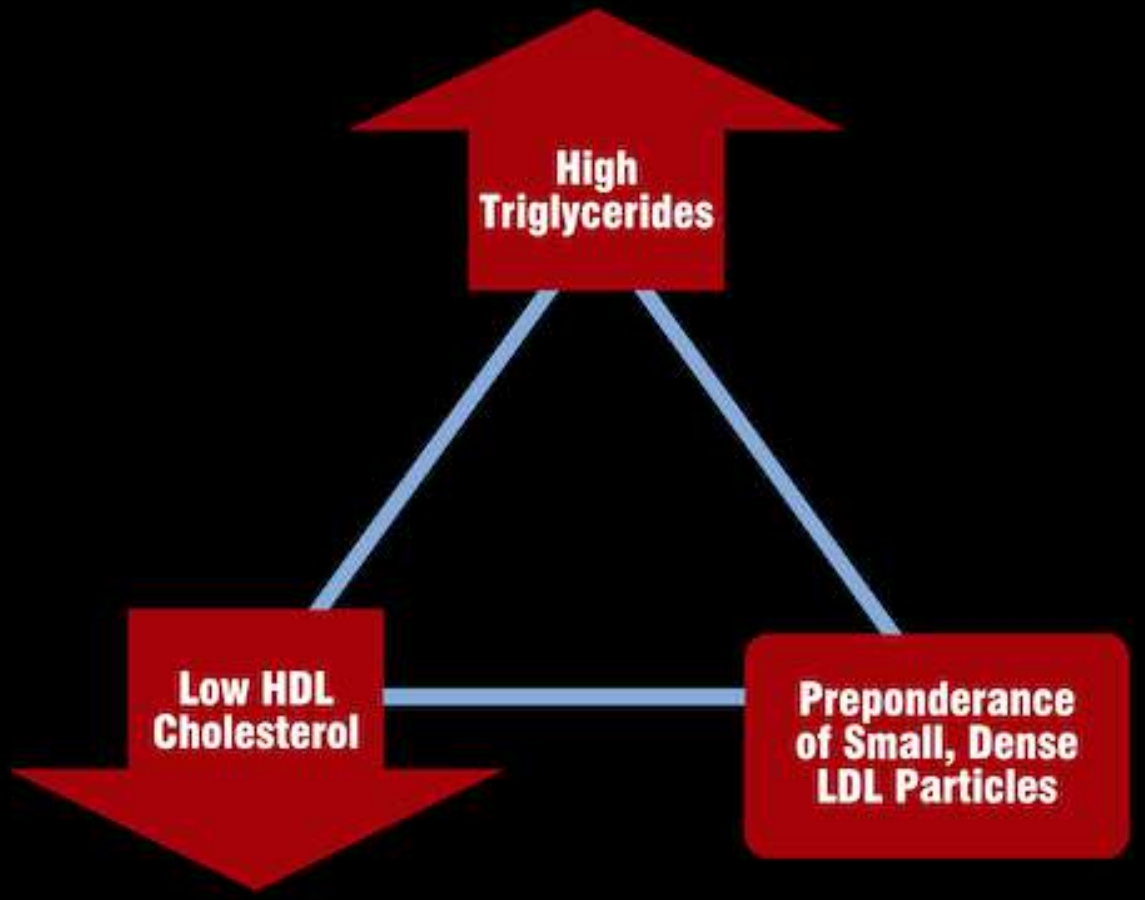


Short Daily Fasting Regimens

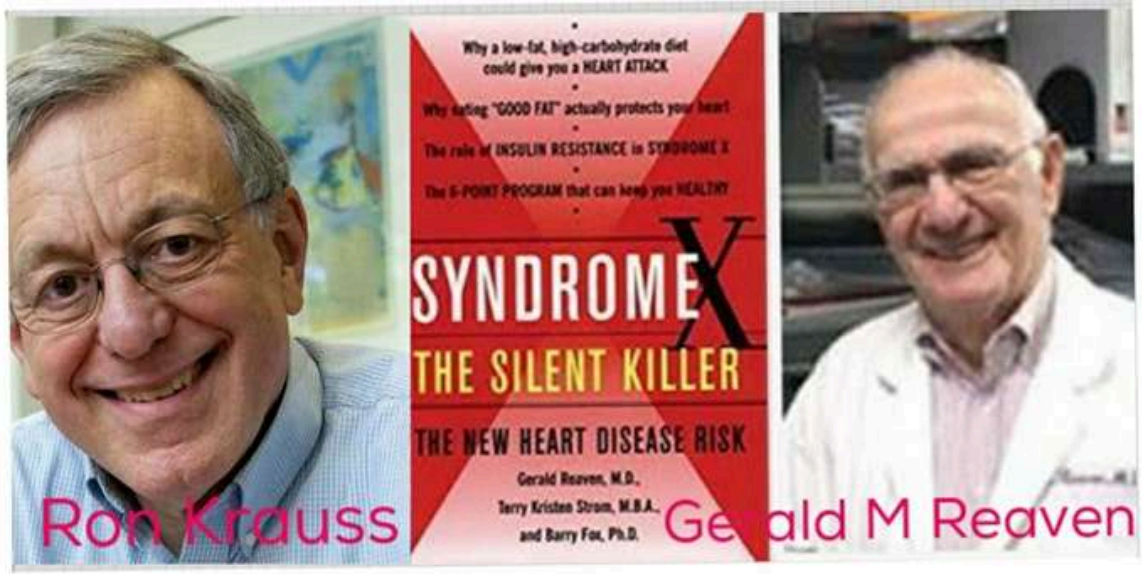




Low Carb Lipid Triad



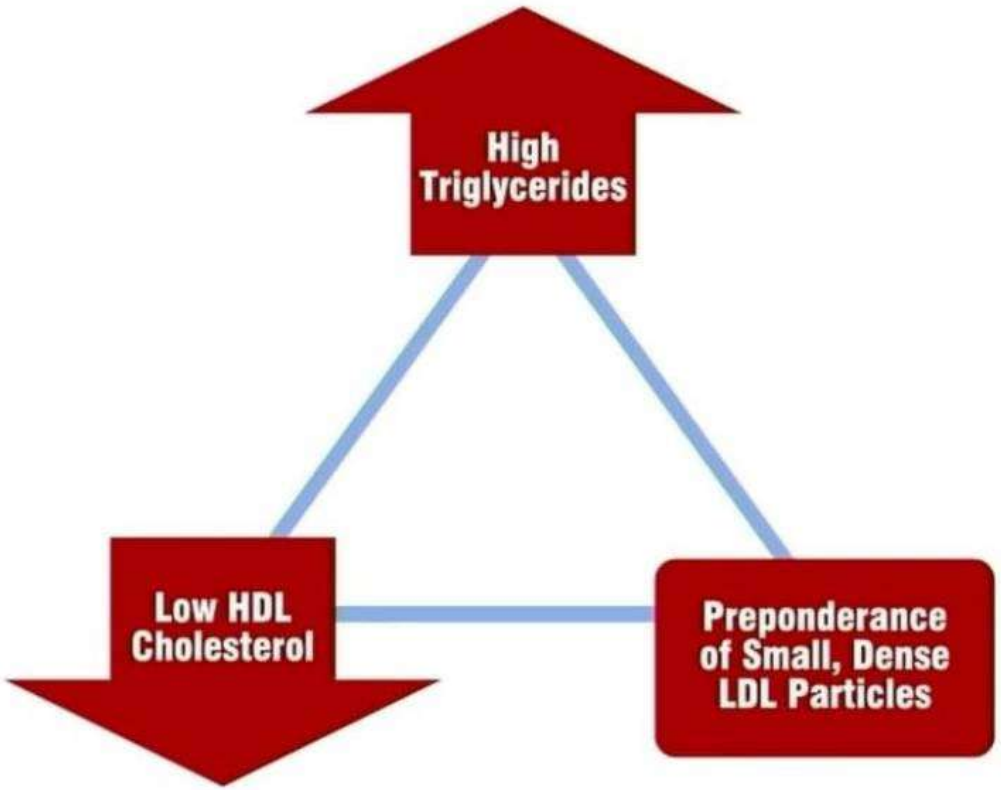
Atherogenic Dyslipiemia



articulations and *ad hoc* modifications of their theory in order to eliminate any apparent conflict." And that's exactly what happened with metabolic syndrome and its dietary implications. The syndrome itself was accepted as real and important; the idea that it was caused or exacerbated by the excessive consumption of carbohydrates simply vanished.

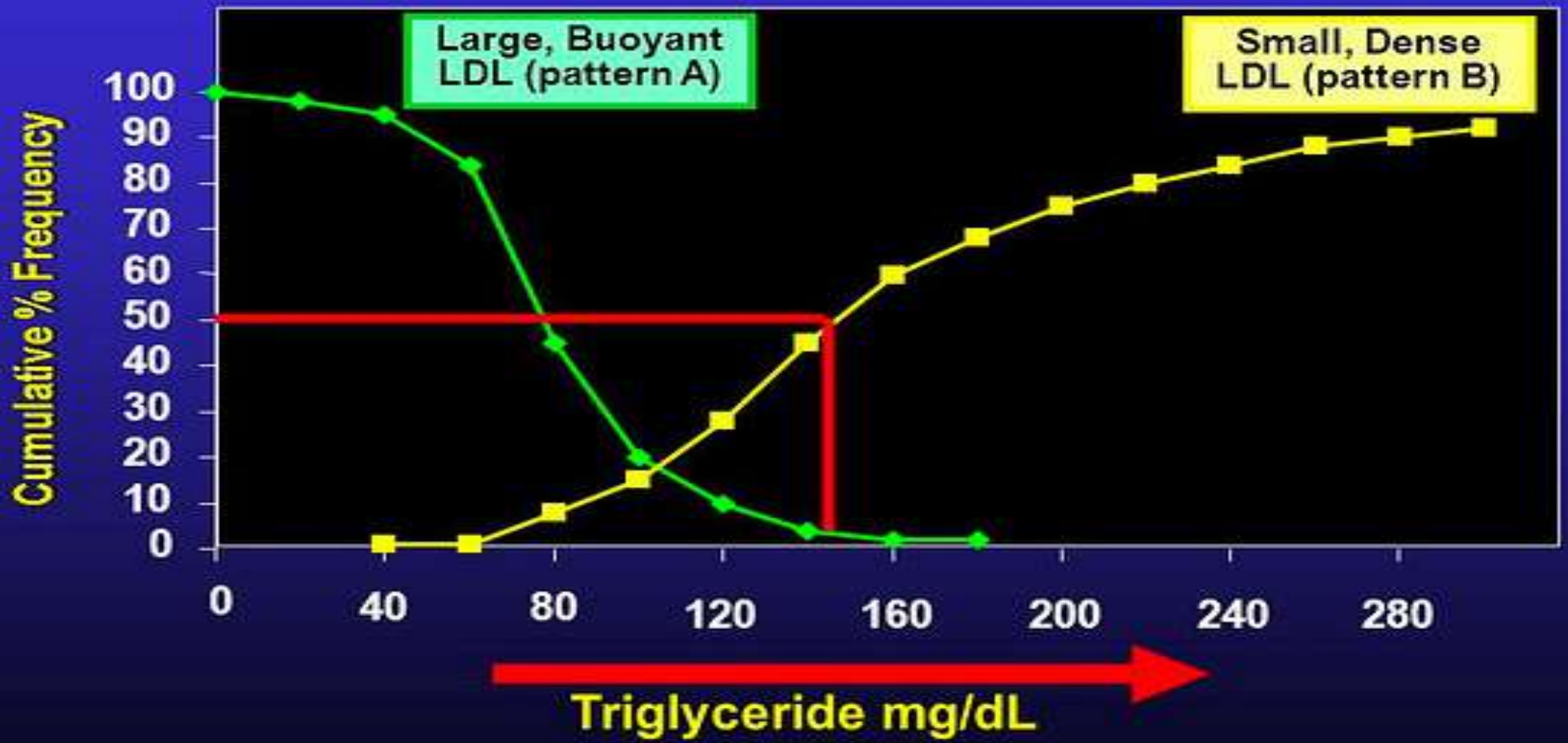
Among the few clinical investigators working on heart disease who paid attention to Reaven's research in the late 1980s was Ron Krauss. In 1993, Krauss and Reaven together reported that small, dense LDL was another of the metabolic abnormalities commonly found in Reaven's Syndrome X. Small, dense LDL, they noted, was associated with insulin resistance, hyperinsulinemia, high blood sugar, hypertension, and low HDL as well. They also reported that the two best predictors of the presence of insulin resistance and the dominance of small, dense LDL are triglycerides and HDL cholesterol—the higher the triglycerides and the lower the HDL, the more likely it is that both insulin resistance and small, dense LDL are present. This offers yet another reason to believe the carbohydrate hypothesis of heart disease, since metabolic syndrome is now considered perhaps the dominant heart-disease risk factor—a "coequal partner to cigarette smok-

Atherogenic Dyslipidemia: Cardiovascular Risk and Dietary Intervention (PMID: 20524075)

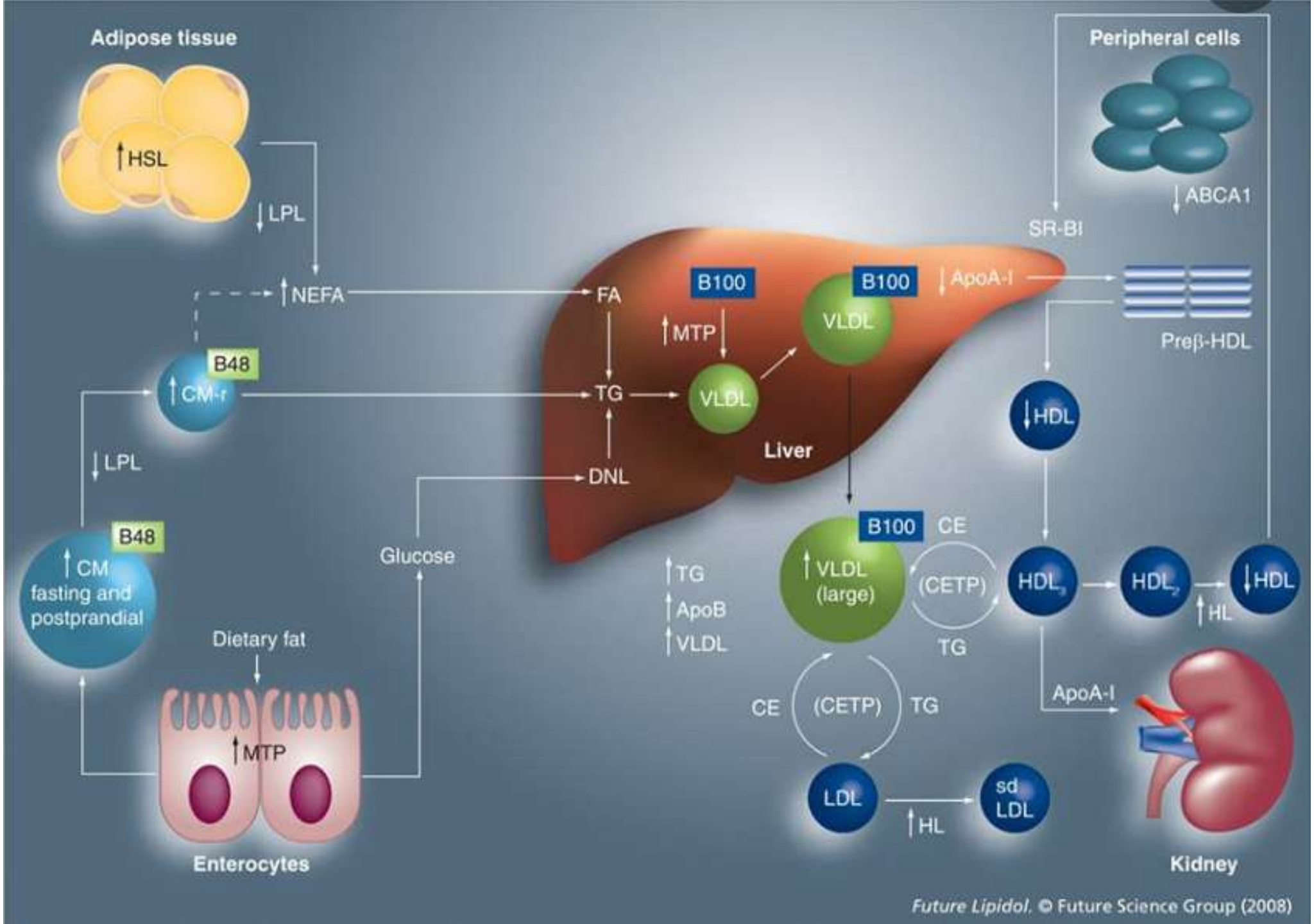


"A typical feature of obesity, the metabolic syndrome, insulin resistance, and type 2 diabetes mellitus, atherogenic dyslipidemia has emerged as an important risk factor for myocardial infarction and cardiovascular disease."

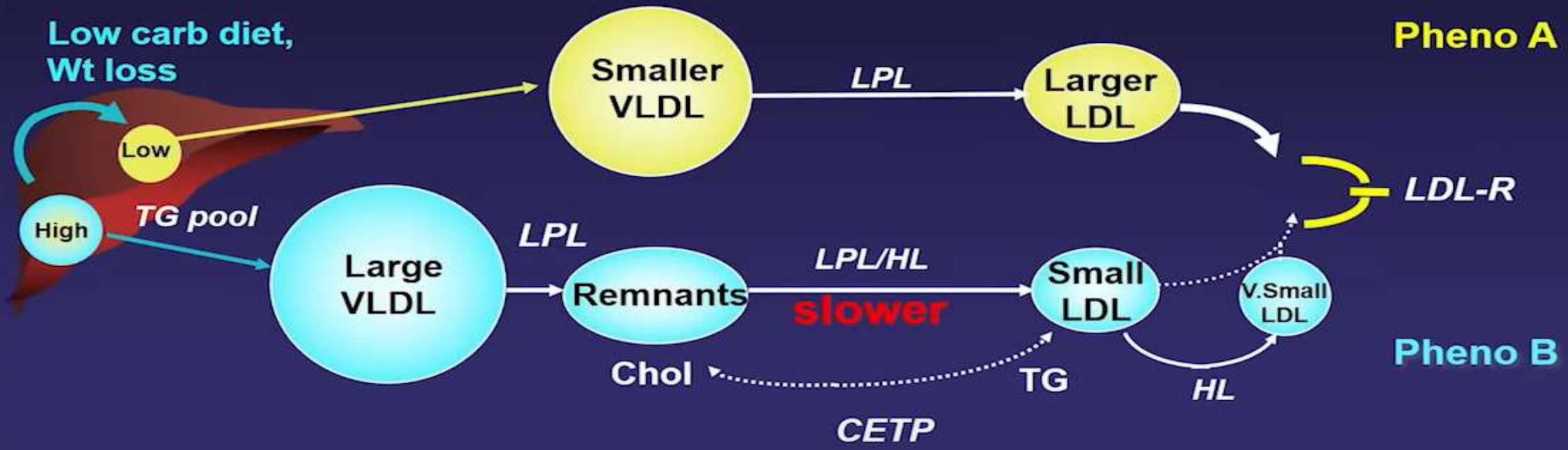
Relationship of Triglycerides and LDL Particle Size

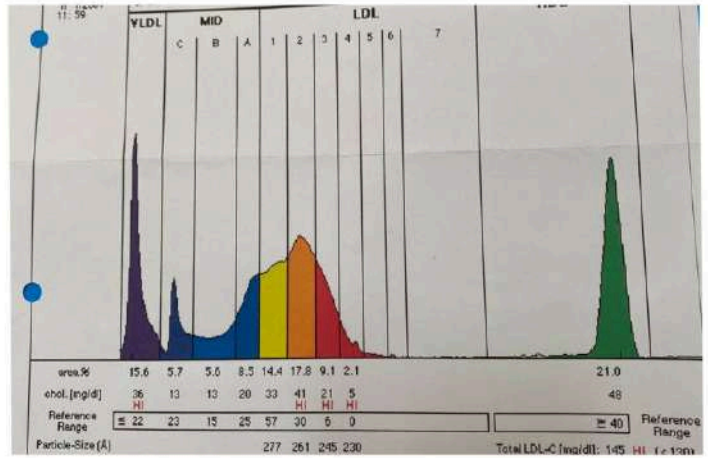


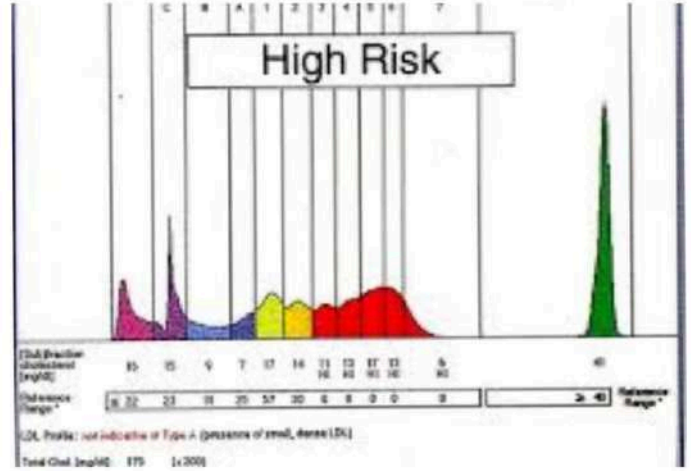
Austin M, et al. *Circulation.* 1990;82:495-506.

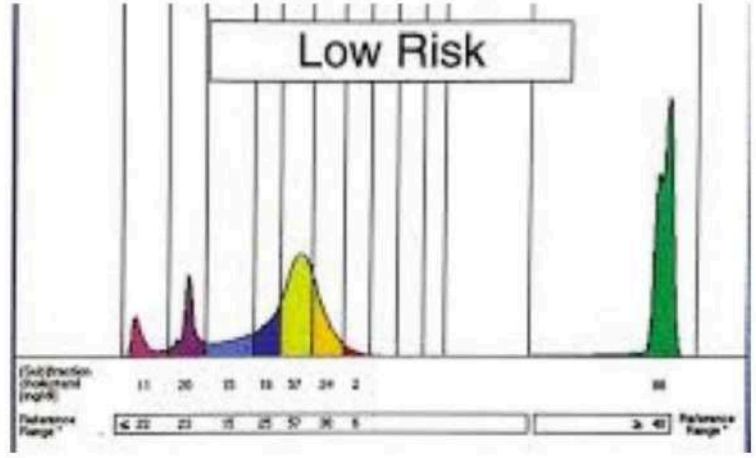


Phenotype B can be reversed by either reduced carbohydrate intake or weight loss

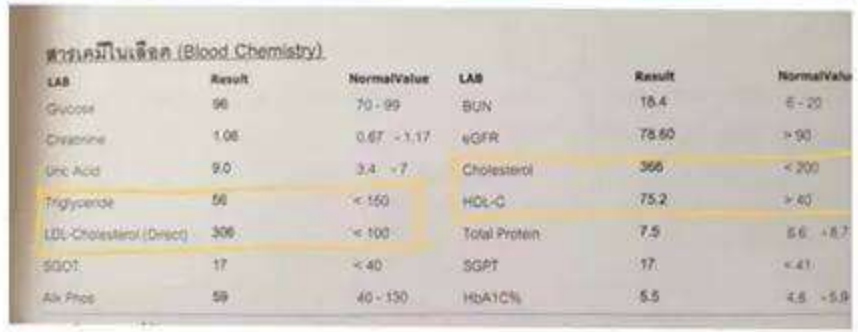



VLDL
MID
LDL
C
B
A
1
2
3
4
5
6
7
area,%
15.6
5.7
5.0
8.5
14.4
17.8
9.1
2.1
21.0
chol. [mg/dl]
26
HI
13
13
20
33
41
HI
21
HI
5
HI
48
Reference Range
≤ 22
23
15
25
57
30
6
0
≥ 40
Reference Range
Particle-Size (Å)
277
261
245
230
Total LDL-C [mg/dl]: 145 HI


High Risk

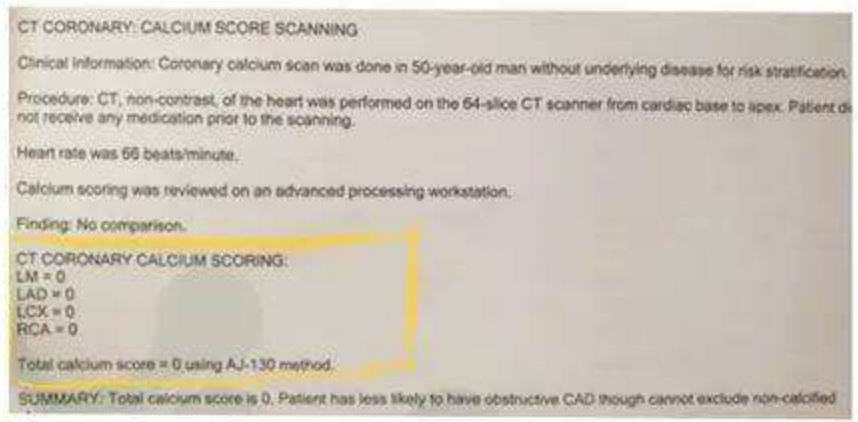

Low Risk

ชายอายุ 50ปี เคยน้ำหนัก 90+กก. ทานคีโต 10เดือน นน.ปัจจุบัน 75กก. ผลคลอเลสเตอรอล และCAC ตามที่เห็น



ตารเคมีในเลือด (Blood Chemistry)

LAB	Result	NormalValue	LAB	Result	NormalValu
Glucose	96	70 - 99	BUN	18.4	6 - 20
Creatinine	1.06	0.67 - 1.17	eGFR	78.60	> 90
Uric Acid	9.0	3.4 - 7	Cholesterol	366	< 200
Triglyceride	56	< 150	HDL-C	75.2	> 40
LDL-Cholesterol (Direct)	306	< 100	Total Protein	7.5	6.6 - 8.7
SGOT	17	< 40	SGPT	17	< 41
Alk Phos	59	40 - 130	HbA1C%	5.5	4.6 - 5.9



CT CORONARY: CALCIUM SCORE SCANNING

Clinical Information: Coronary calcium scan was done in 50-year-old man without underlying disease for risk stratification.

Procedure: CT, non-contrast, of the heart was performed on the 64-slice CT scanner from cardiac base to apex. Patient d not receive any medication prior to the scanning.

Heart rate was 66 beats/minute.

Calcium scoring was reviewed on an advanced processing workstation.

Finding: No comparison.

CT CORONARY CALCIUM SCORING:
LM = 0
LAD = 0
LCX = 0
RCA = 0

Total calcium score = 0 using AJ-130 method.

SUMMARY: Total calcium score is 0. Patient has less likely to have obstructive CAD though cannot exclude non-calcified

สวัสดีครับคุณหมอ ยินดีด้วยนะครับที่มีผู้ติดตามคุณหมอเพิ่มมากขึ้นเรื่อยๆ ครับ. พอดีผมเพิ่งไปตรวจร่างกายมาอีกครั้ง ห่างจากครั้งที่แล้วประมาณ 1 ปี เลยอยากฝากให้คุณหมอเป็นข้อมูลเผื่อว่าจะเป็นประโยชน์. ผลเป็นดังนี้ครับ

	Feb 2020	Mar 2019
T. Choles	402	366
LDL	315	306
HDL	78	75
Triglyceride	46	56
CAC	0	0

HDL กับ Triglyceride ดีขึ้น แต่ LDL ไปต่อ. ส่วน CAC เป็น 0 เท่าเดิมครับ. (จริงๆก่อนตรวจผมกังวลค่า CAC แต่พอรู้ว่าเป็น 0 เท่าเดิม ก็สบายใจครับ)

Age : 37.10.9 Sex : Female Dept : Social welfare AN/VN

Description		Result	Unit	
FBS # (SS FOC)		91	mg/dl	
Lipid profile #				
Cholesterol (serum)#(ไขมันโคเลสเตอรอล)	High	747	mg/dl	
Triglyceride (serum)#(ไขมันไตรกลีเซอไรด์)		66	mg/dl	
HDL (serum)# (ไขมัน เอชดีแอล)		97	mg/dl	Male > 40 r
LDL (serum)# (ไขมันแอลดีแอล)	High	637.0	mg/dl	Female > 4 Direct meth Triglyceride mg/dl
Liver Function Test # (หน้าที่ของตับ)				
Total protein (serum)#		7.0	gm/dl	
Albumin(Serum) #		4.2	gm/dl	
Globulin (serum)#		2.8	gm/dl	
SGOT (AST) (serum) # (เอ็นไซม์ตับ)		16	IU/L	
SGPT (ALT)(serum) # (เอ็นไซม์ตับ)		14	IU/L	

43.10.9 Sex : Female Ethnic Group: Thai

Description	Result
Lipid profile # (SS FOC)	
Cholesterol (clot blood)#	484 Repeated
Triglyceride (clot blood)#	54
HDL (clot blood)#	69 12/2565
LDL (clot blood)#	H. 405.0
FBS # (SS FOC)(NaF blood)	86

LABORATORY R
LANNA HOSPITAL 1 SUKKASEM Rd. T. PATON A. MUA
TEL. 052-134-777 EXT 1154

Patient Name น.ส. สิริประกาย ดวงแก้ว Id
Age : 44.4.23 Sex : Female Ethnic Group: Thai

Description	Result
Lipid profile #	
Cholesterol (clot blood)#	H. 513
Triglyceride (clot blood)#	73
HDL (clot blood)#	73 3/2566
LDL (clot blood)#	H. 425.8

LABORATORY RI
LANNA HOSPITAL 1 SUKKASEM Rd. T. PATON A. MUA
TEL. 052-134-777 EXT 1154

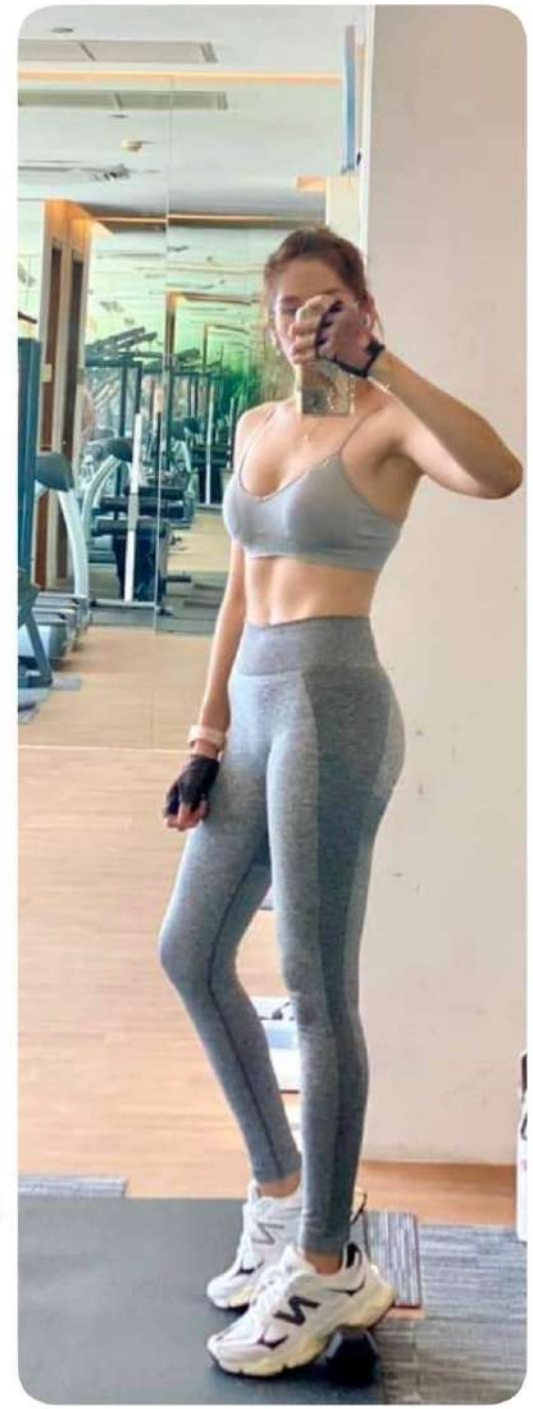
Patient Name น.ส. สิริประกาย ดวงแก้ว Id
Age : 44.1.9 Sex : Female Ethnic Group: Thai

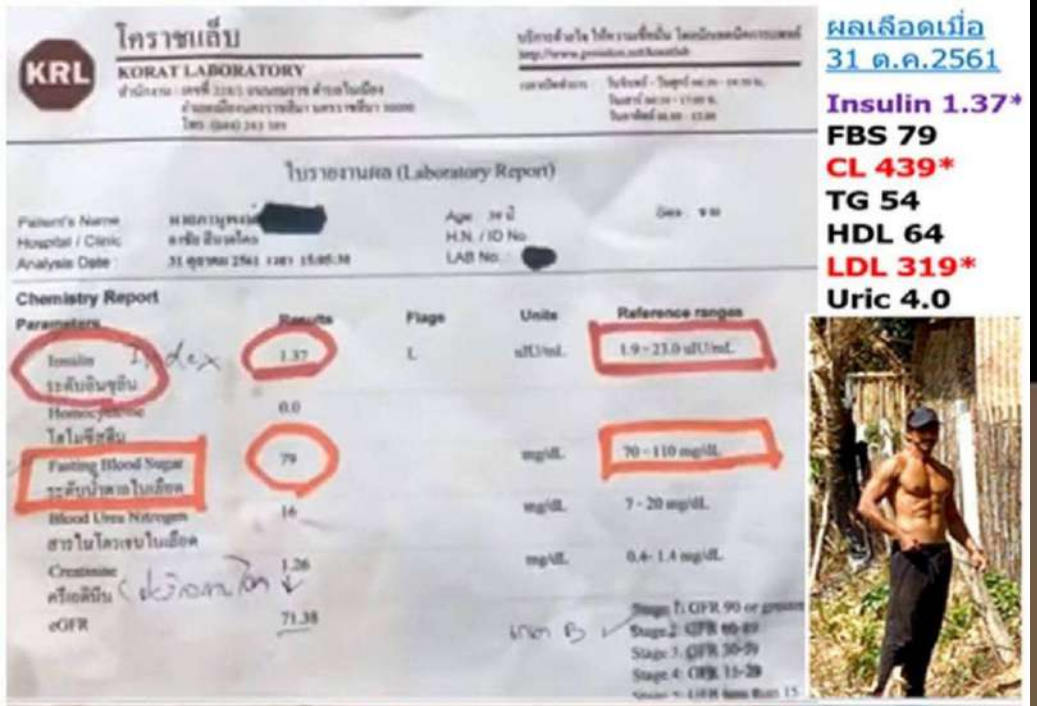
Description	Result
Lipid profile #	
Cholesterol (clot blood)#	515 Repeated
Triglyceride (clot blood)#	58
HDL (clot blood)#	81 6/2566
LDL (clot blood)#	H. 421.5

LABORATORY
LANNA HOSPITAL 1 SUKKASEM Rd. T. PATON A. MU
TEL. 052-134-777 EXT 1154

Patient Name น.ส. สิริประกาย ดวงแก้ว
Age : 44.8.15 Sex : Female Ethnic Group: Thai

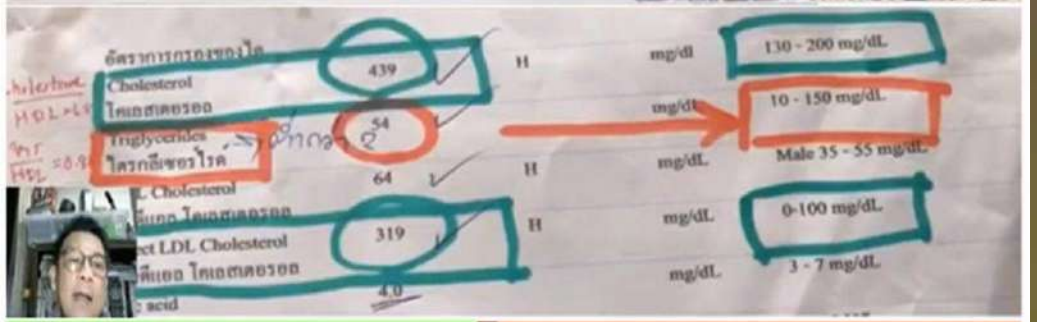
Description	Result
Creatinine (Clot blood)#	0.6
GFR	111
BUN (clot blood) #	14
Lipid profile #	
Cholesterol (clot blood)#	H. 403 10/2566
Triglyceride (clot blood)#	45
HDL (clot blood)#	H. 77
LDL (clot blood)#	H. 317.4
FBS # (NaF blood)(น้ำตาลในเลือด)	81





ผลเลือดเมื่อ
31 ต.ค.2561

Insulin 1.37*
FBS 79
CL 439*
TG 54
HDL 64
LDL 319*
Uric 4.0



Roy Taathaata
3 ชม. ·

คอเลสเตอรอลผมสูง 400++
ตั้งแต่ทำ Fasting เมื่อ 2014 แล้ว
ปัจจุบันก็ผ่านมาร่วม 10 ปี

.

คำถามคือ ผมต้องกินยาลดคอเลสเตอรอล?

ผมต้อง แกล้งทำเป็นป่วยใช่ไหมครับ?
รูปร่าง ผมแบบนี้คือ ไม่ดีต่อสุขภาพ?

.

เฮ้อ เราอยู่ในยุคที่ ความจริงตรงหน้า
สู้งานวิจัยและ ตำแหน่งสถานะทางวิทยาศาสตร์ไม่ได้

.

วิทยาศาสตร์ไม่ใช่ความจริงทั้งหมด
แต่ความจริง คือ ทั้งหมดของวิทยาศาสตร์

"People who are not overweight, have low blood sugar, exercise and are on a low-carb diet typically have optimal triglycerides and HDL, and sometimes they have high LDL. Our findings show that the people who have this healthy combination of diet and lifestyle, as well as high LDL, showed no benefit from taking a statin."

David Diamond, neuroscientist and cardiovascular disease researcher, Department of Psychology, University of South Florida

4. Obesity

Investigation

Blood chemistry

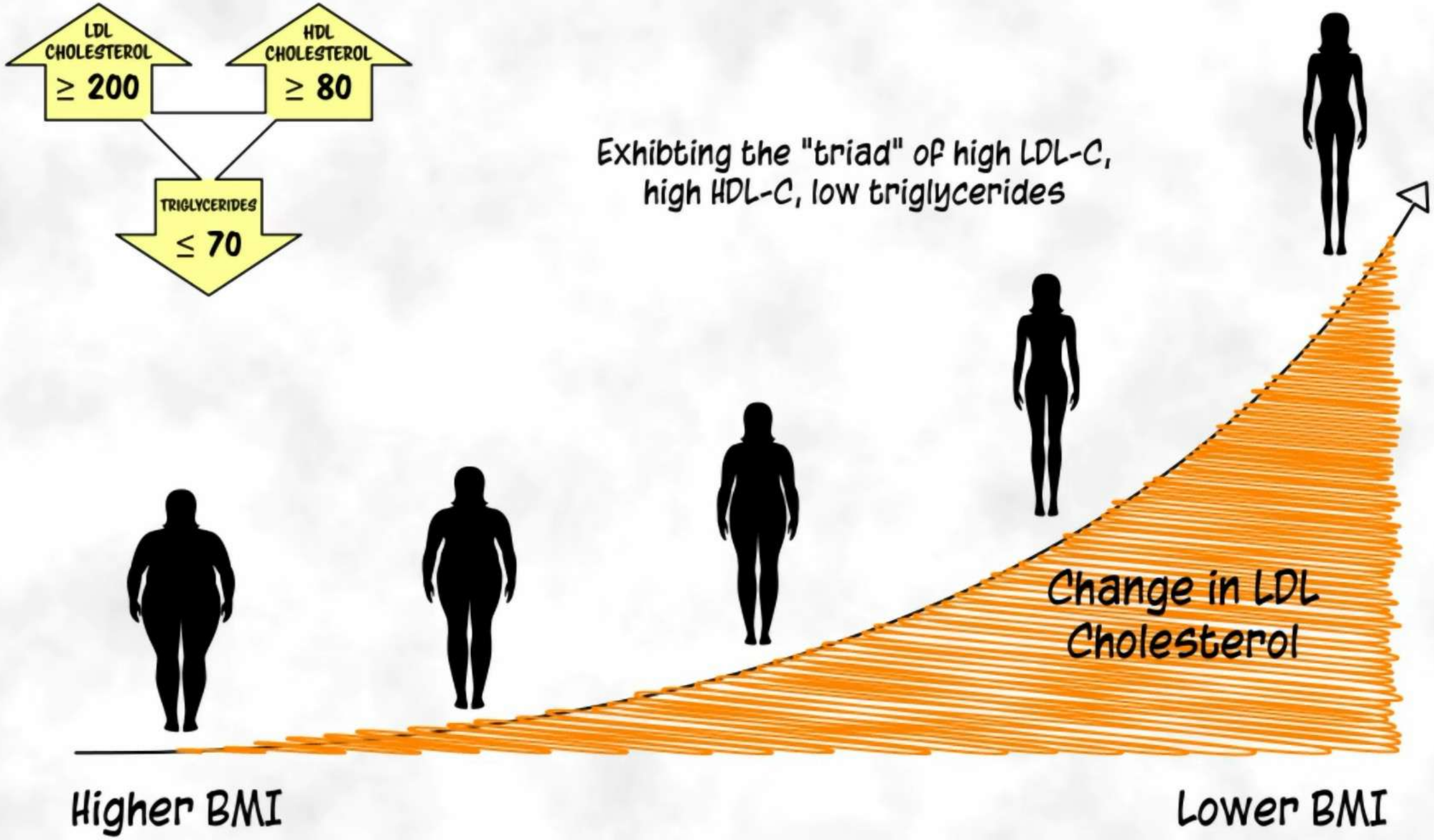
- Blood sugar 407 mg/dL ******
- HbA1C 11.3%. *****
- Lipase 456 U/L (13-60) ******
- Amylase 134 U/L (25-125)
- Urine Amylase 40 U/L (0-650)
- Serum ketone 3.2 mmol/L (0.03-0.30)
- Bun 10 mg/dL
- Cr 0.5 mg/dL, eGFR(CKD-EPI) 155

LFT

- ALT 6 U/L (0-33)
- AST 14 U/L (0-40)
- ALP 56 U/L (40-129)
- TB 0.7 mg/dL (0.3-1.2)
- DB 0.7 mg/dL (0-0.5)
- Cholesterol 735 mg/dL (0-200)
- Total protein 6.6 g/dL (6.6-8.7)
- Albumin 4.6 g/dL (3.5-5.2)
- Globulin 2.0 g/dL (2.6-3.4)

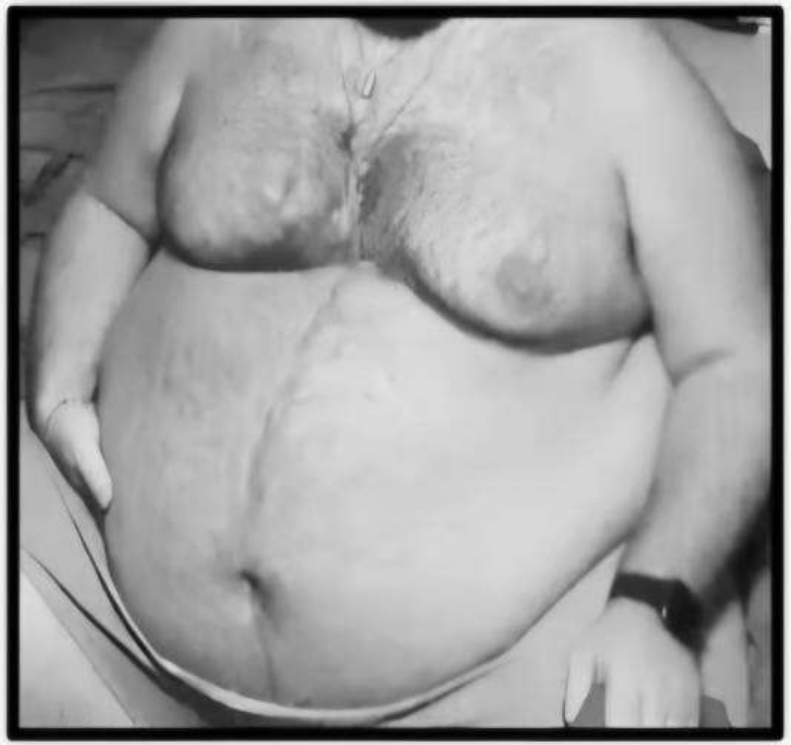
Lipid profile

- Triglyceride 3569 mg/dL *********
- HDL-C 22 mg/dL
- LDL-C 39 mg/dL


LDL CHOLESTEROL
≥ 200
HDL CHOLESTEROL
≥ 80
TRIGLYCERIDES
≤ 70
Exhibting the "triad" of high LDL-C, high HDL-C, low triglycerides
Change in LDL Cholesterol
Higher BMI
Lower BMI

WHO IS AT HIGHER CARDIOVASCULAR RISK?

LDL: 120-140 MG/DL



BMI: 60.5 KG/M2

LDL: >400 MG/DL

BMI: 20.8 KG/M2

> Am Heart J. 2009 Jan;157(1):111-117.e2. doi: 10.1016/j.ahj.2008.08.010. Epub 2008 Oct 22.

Lipid levels in patients hospitalized with coronary artery disease: an analysis of 136,905 hospitalizations in Get With The Guidelines

Amit Sachdeva [1], Christopher P Cannon, Prakash C Deedwania, Kenneth A Labresh, Sidney C Smith Jr, David Dai, Adrian Hernandez, Gregg C Fonarow

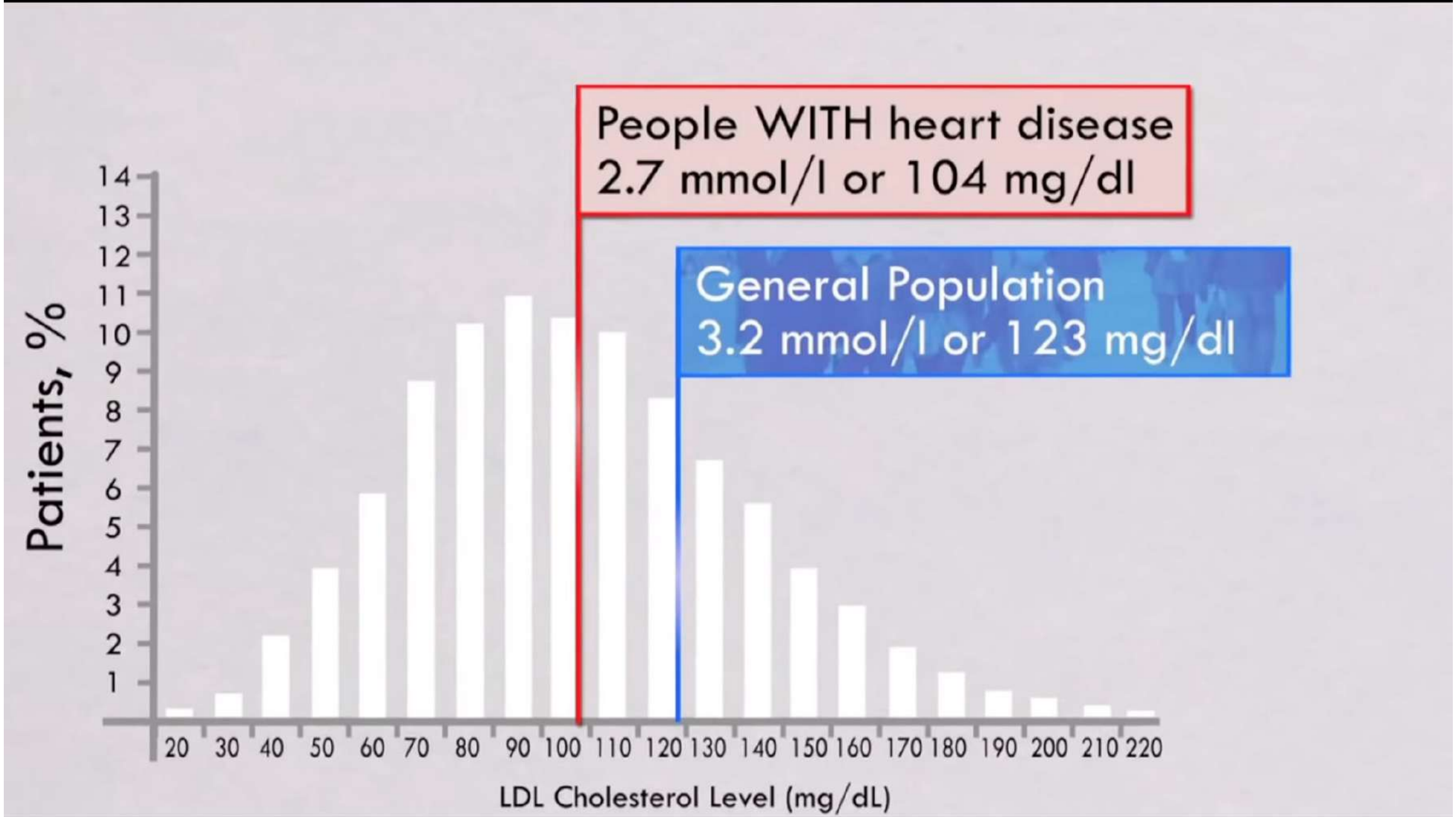
Abstract

Background: Lipid levels among contemporary patients hospitalized with coronary artery disease (CAD) have not been well studied. This study aimed to analyze admission lipid levels in a broad contemporary population of patients hospitalized with CAD.

Methods: The Get With The Guidelines database was analyzed for CAD hospitalizations from 2000 to 2006 with documented lipid levels in the first 24 hours of admission. Patients were divided into low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL), and triglyceride categories. Factors associated with LDL and HDL levels were assessed along with temporal trends.

Results: Of 231,986 hospitalizations from 541 hospitals, admission lipid levels were documented in 136,905 (59.0%). Mean lipid levels were LDL 104.9 +/- 39.8, HDL 39.7 +/- 13.2, and triglyceride 161 +/- 128 mg/dL. Low-density lipoprotein cholesterol <70 mg/dL was observed in 17.6% and ideal levels (LDL <70 with HDL > or =60 mg/dL) in only 1.4%. High-density lipoprotein cholesterol was <40 mg/dL in 54.6% of patients. Before admission, only 28,944 (21.1%) patients were receiving lipid-lowering medications. Predictors for higher LDL included female gender, no diabetes, history of hyperlipidemia, no prior lipid-lowering medications, and presenting with acute coronary syndrome. Both LDL and HDL levels declined over time (P < .0001).

Conclusions: In a large cohort of patients hospitalized with CAD, almost half have admission LDL levels <100 mg/dL. More than half the patients have admission HDL levels <40 mg/dL, whereas <10% have HDL > or =60 mg/dL. These findings may provide further support for recent guideline revisions with even lower LDL goals and for developing effective treatments to raise HDL.


People WITH heart disease
2.7 mmol/l or 104 mg/dl
General Population
3.2 mmol/l or 123 mg/dl
Patients, %
14
13
12
11
10
9
8
7
6
5
4
3
2
1
20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200 210 220
LDL Cholesterol Level (mg/dL)

BMJ Open. 2016 Jun 12;6(6):e010401. doi: 10.1136/bmjopen-2015-010401.

Lack of an association or an inverse association between low-density-lipoprotein cholesterol and mortality in the elderly: a systematic review.

Ravnskov U[1], Diamond DM[2], Hama R[3], Hamazaki T[4], Hammarskjöld B[5], Hynes N[6], Kendrick M[7], Langsjoen PH[8], Malhotra A[9], Mascitelli L[10], McCully KS[11], Ogushi Y[12], Okuyama H[13], Rosch PJ[14], Schersten T[15], Sultan S[6], Sundberg R[16].

Author information

Abstract

OBJECTIVE: It is well known that total cholesterol becomes less of a risk factor or not at all for all-cause and cardiovascular (CV) mortality with increasing age, but as little is known as to whether low-density lipoprotein cholesterol (LDL-C), one component of total cholesterol, is associated with mortality in the elderly, we decided to investigate this issue.

SETTING, PARTICIPANTS AND OUTCOME MEASURES: We sought PubMed for cohort studies, where LDL-C had been investigated as a risk factor for all-cause and/or CV mortality in individuals ≥60 years from the general population.

RESULTS: We identified 19 cohort studies including 30 cohorts with a total of 68 094 elderly people, where all-cause mortality was recorded in 28 cohorts and CV mortality in 9 cohorts. Inverse association between all-cause mortality and LDL-C was seen in 16 cohorts (in 14 with statistical significance) representing 92% of the number of participants, where this association was recorded. In the rest, no association was found. In two cohorts, CV mortality was highest in the lowest LDL-C quartile and with statistical significance; in seven cohorts, no association was found.

CONCLUSIONS: High LDL-C is inversely associated with mortality in most people over 60 years. This finding is inconsistent with the cholesterol hypothesis (ie, that cholesterol, particularly LDL-C, is inherently atherogenic). Since elderly people with high LDL-C live as long or longer than those with low LDL-C, our analysis provides reason to question the validity of the cholesterol hypothesis. Moreover, our study provides the rationale for a re-evaluation of guidelines recommending pharmacological reduction of LDL-C in the elderly as a component of cardiovascular disease prevention strategies.

KEYWORDS: EPIDEMIOLOGY; GERIATRIC MEDICINE; PREVENTIVE MEDICINE; Risk factor, LDL-cholesterol, cardiovascular mortality, total mortality, elderly,

PMID: 27292972 PMCID: PMC4908872 DOI: 10.1136/bmjopen-2015-010401

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Study name Author, Year* Follow-up *Quality*	All-cause Mortality	CV Mortality	Stroke	MI	Revascularization	Composite CV Outcomes
ASCOT-LLA Sever, 2003[5] 3 years *Fair*	3.6% (185/5168) vs. 4.1% (212/5137) HR 0.87 (95% CI, 0.71 to 1.06) RR 0.87 (95% CI, 0.71 to 1.05) ARD −0.55% (95% CI, −1.29 to 0.20) NNT 182	1.4% (74/5168) vs. 1.6% (82/5137) HR 0.90 (95% CI, 0.66 to 1.23) RR 0.90 (95% CI, 0.66 to 1.23) ARD −0.16% (95% CI, −0.64 to 0.31) NNT 625	*Fatal and nonfatal stroke*: 1.7% (87/5168) vs. 2.3% (121/5137) HR 0.73 (95% CI, 0.59 to 0.96) RR 0.73 (95% CI, 0.56 to 0.96) ARD −0.63% (95% CI −1.18 to −0.09) NNT 159	*Fatal and nonfatal MI*: 2.2% (114/5168) vs. 3.3% (171/5137) RR 0.66 (95% CI, 0.52 to 0.84) ARD −1.10% (95% CI, −1.73 to −0.47) NNT 91	NR	*Fatal CHD, nonfatal MI, chronic stable angina, unstable angina, or fatal and nonfatal heart failure*: 3.4% (178/5168) vs. 4.8% (247/5137) HR 0.71 (95% CI, 0.59 to 0.86) RR 0.72 (95% CI, 0.59 to 0.87) ARD −1.36% (95% CI, −2.13 to −0.60) NNT, 74
ASPEN Knopp, 2006[6] 4 years[†] *Fair*	4.6% (44/959) vs. 4.3% (41/946) RR 1.06 (95% CI, 0.70 to 1.60) ARD 0.25% (95% CI, −1.60 to 2.11) NNH 400	NR	*Fatal and nonfatal stroke*: 2.8% (27/959) vs. 3.1% (29/946) RR 0.92 (95% CI, 0.55 to 1.54) ARD −0.25% (95% CI, −1.77 to 1.27) NNT 400	*Fatal and nonfatal MI*: 2.9% (28/959) vs. 3.6% (34/946) RR 0.81 (95% CI, 0.50 to 1.33) ARD −0.67% (95% CI, −2.27 to 0.92) NNT 149	NR	*CV event*: 10.4% (100/959) vs.10.8% (102/946) HR 0.97 (95% CI, 0.74 to 1.28) RR 0.97 (95% CI, 0.75 to 1.26) ARD −0.35% (95% CI, −3.12 to 2.41) NNT 286
ASTRONOMER Chan, 2010[7] 4 years *Good*	NR	1.5% (2/134) vs. 3.7% (5/135) RR 0.40 (95% CI, 0.08 to 2.04) ARD −2.21% (95% CI, −6.00 to −1.58) NNT 45	*Fatal and nonfatal stroke*: 0% (0/134) vs. 0.7% (1/135) RR 0.34 (95% CI, 0.01 to 8.17) ARD −0.74% (95% CI, −2.77 to 1.29) NNT 135	*Fatal and nonfatal MI*: 0% (0/134) vs. 2.2% (3/135) RR 0.14 (95% CI, 0.01 to 2.76) ARD −2.22% (95% CI, −5.07 to 0.63) NNT 45	NR	NR
Beishuizen, 2004[8] 2 years *Fair*	2.9% (3/103) vs. 5.1% (4/79) RR 0.58 (95% CI, 0.13 to 2.50) ARD −2.15% (95% CI, −7.79 to 3.67) NNT 47	NR	NR	NR	NR	*Unspecified CV events*: , 1.9% (2/103) vs. 15.1% (12/79) RR 0.13 (95% CI, 0.03 to 0.55) ARD 13.25% (95% CI −21.60 to −4.90) NNT 8

Study name **Author, Year*** **Follow-up** ***Quality***	**All-cause Mortality**	**CV Mortality**	**Stroke**	**MI**	**Revascularization**	**Composite CV Outcomes**
JUPITER Ridker, 2008[16] 2 years *Good*	2.2% (198/8901) vs. 2.8% (247/8901) HR 0.80 (95% CI, 0.67 to 0.97) RR 0.80 (95% CI, 0.67 to 0.96) ARD −0.55% (95% CI, −1.01 to −0.09) NNT 182	0.3% (29/8,901) vs. 0.4% (37/8,901) RR 0.78 (95% CI, 0.48 to 1.27) ARD −0.09% (95% CI, −0.27 to 0.09) NNT 1,111	*Fatal or nonfatal stroke*: , 0.4% (33/8901) vs. 0.7% (64/8901) HR 0.52 (95% CI, 0.34 to 0.79) RR 0.52 (95% CI, 0.34 to 0.78) ARD, −0.35% (95% CI, −0.56 to −0.13) NNT 286 *Fatal stroke*: 0.03% (3/8901) vs. 0.06% (6/8901) RR 0.50 (95% CI, 0.13 to 2.00) ARD, −0.03% (95% CI, −0.10 to 0.03) NNT 3333 *Nonfatal stroke*: 0.3% (30/8901) vs. 0.7% (58/8901) RR 0.52 (95% CI, 0.33 to 0.80) ARD −0.31% (95% CI −0.52 to −0.11) NNT 323	*Fatal and nonfatal MI*: 0.3% (31/8901) vs. 0.8% (68/8901) HR 0.35 (95% CI, 0.22 to 0.58) RR 0.46 (95% CI, 0.30 to 0.70) ARD −0.43% (95% CI, −0.65 to −0.21) NNT 233 *Fatal MI*: 0.1% (9/8901) vs. 0.07% (6/8901) RR 1.50 (95% CI, 0.53 to 4.21) ARD 0.04% (95% CI, −0.20 to 0.13) NNH 2500 *Nonfatal MI*: 0.2% (22/8901) vs. 0.7% (62/8901) HR 0.35 (95% CI, 0.22 to 0.58) RR 0.35 (95% CI, 0.22 to 0.58) ARD −0.45% (95% CI, 0.65 to −0.25) NNT 222	0.8% (71/8901) vs. 1.5% (131/8901) HR 0.54 (95% CI, 0.41 to 0.72) RR 0.54 (95% CI, 0.41 to 0.72) ARD −0.67% (95% CI, −0.99 to −0.36) NNT 149	*Nonfatal MI, nonfatal CVA, hospitalization for unstable angina, arterial revascularization or CV mortality*: 2% (142/8901) vs. 3% (251/8901) HR 0.56 (95% CI, 0.46 to 0.69) RR 0.57 (95% CI, 0.46 to 0.69) ARD −1.16% (95% CI, −1.59 to −0.72) NNT 86


คุณป่วย"คอเลสเตอรอลสูง"


"ผลเลือดคุณดีมากครับ ทานยาต่อไป"

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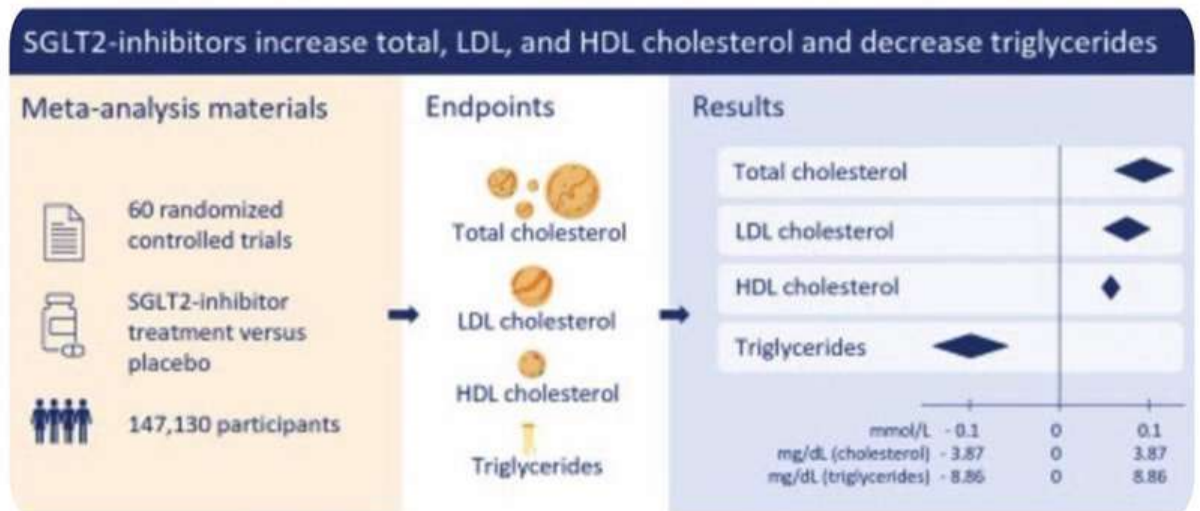
SGLT2-inhibition increases total, LDL, and HDL cholesterol and lowers triglycerides: Meta-analyses of 60 randomized trials, overall and by dose, ethnicity, and drug type

Louise E Bechmann [1], Frida Emanuelsson [2], Børge G Nordestgaard [3], Marianne Benn [4]



Conclusion: In meta-analyses, SGLT2-inhibition increased total, LDL, and HDL cholesterol and decreased triglycerides. Effect sizes varied slightly by drug dose and ethnicity but were generally robust by drug type.

Keywords: All-cause mortality; Glucose-lowering; Heart failure; Major cardiovascular event; Randomized trial.

Carbohydrate restriction-induced elevations in LDL-cholesterol and atherosclerosis: The KETO Trial

NCT05733325

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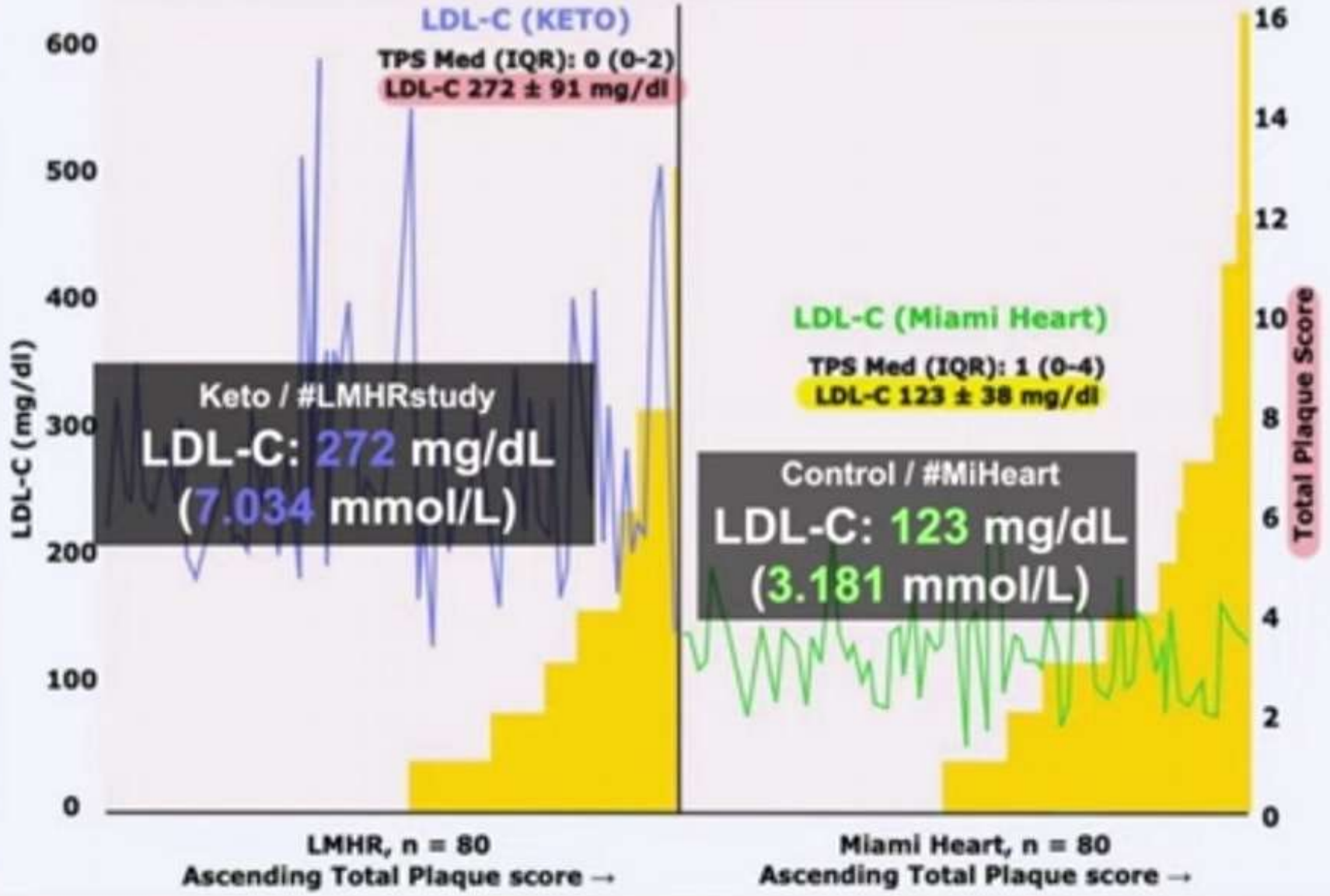
METHODS: 80 of our keto participants with were matched 1:1 for age, gender, race, diabetes mellitus, hyperlipidemia, hypertension, and past smoking to asymptomatic subjects from the MiHeart cohort.

	KETO n = 80	MiHeart n=80	P
Age (years)	55.5 ± 7.9	55.5 ± 7.4	0.951[a]
Duration on ketogenic diet (years)	4.7 ± 2.8	--	
Body Mass Index (kg/m^2)	22.5 ± 2.7	25.8 ± 3.6	<.001[a]
Male (%)	47 (59)	47 (59)	-
Race			-
White, non-Hispanic	72 (90)	72 (90)	
Asian/ Asian-Indian	2 (3)	2 (3)	
Hispanic	6 (8)	6 (8)	
Lipid markers			
Total Cholesterol, mg/dL	369 ± 95	205 ± 40	<0.001[a]
LDL-C, mg/dL	272 ± 91	123 ± 38	<0.001[a]
Non-HDL-C, mg/dL	279 ± 90	142 ± 40	<0.001[a]
HDL-C, mg/dL	90 ± 20	63 ± 19	<0.001[a]
Triglycerides, mg/dL	64 ± 23	96 ± 45	<0.001[a]
Other risk factors or medications			
Systolic BP, mmHg	117 ± 12	116 ± 10	0.488[a]
Diastolic BP, mmHg	76 ± 8	73 ± 6	0.012[a]
hsCRP (mg/L)[&]	0.5 [0.3-0.9]	0.7 [0.4-1.5]	0.007[c]
Hemoglobin A1C (%)	5.4 ± 0.3	5.5 ± 0.2	0.075[a]
Hyperlipidemia Medication	0 (0)	26 (33)	-
Hypertension Medication	1 (1)	0 (0)	-
Past smoker	2	2	-



FINDINGS

At baseline, KETO subjects exhibited mean LDL-C 272 ± 91, HDL-C 90 ± 20, and TG 64 ± 23 mg/dl. Mean age and time on a ketogenic diet were 55 years, and 4.7 years.

When matched to the Miami Heart cohort (LDL-C of 123 ± 38 gm/dl), KETO group exhibited no increased plaque burden at baseline, despite almost 5 years of extreme hypercholesterolemia.

There was no significant correlation between LDL-C and CCTA-measured total plaque.

CONCLUSIONS

- After mean duration of **4.7 years** with carbohydrate restriction-induced elevations in **LDL-C (mean 272 mg/dl)**, a metabolically healthy cohort of **LMHR and near-LMHR** subjects on a **ketogenic diet did not have greater atherosclerotic burden** than participants from a population-based cohort with similar risk profiles but markedly lower LDL-C.

- There was also **no correlation between LDL-C level and plaque burden**

Open Access Communication

Oreo Cookie Treatment Lowers LDL Cholesterol More Than High-Intensity Statin therapy in a Lean Mass Hyper-Responder on a Ketogenic Diet: A Curious Crossover Experiment

by **Nicholas G. Norwitz** [1,*] and **William C. Cromwell** [2]

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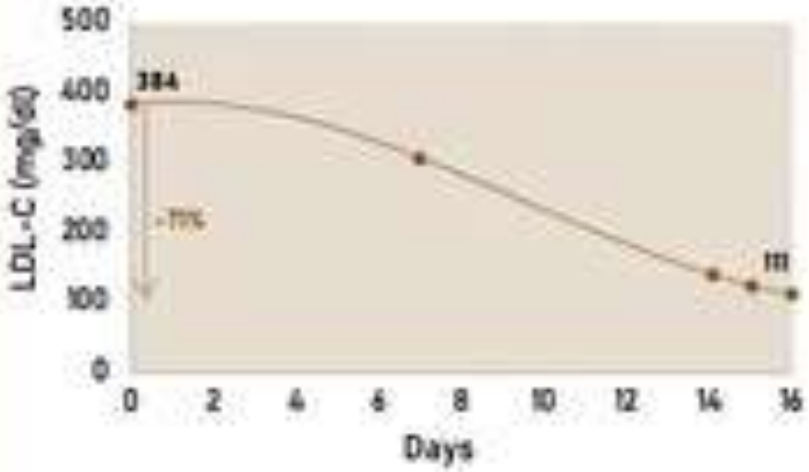
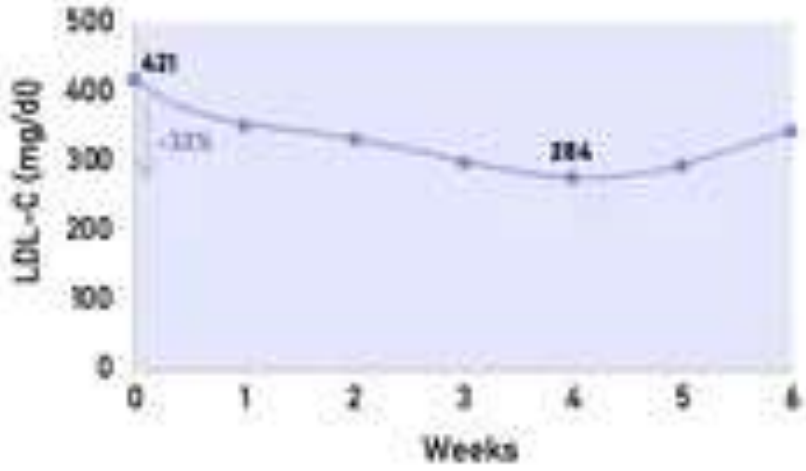
(This article belongs to the Section **Nutrition and Metabolism**)

Oreo Cookies
Lowers LDL Cholesterol More Than Statin Therapy in a Lean Mass Hyper-Responder on a Ketogenic Diet





This experiment was intended as a metabolic demonstration and is not meant to imply any form of health advice.


Thanks for your
Attention