Obesity-the growing epidemic

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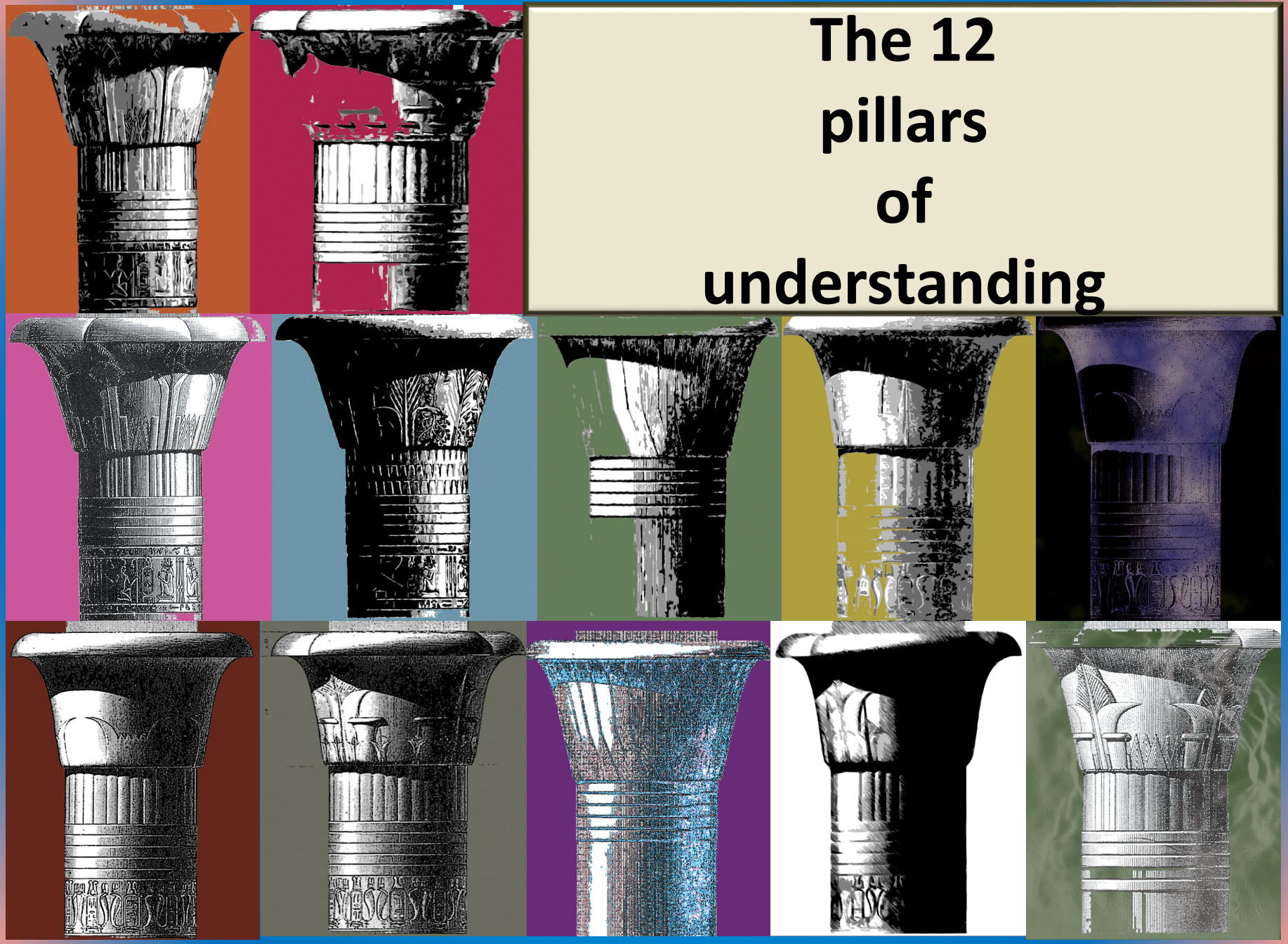


The 12 pillars of understanding

1.Recognizing the magnitude of the disease:

Epidemiology

10 Billion

is the estimated size of the global population by 2015

900 Million

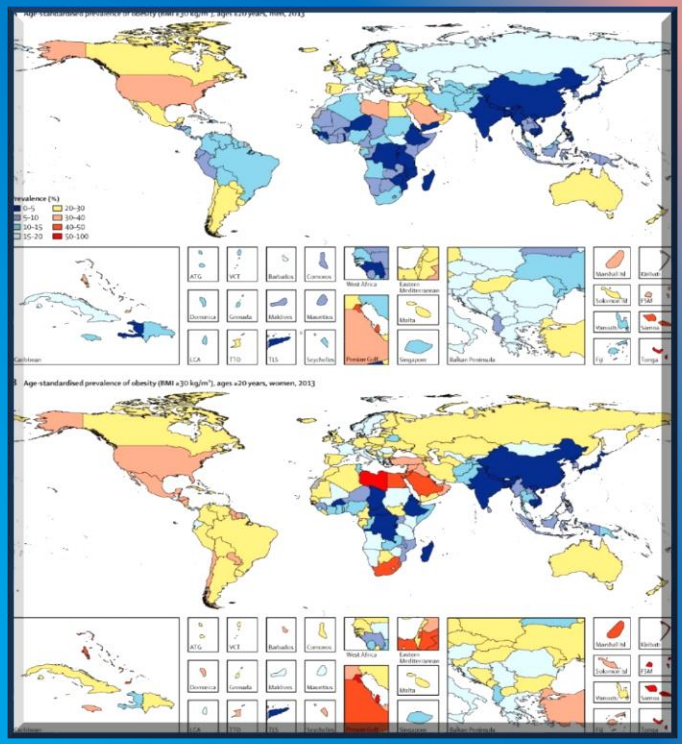
People worldwide are hungry

2 billion have micronutrient deficiencies and

2 billion are overweight or obese

SA statistics

►46% of the world's 600 million obese people are from the developing world

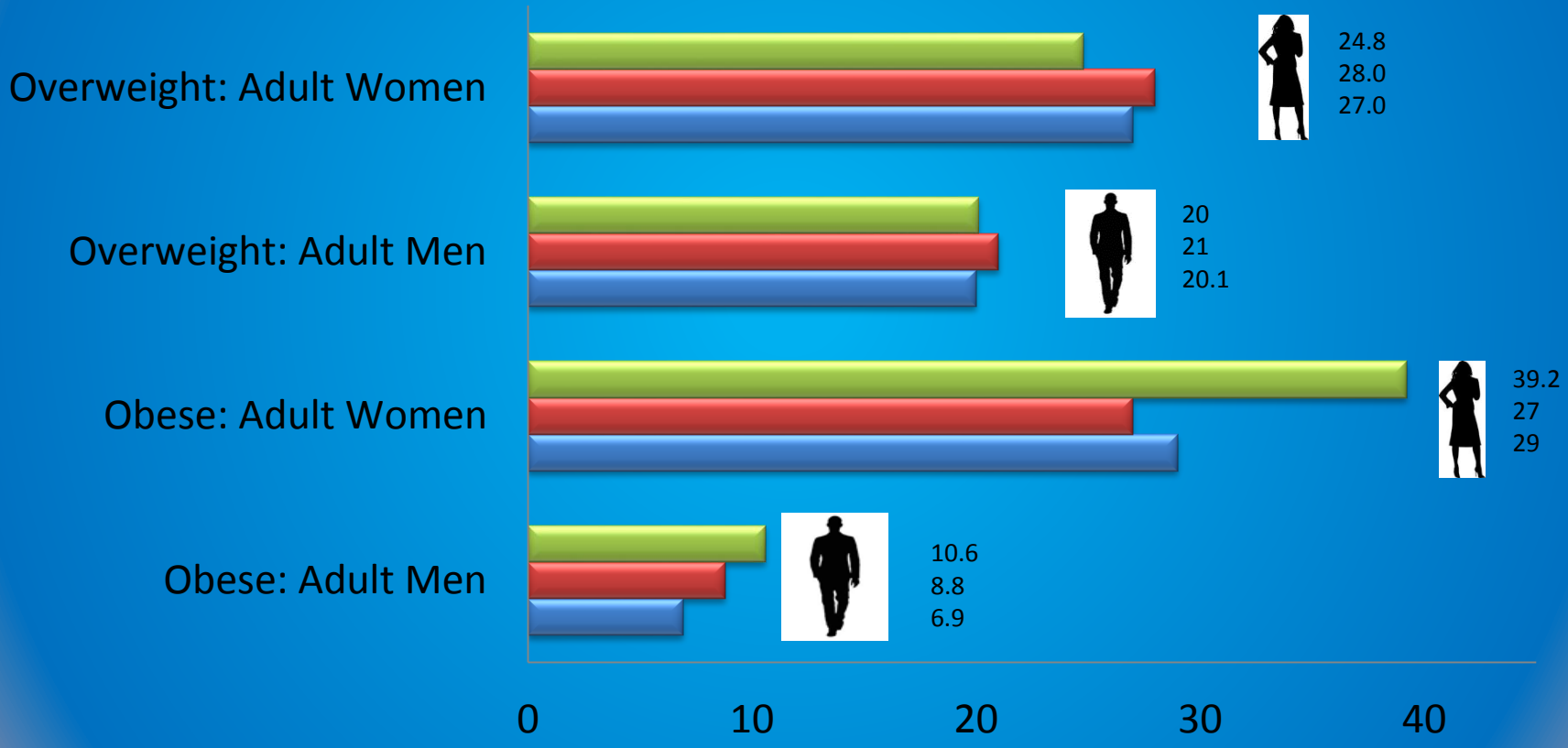


- **50% of SA's will die before 65y from chronic disease**
- **15% of SA's buy monthly "vitamins" to aid "weight loss"**
- **40 % of SA's buy OTC's for their weight related co morbidities.**

Age-standardised regional and national estimates of the prevalence of and obesity men and women for 2013, for 188 countries and 21 GBD regions
Lancet 2014: online 60460-8

South Africa is becoming

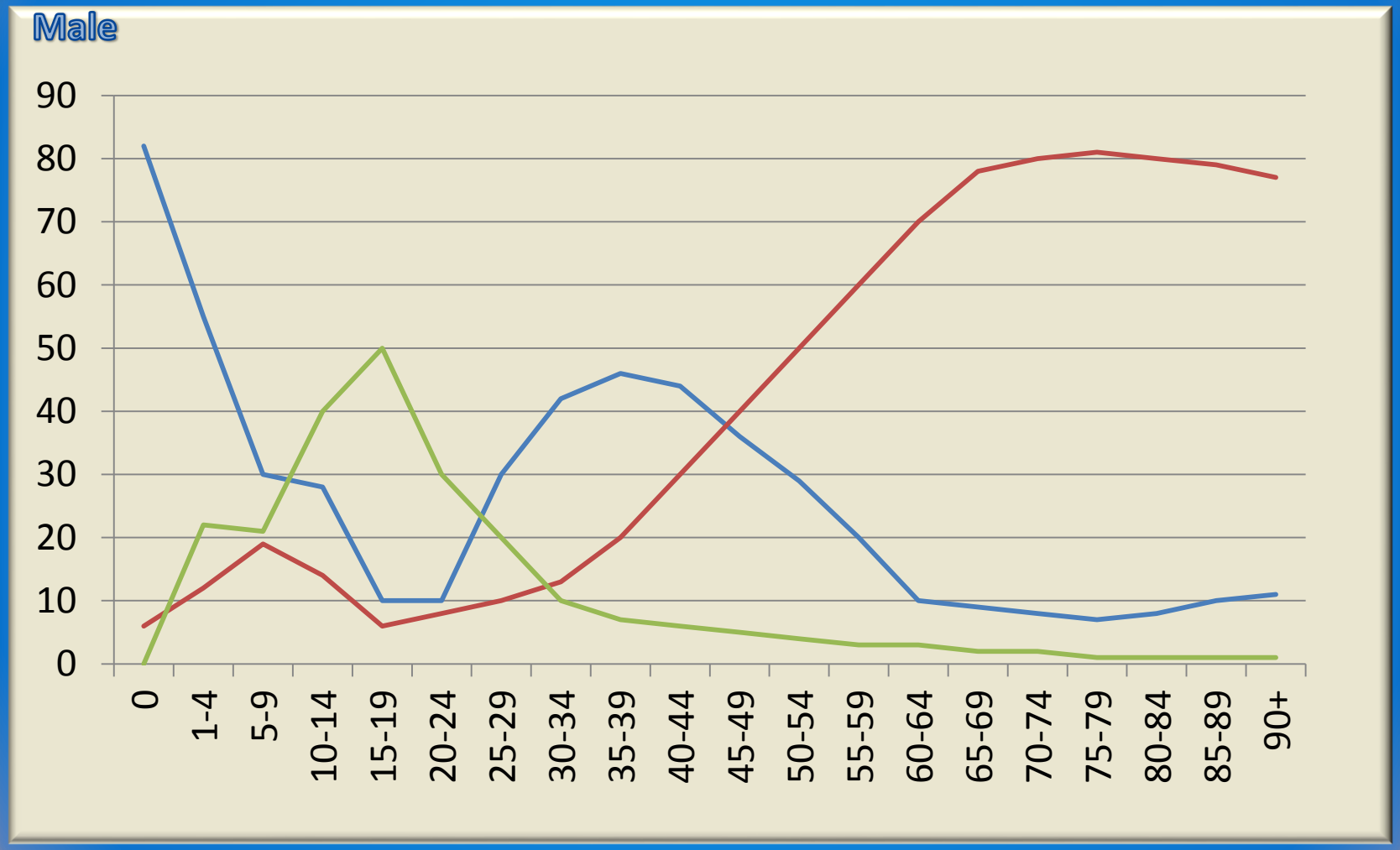
a fatter nation



** South African Demographic & Health Survey*
** South African National Health & Nutrition Examination Survey*

NCD'S STRIKING EARLIER

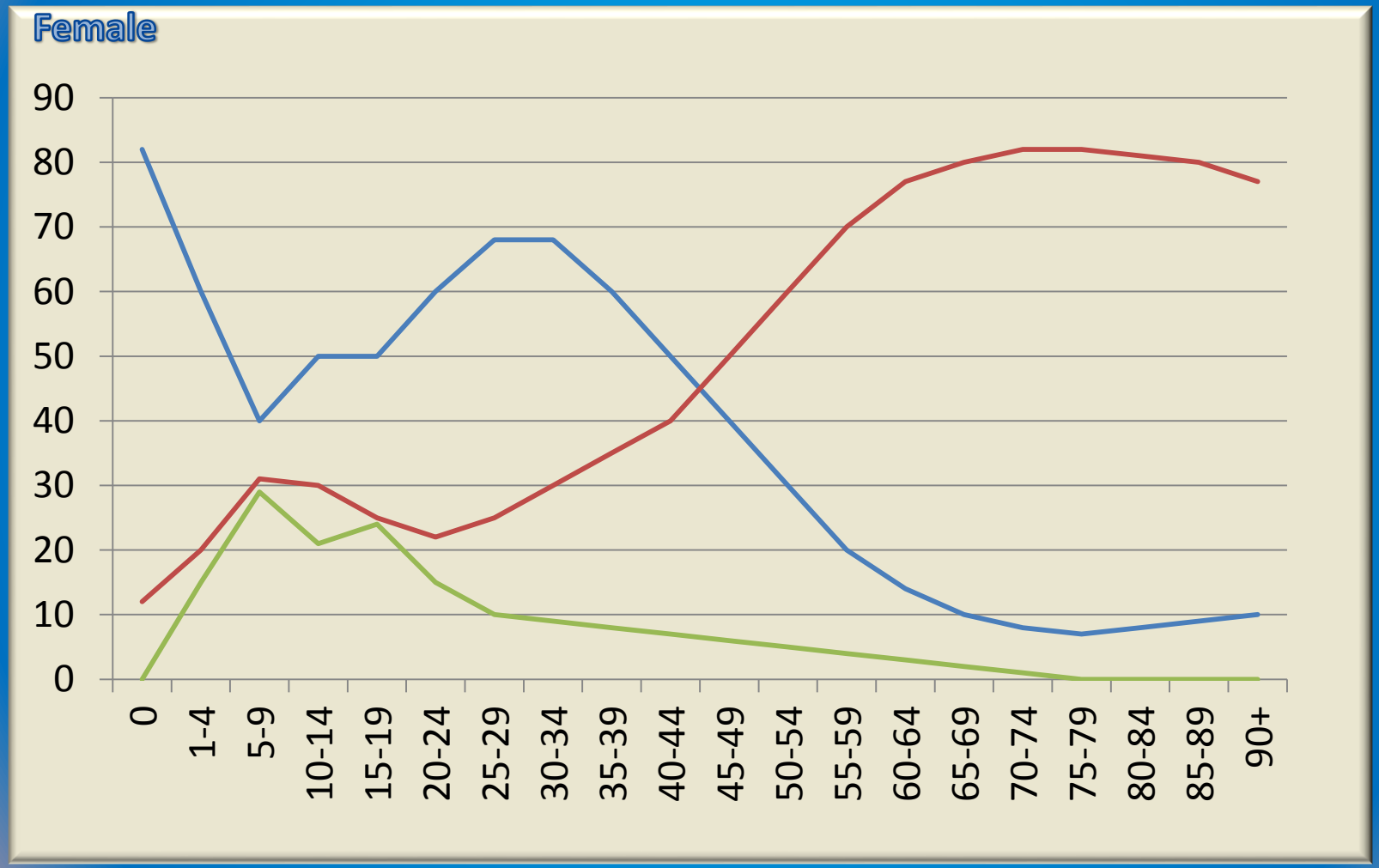
Percentage of total deaths by age group, 2013



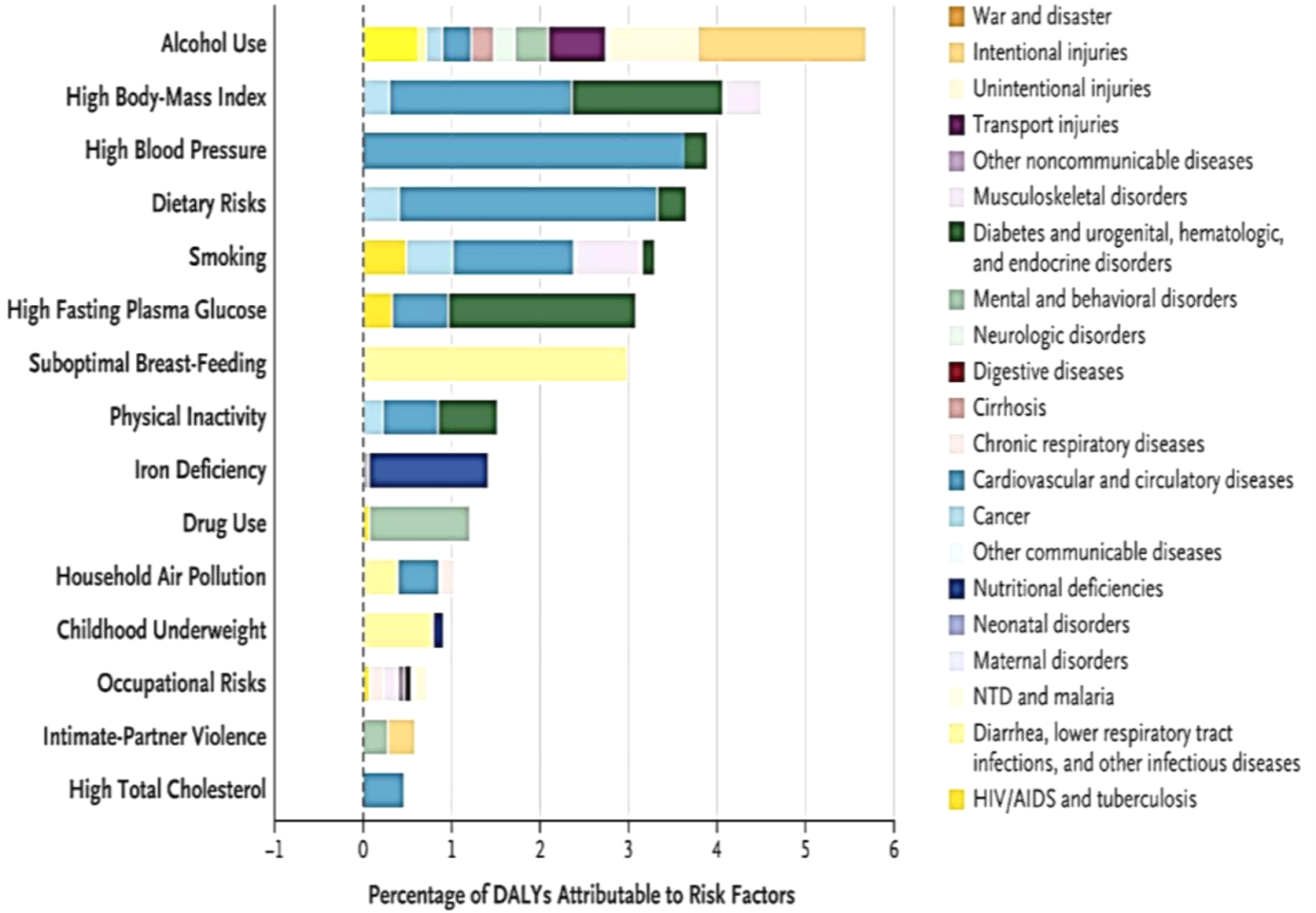
Financial Mail, Dec 2014

NCD'S STRIKING EARLIER

Percentage of total deaths by age group, 2013



Financial Mail Dec 2014



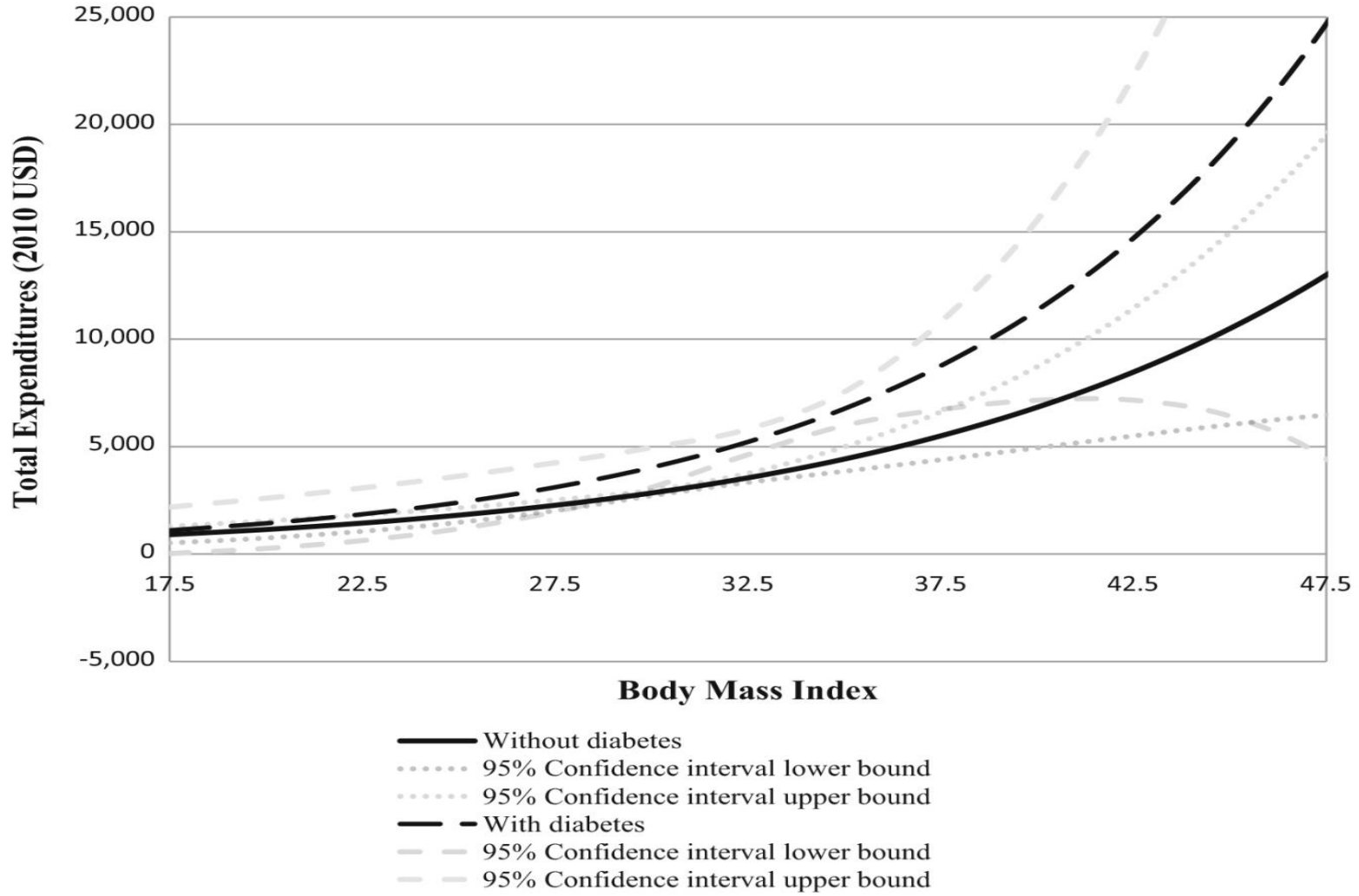
Benatar S. *et al* N Eng J Med 2014

2. Health Economic Burden of the disease

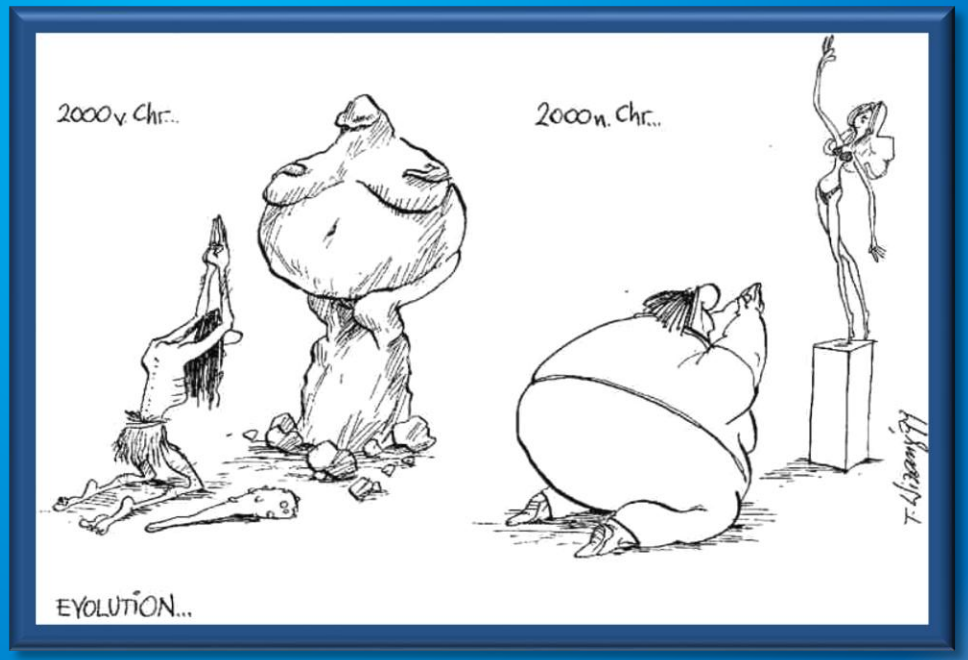
- Each 1-point increase in BMI yields a **4%** increase in medical costs and a **7%** increase in pharmaceutical costs (Wang *et al*).
- Direct medical costs are **42%** higher among obese adults compared to their normal weight counterparts.
- Indirect costs due to obesity, including absenteeism and reduced productively whilst working has been shown to exceed the direct costs.
- Obesity is now responsible for **>9%** of all medical expenditures,

- Combined value of these costs > R**60 000/year/ capita.**

The Medical Care Costs of Adult Obesity: Per Case and Aggregate for the USA



3. Genetic and Epigenetic control of Obesity



Rare to find MZ twins that are discordant for weight

clear evidence for the well-known fact that body weight is **one of the most heritable complex traits found in man**

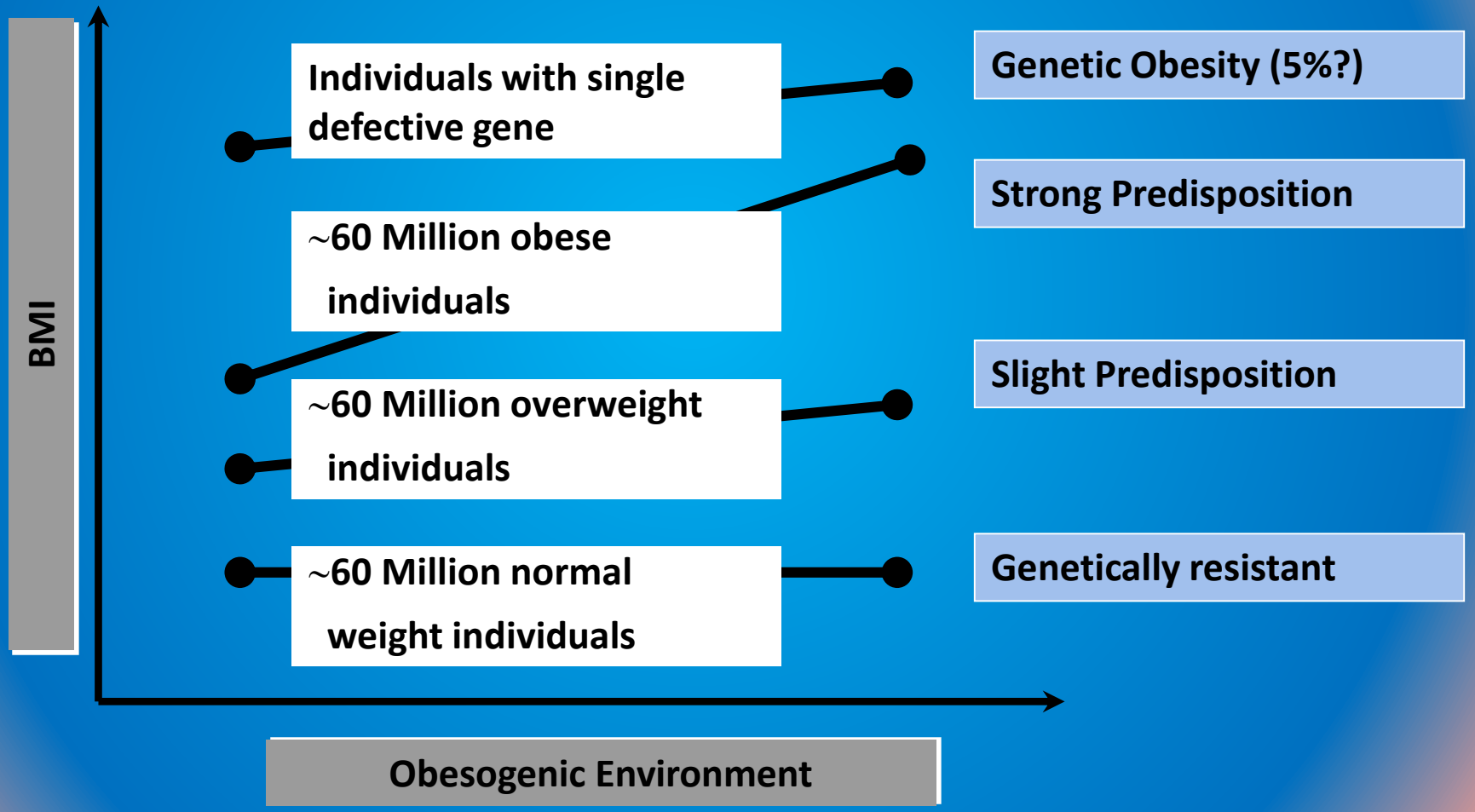
only marginally less heritable than height

- Both baseline BMI and weight gain are genetically determined, and they are each regulated by a different set of genes
- While one set of genes may determine how big you are, other genes may determine how large you can get

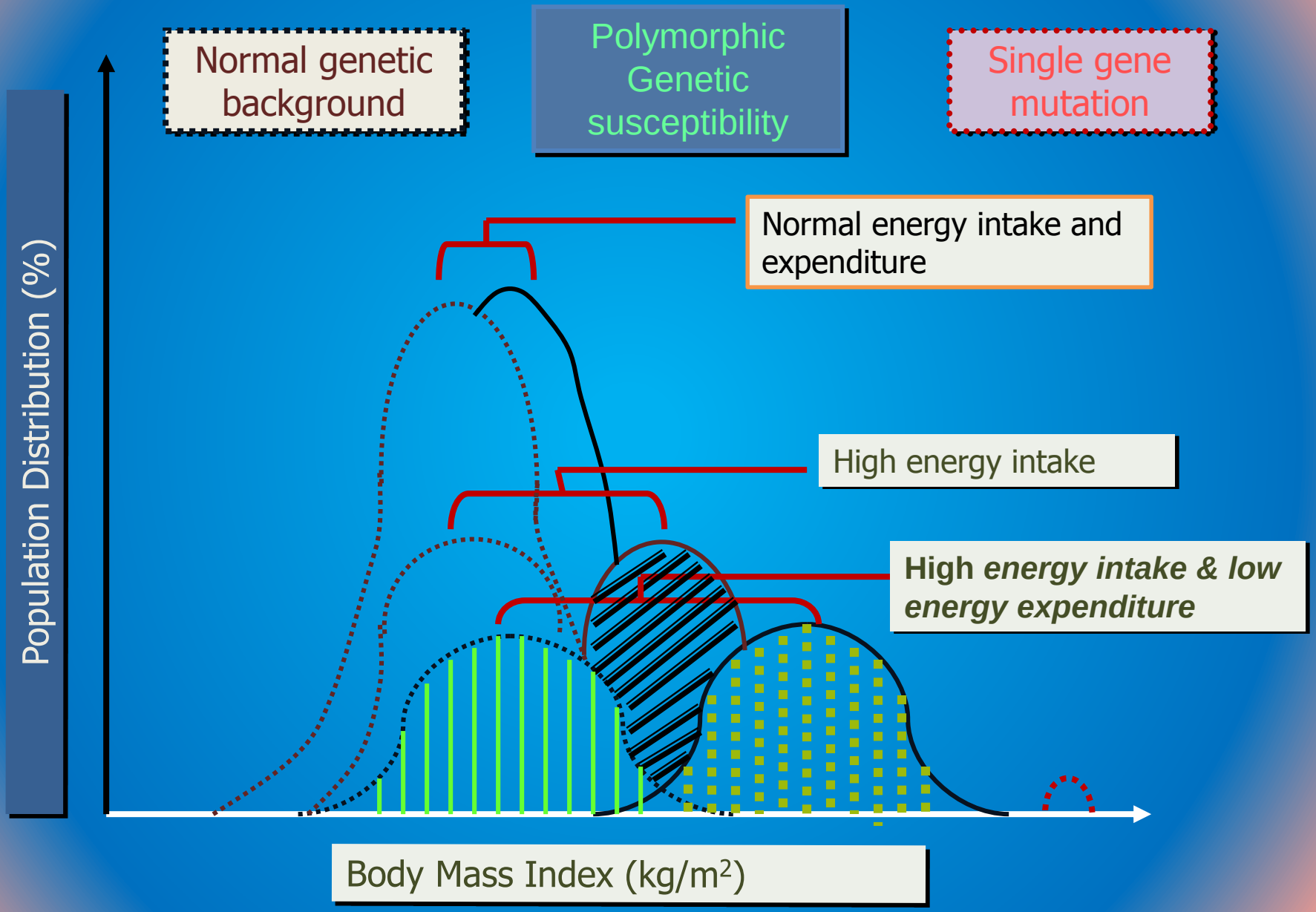
Hjelmsberg and Colleagues – cohort of Finnish twins

GENETIC SUSCEPTIBILITY

- USA Adult Population

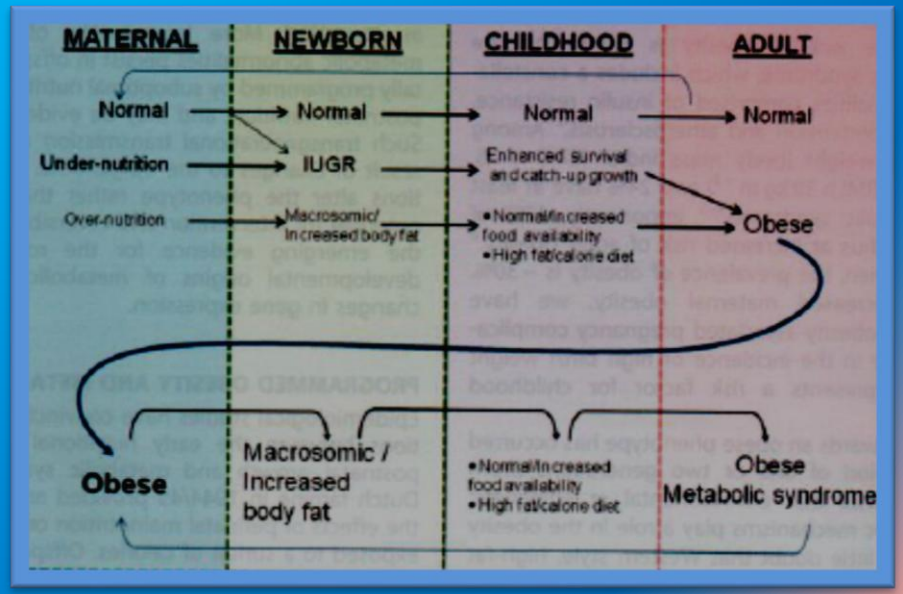


Bouchard, Rankinen. Ob Management. 2005

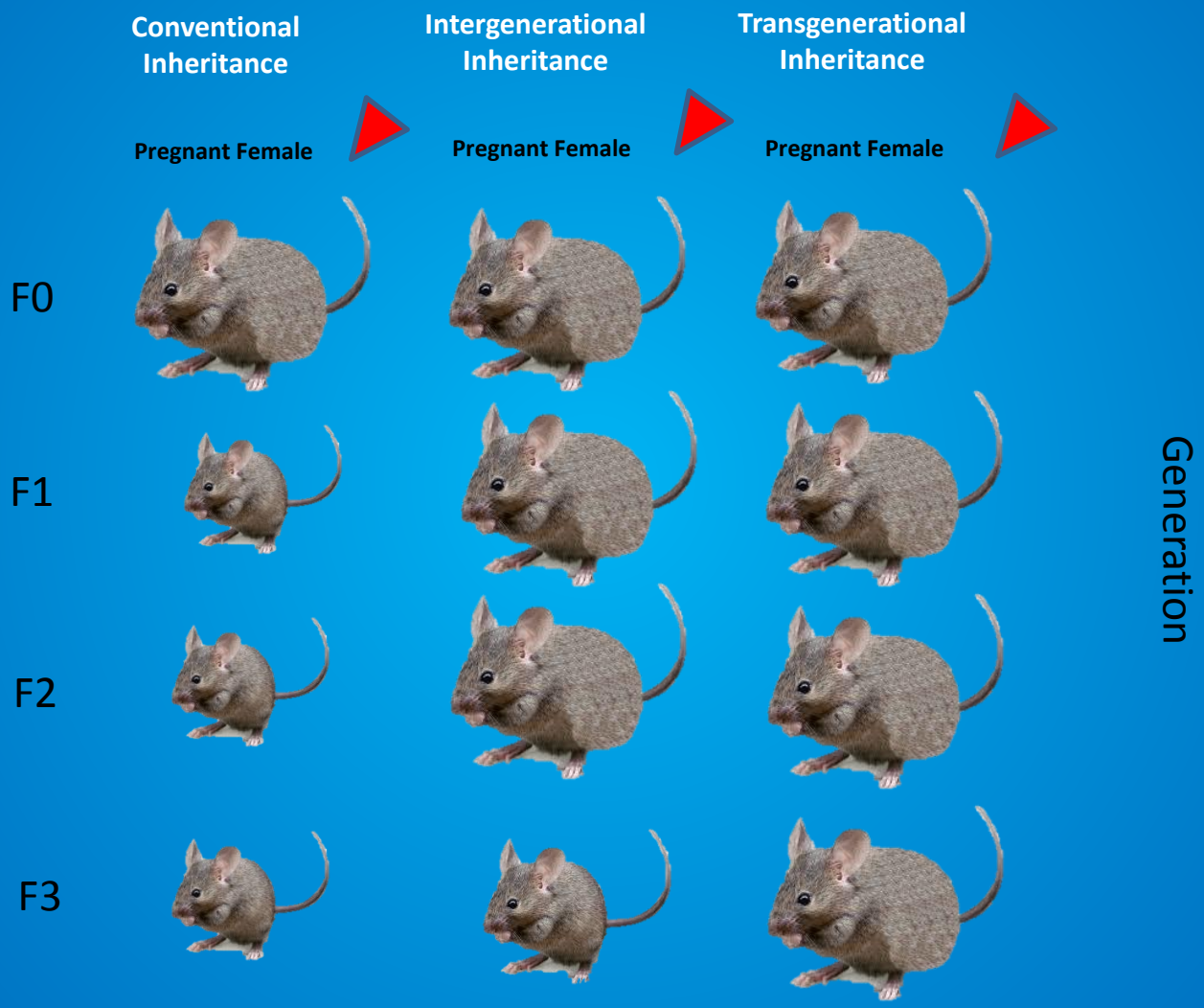

Normal genetic background
Polymorphic Genetic susceptibility
Single gene mutation
Population Distribution (%)
Normal energy intake and expenditure
High energy intake
High *energy intake & low energy expenditure*
Body Mass Index (kg/m²)

- **List of studies demonstrating trans generational inheritance of metabolic disease**
- **Genetic modification: change in base sequence of DNA**
- **Epigenetic modification: chemical modification to DNA**
- **Epigenetic marks are heritable through mitotic cell division by either methylation or acetylation**

Genome is largely stable but epi-genome has the potential to be irreversibly modified by exposure to a range of nutritional and environmental factors

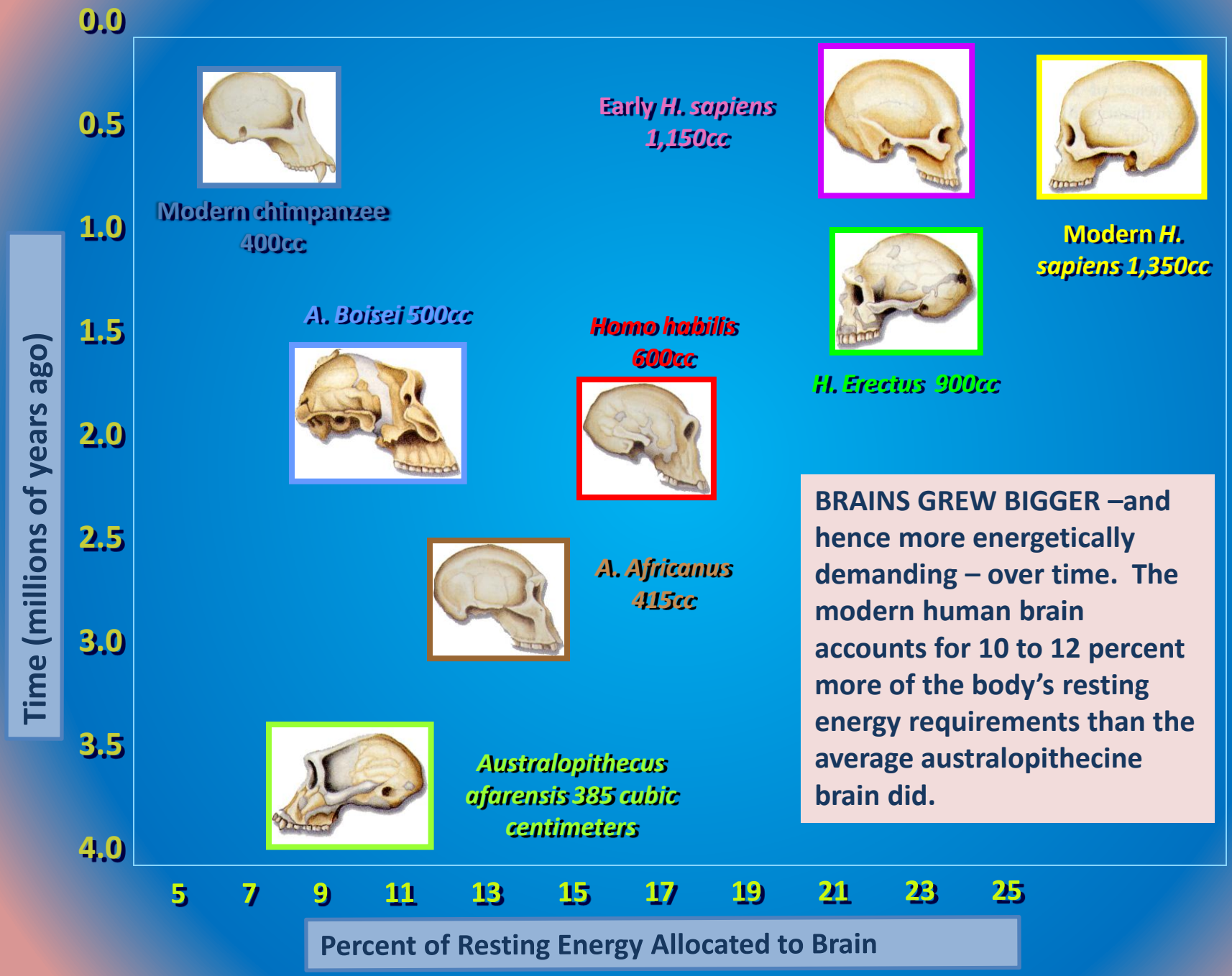


Trans generational inheritance of metabolic disease



4. Understand the complex Aetiology

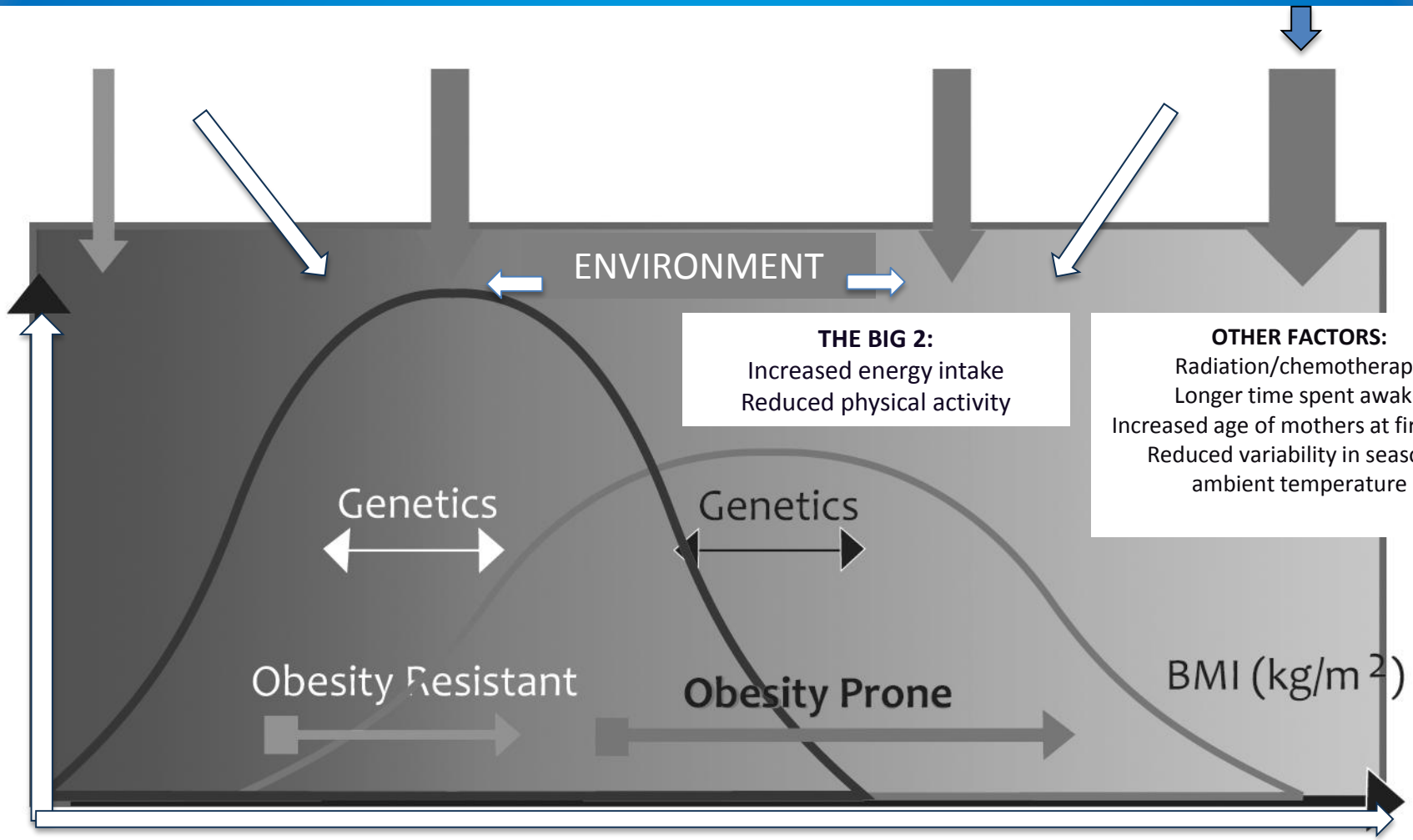
- Neuropeptides
- Gut peptides
- Stress axis/cortisol
- Biological factors
- Inflammation-cytokines
- Adipogenesis
- Endocrinopathies
- Medication
- Gender related
- Social/economic


0.0
0.5
1.0
1.5
2.0
2.5
3.0
3.5
4.0
Time (millions of years ago)
Modern chimpanzee 400cc
Early H. sapiens 1,150cc
Modern H. sapiens 1,350cc
A. Boisei 500cc
Homo habilis 600cc
H. Erectus 900cc
A. Africanus 415cc
Australopithecus afarensis 385 cubic centimeters
BRAINS GREW BIGGER –and hence more energetically demanding – over time. The modern human brain accounts for 10 to 12 percent more of the body's resting energy requirements than the average australopithecine brain did.
5
7
9
11
13
15
17
19
21
23
25
Percent of Resting Energy Allocated to Brain

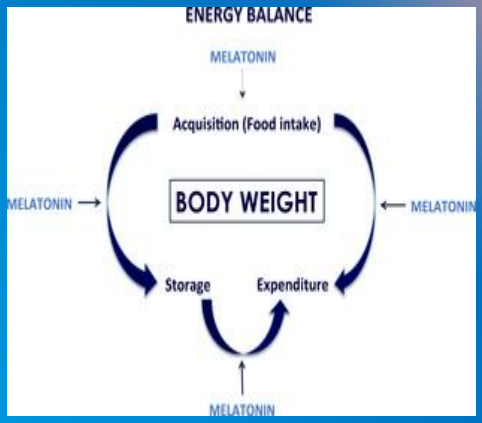
Obesogenic Susceptibility

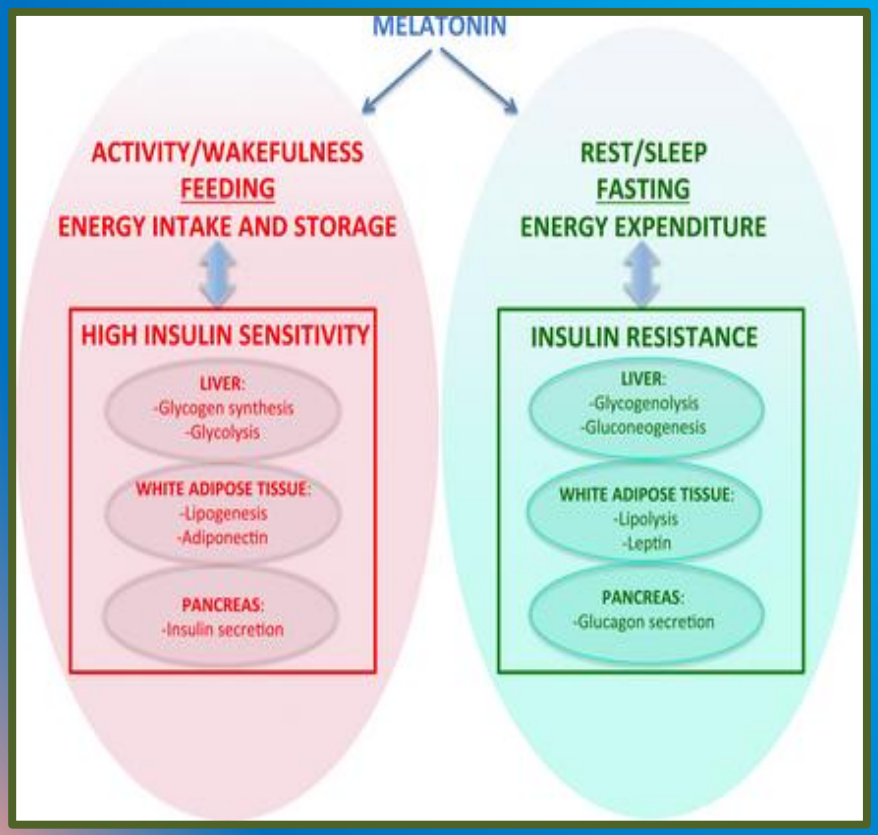
Obesogenic Susceptibility

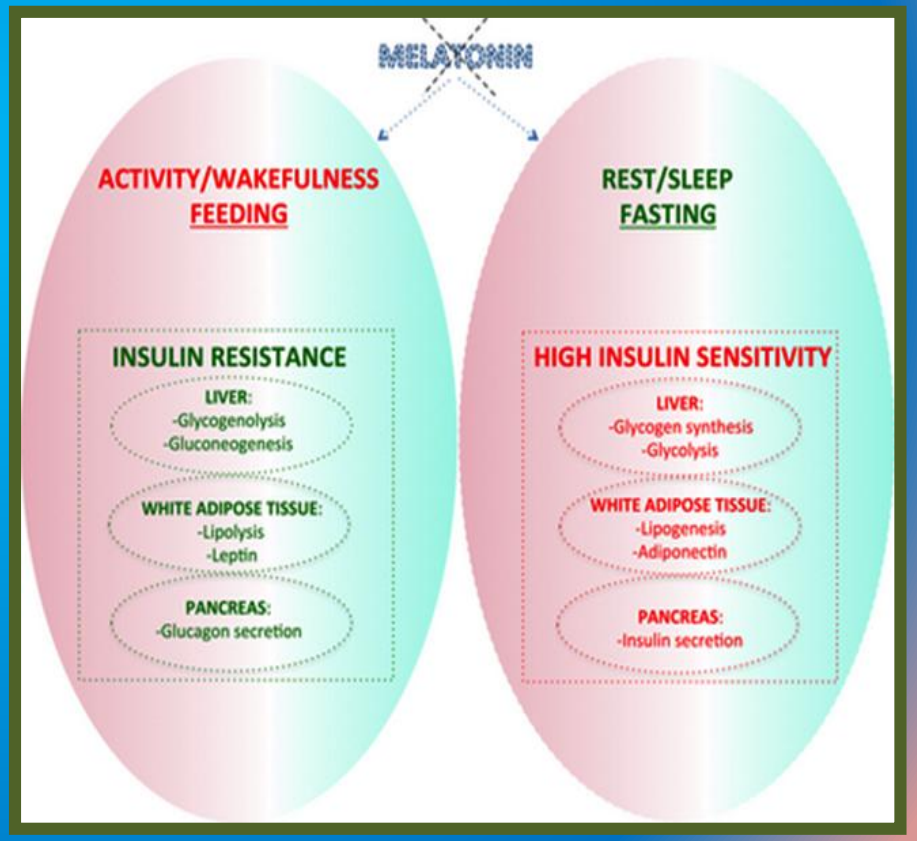
Built Environment




Metabolic disorders
OBESITY
Chronodisruption


ENERGY BALANCE
MELATONIN
Acquisition (Food intake)
MELATONIN
BODY WEIGHT
MELATONIN
Storage
Expenditure
MELATONIN


MELATONIN
ACTIVITY/WAKEFULNESS
FEEDING
ENERGY INTAKE AND STORAGE
HIGH INSULIN SENSITIVITY
LIVER:
-Glycogen synthesis
-Glycolysis
WHITE ADIPOSE TISSUE:
-Lipogenesis
-Adiponectin
PANCREAS:
-Insulin secretion
REST/SLEEP
FASTING
ENERGY EXPENDITURE
INSULIN RESISTANCE
LIVER:
-Glycogenolysis
-Gluconeogenesis
WHITE ADIPOSE TISSUE:
-Lipolysis
-Leptin
PANCREAS:
-Glucagon secretion


MELATONIN
ACTIVITY/WAKEFULNESS
FEEDING
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LIVER:
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REST/SLEEP
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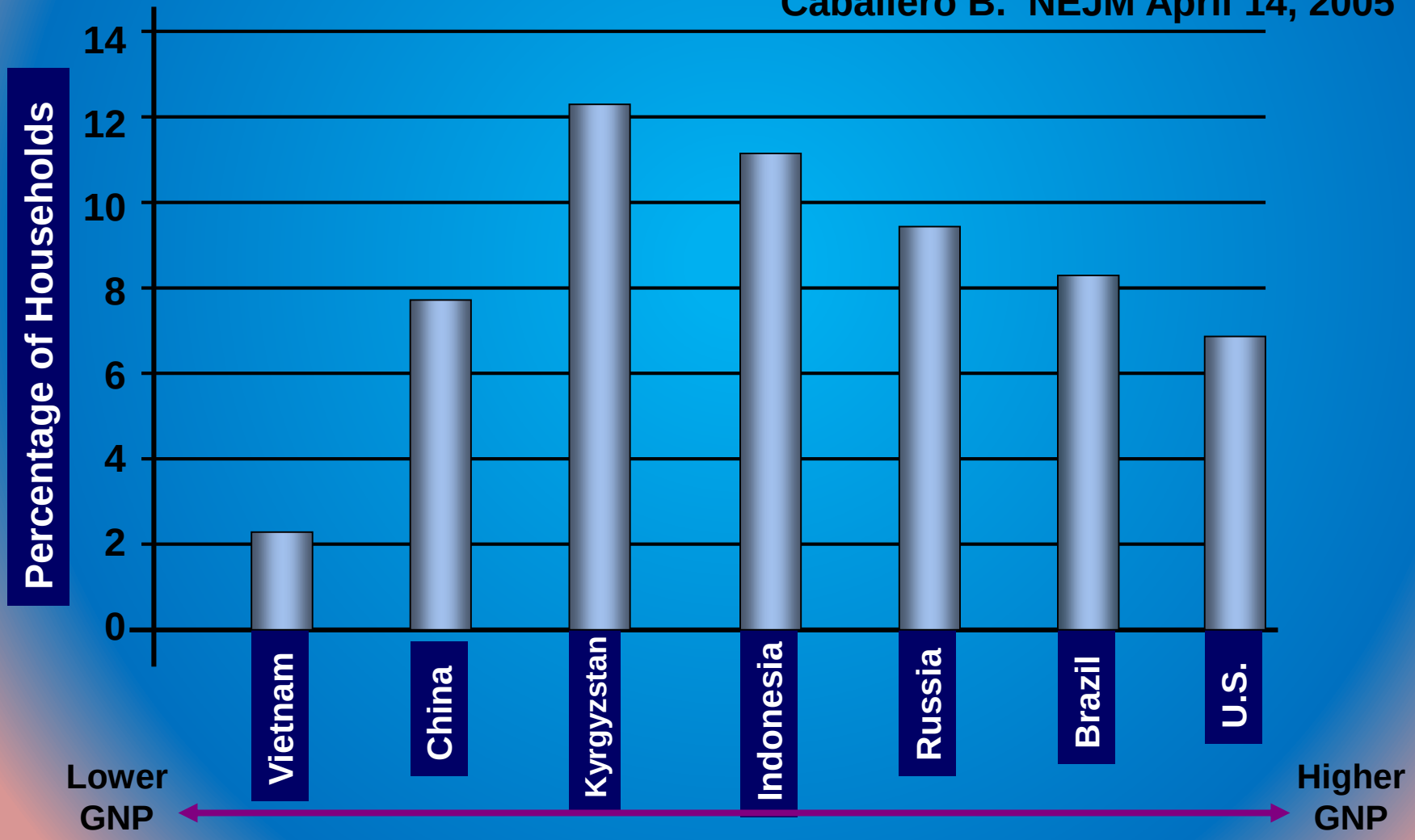
HIGH INSULIN SENSITIVITY
LIVER:
-Glycogen synthesis
-Glycolysis
WHITE ADIPOSE TISSUE:
-Lipogenesis
-Adiponectin
PANCREAS:
-Insulin secretion

Percentage of Households having both Underweight and Overweight members (7 Countries)

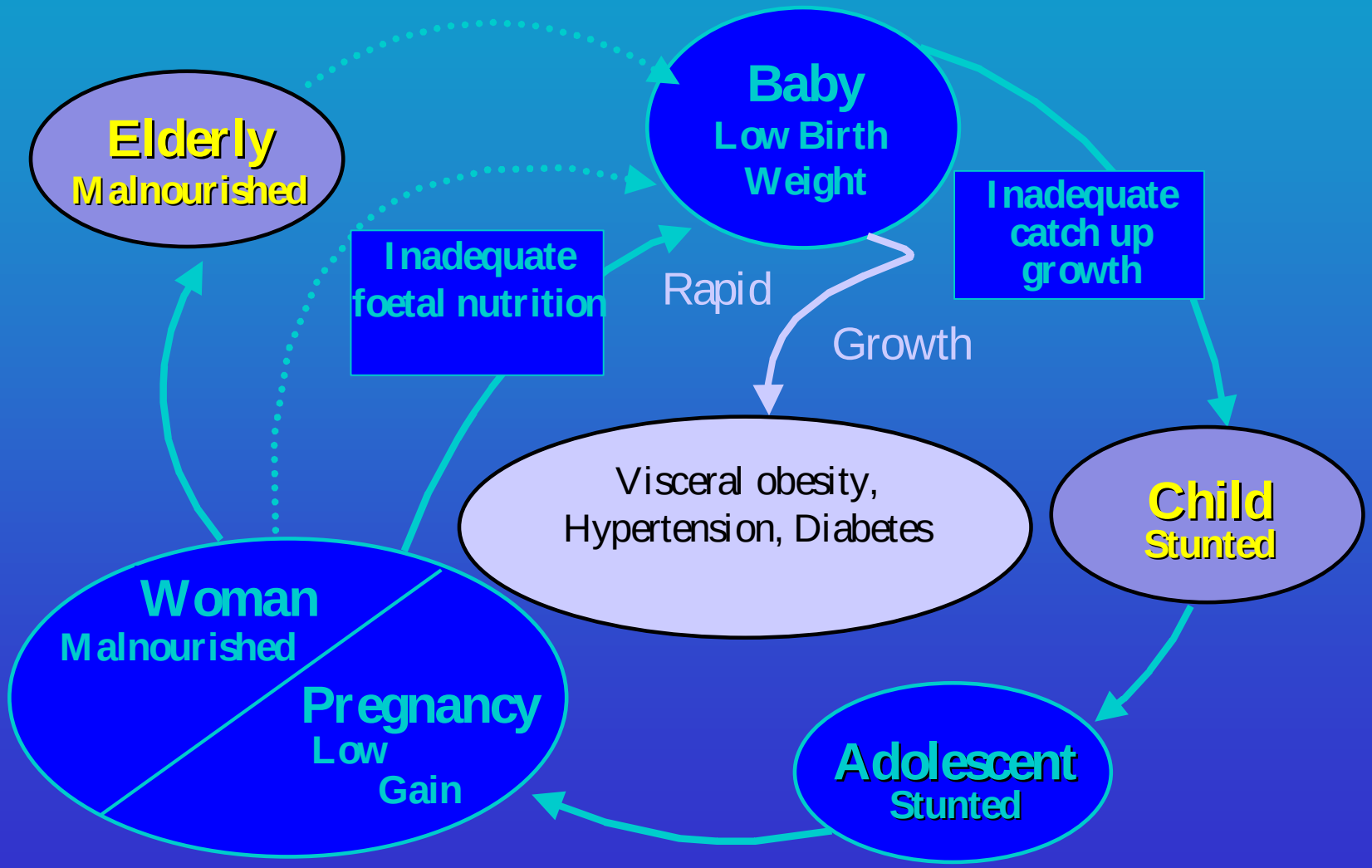
Caballero B. NEJM April 14, 2005



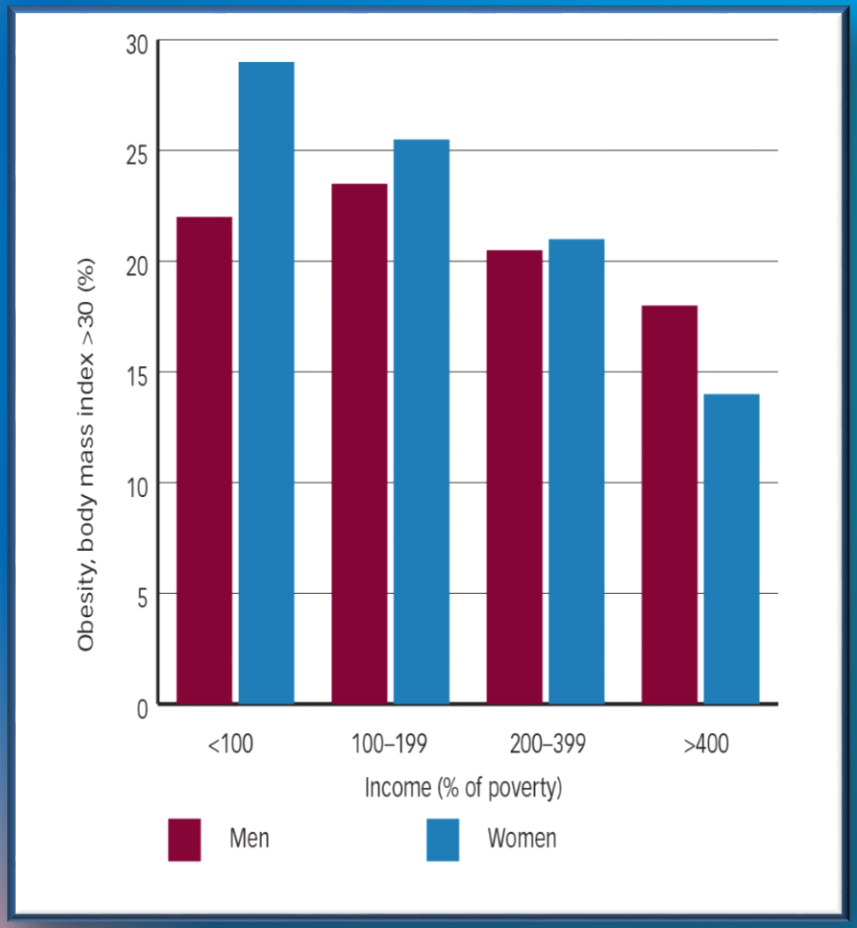
Infant weight and risk of IR and Diabetes

- Small for gestational age
- Large for gestational age
- Premature infants
- **Catch–up growth of low birth weight babies**

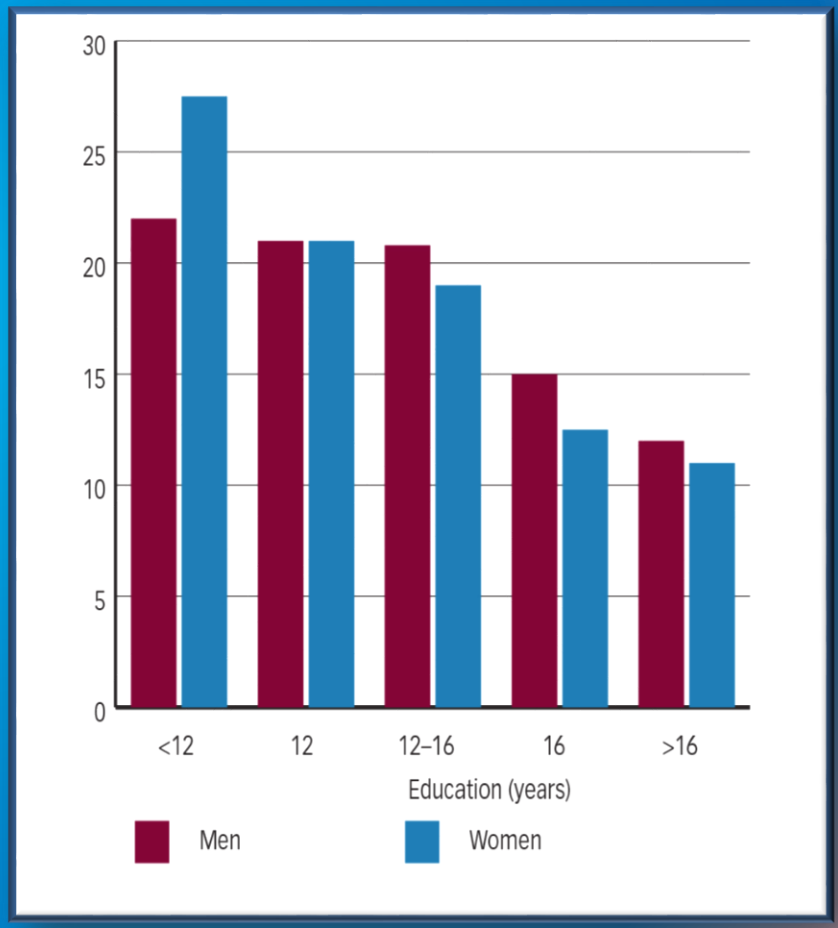
Barker hypothesis:
Lifecycle - the proposed causal links

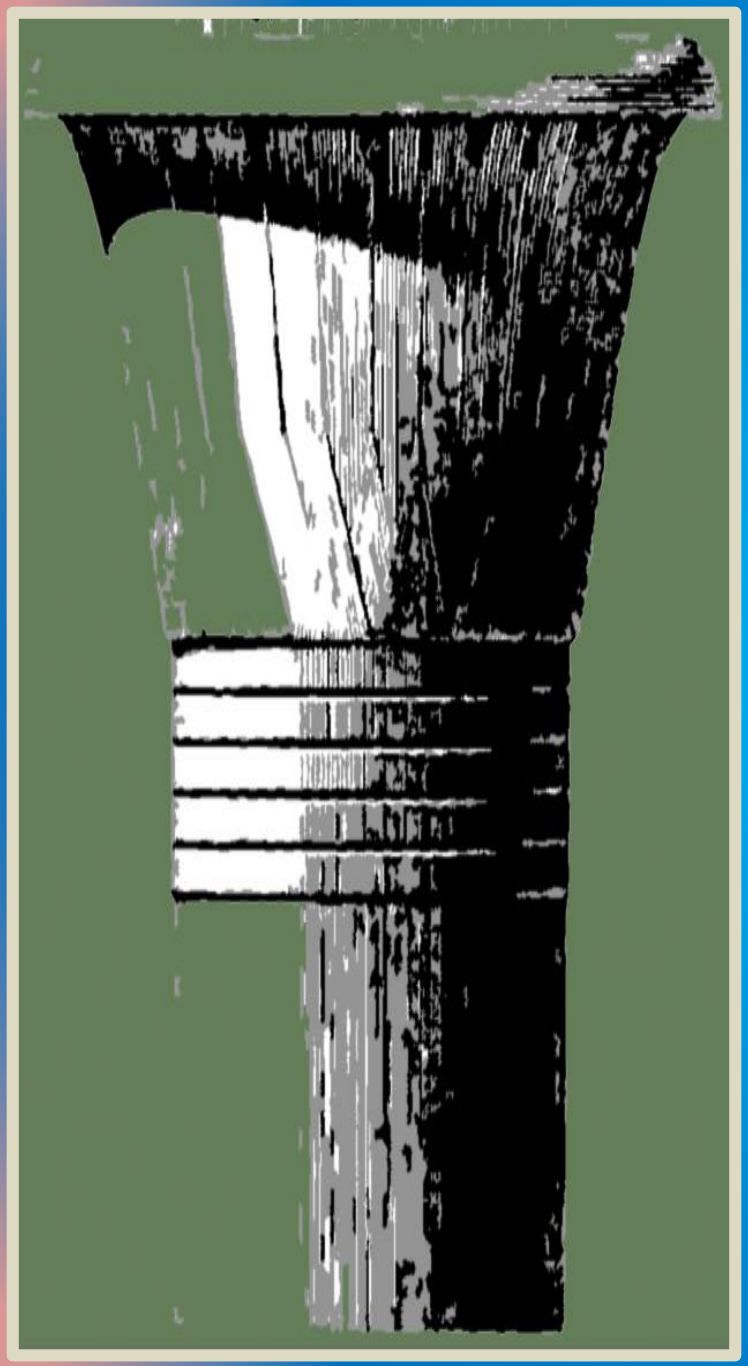


The Influence of Poverty on Obesity (Body Mass Index >30 kg/m^2)



The Influence of Education on Obesity (Body Mass Index >30 kg/m^2)





5. Obese Children

=

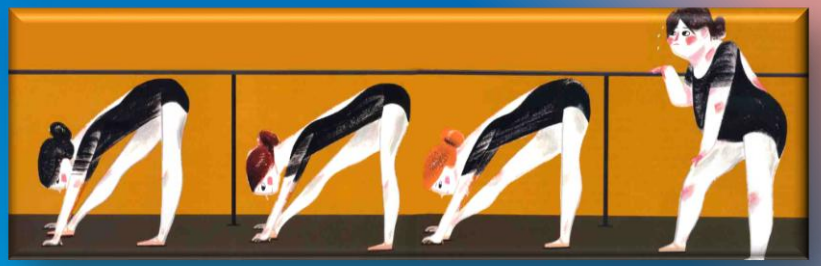
Obese Adults

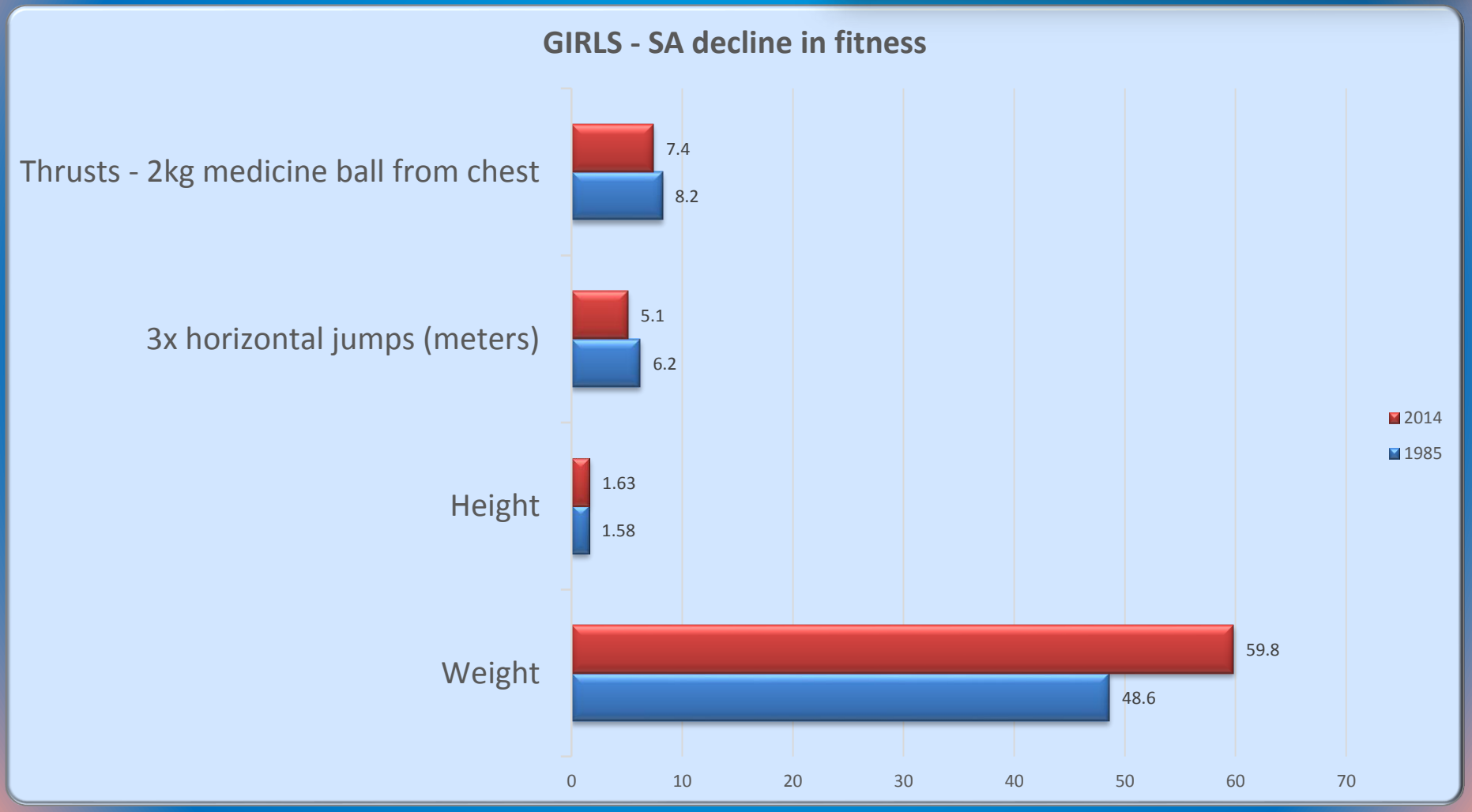
22% of children aged 1y-9y at BMI > 25

25-28% of adolescent girls obese

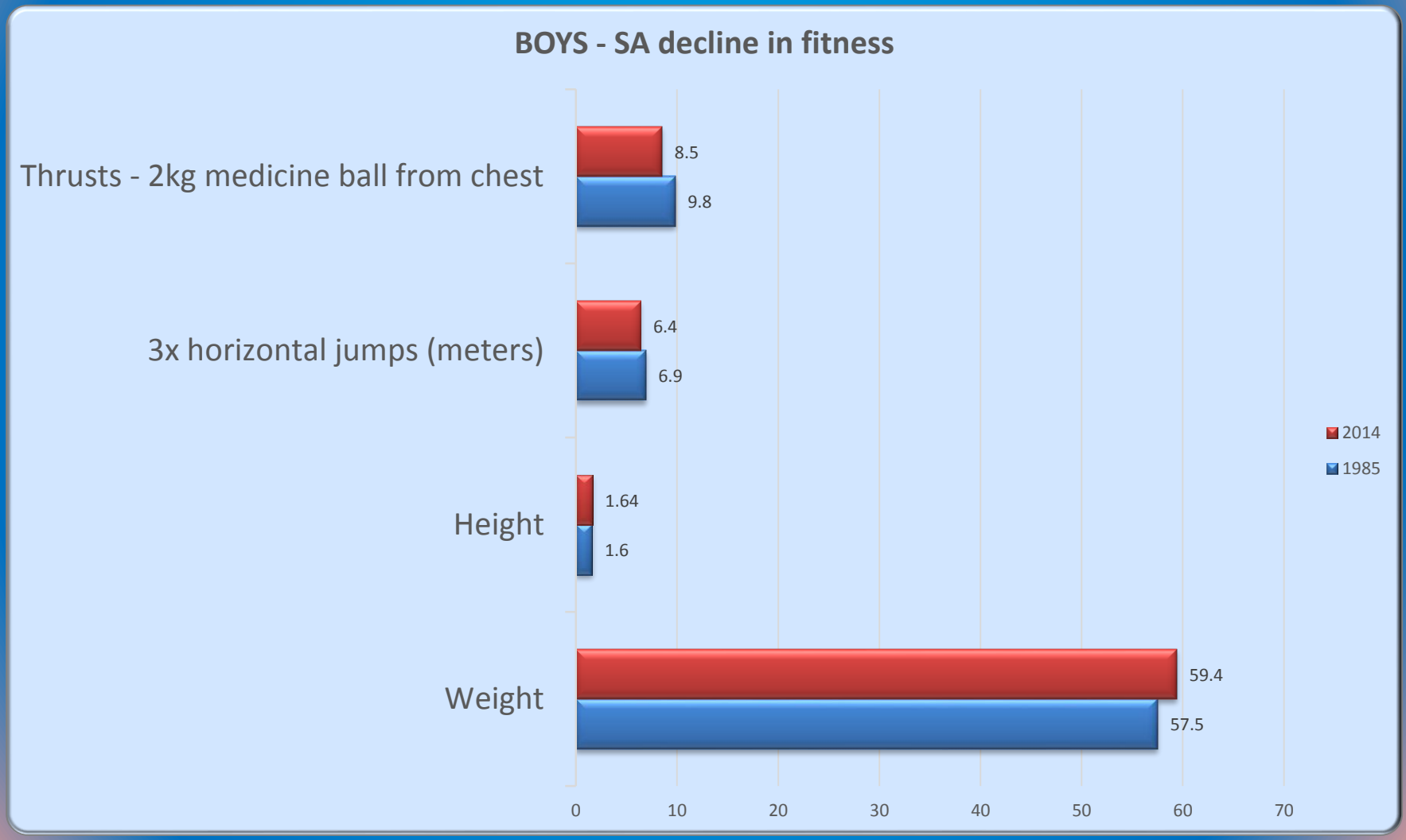
National Representative Study:	**Overweight:** **20.1 % Urban Children** **15.8 % Tribal children** **10.8% Children on farms**

GIRLS – SA decline in fitness





BOYS – SA decline in fitness



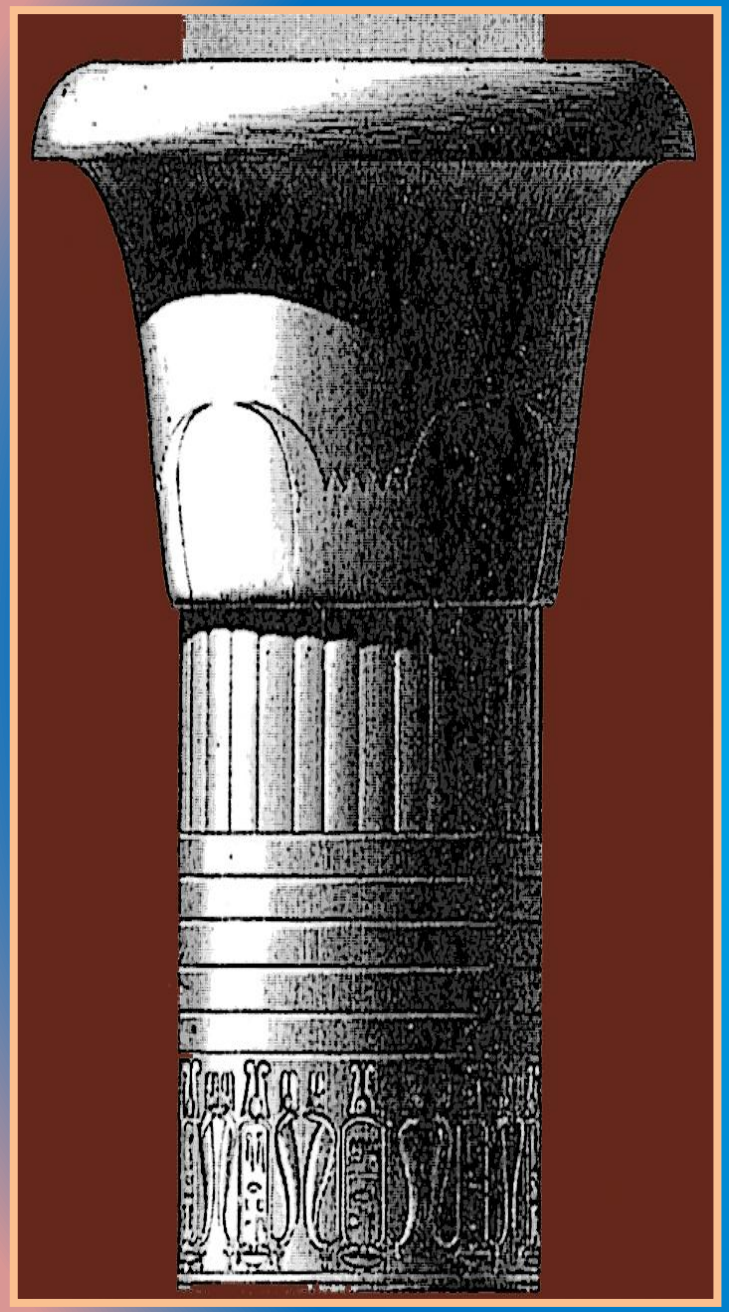
CHILDHOOD OBESITY ⟺ ADULT OBESITY

Childhood obesity is a strong predictor of adult obesity, particularly where both parents are obese

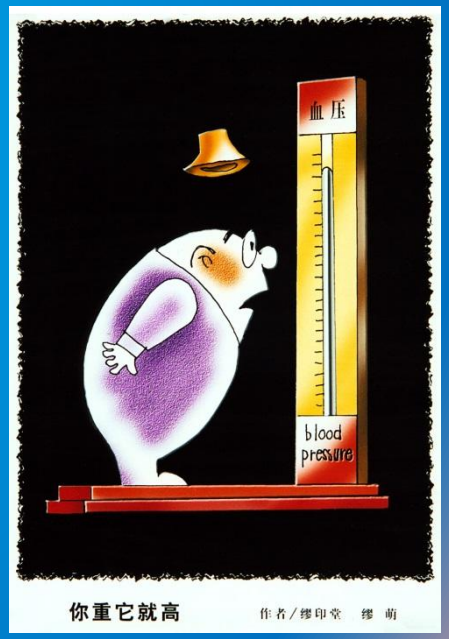
Prevalence of Metabolic Syndrome in adults:
Childhood obesity: 28%
Without childhood obesity: < 10%

Prevalence of adult obesity

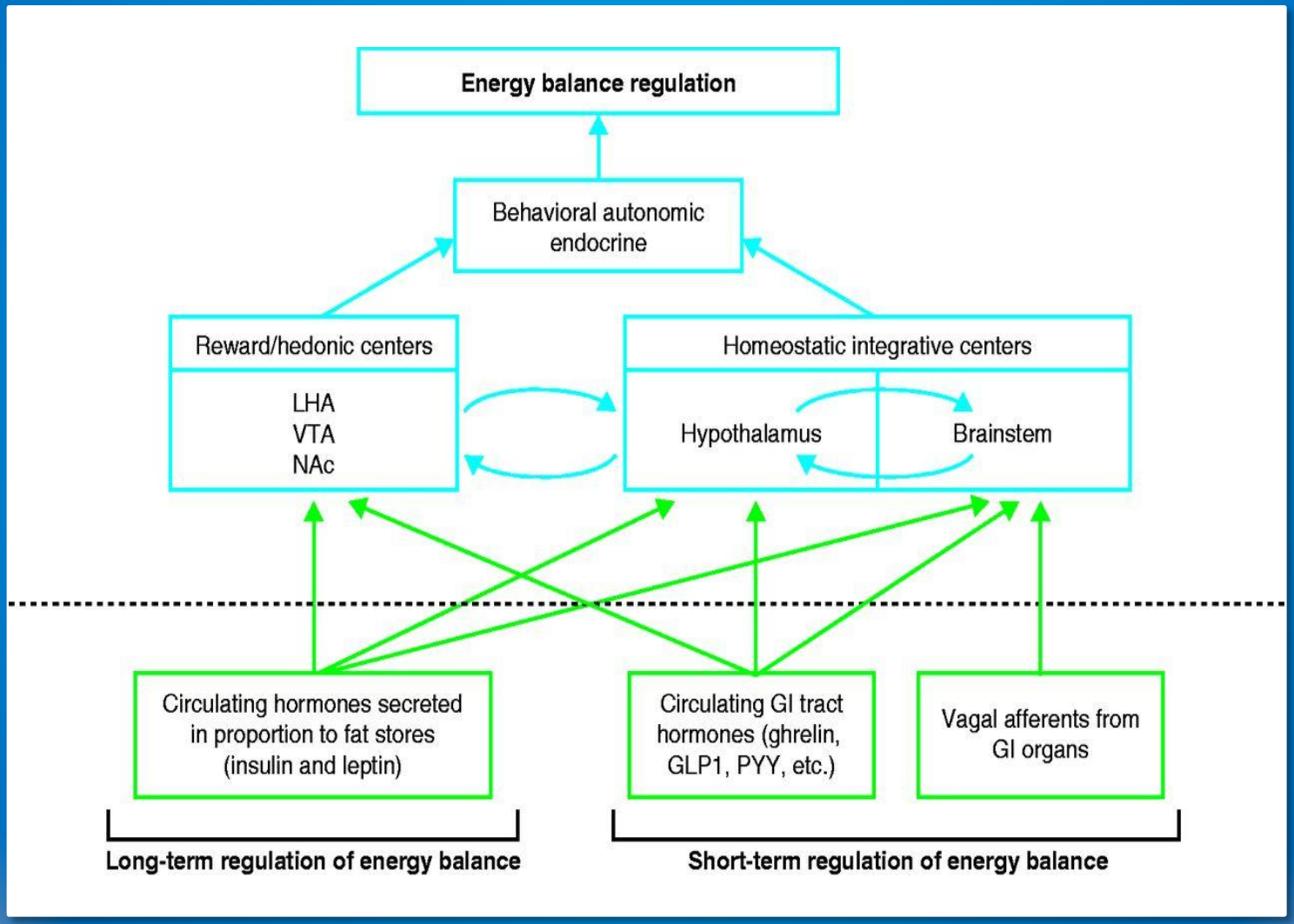
Age group	Neither parent obese	At least one parent obese
3-5 years	24%	62%
10-14 years	64%	79%

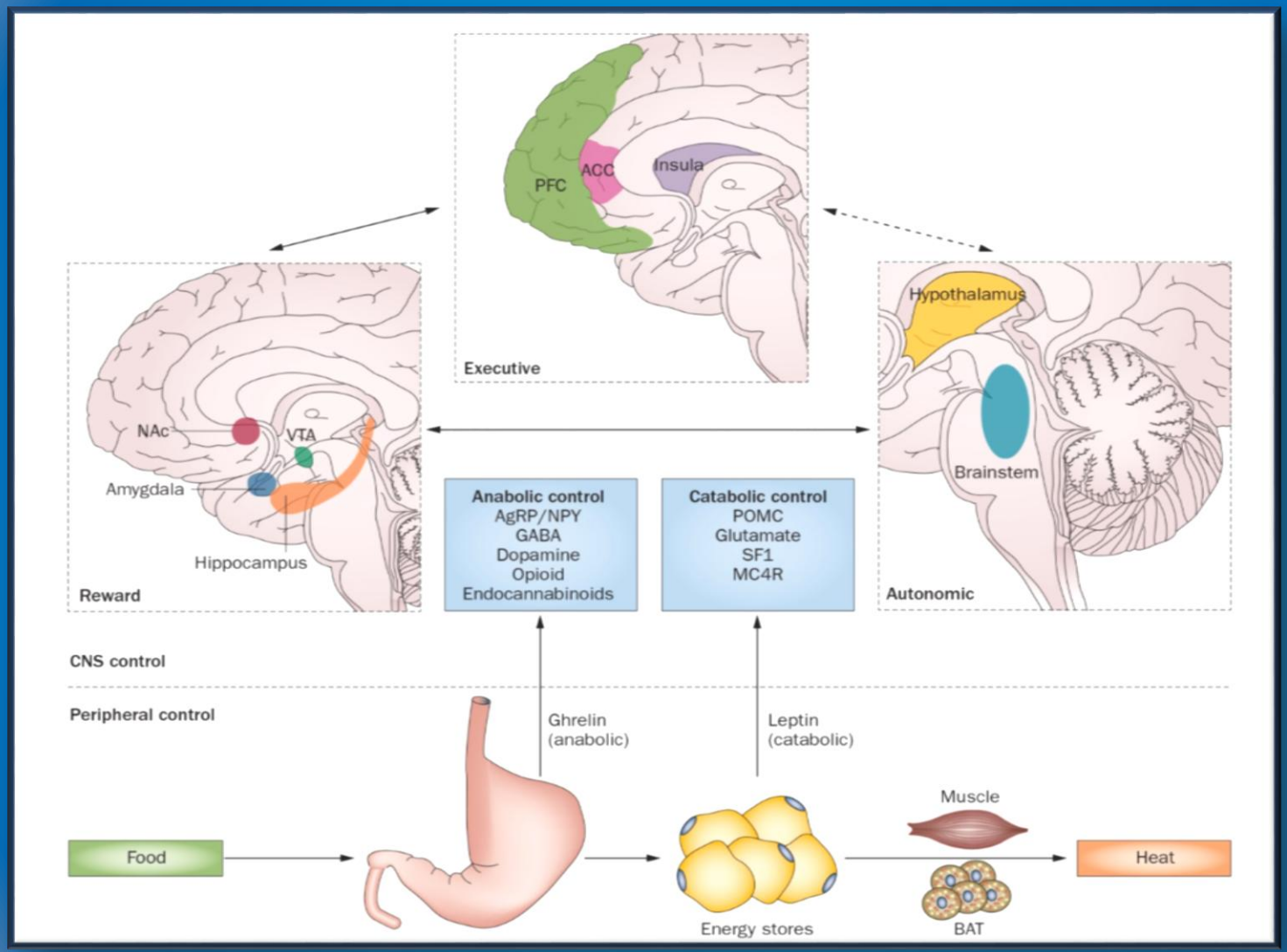


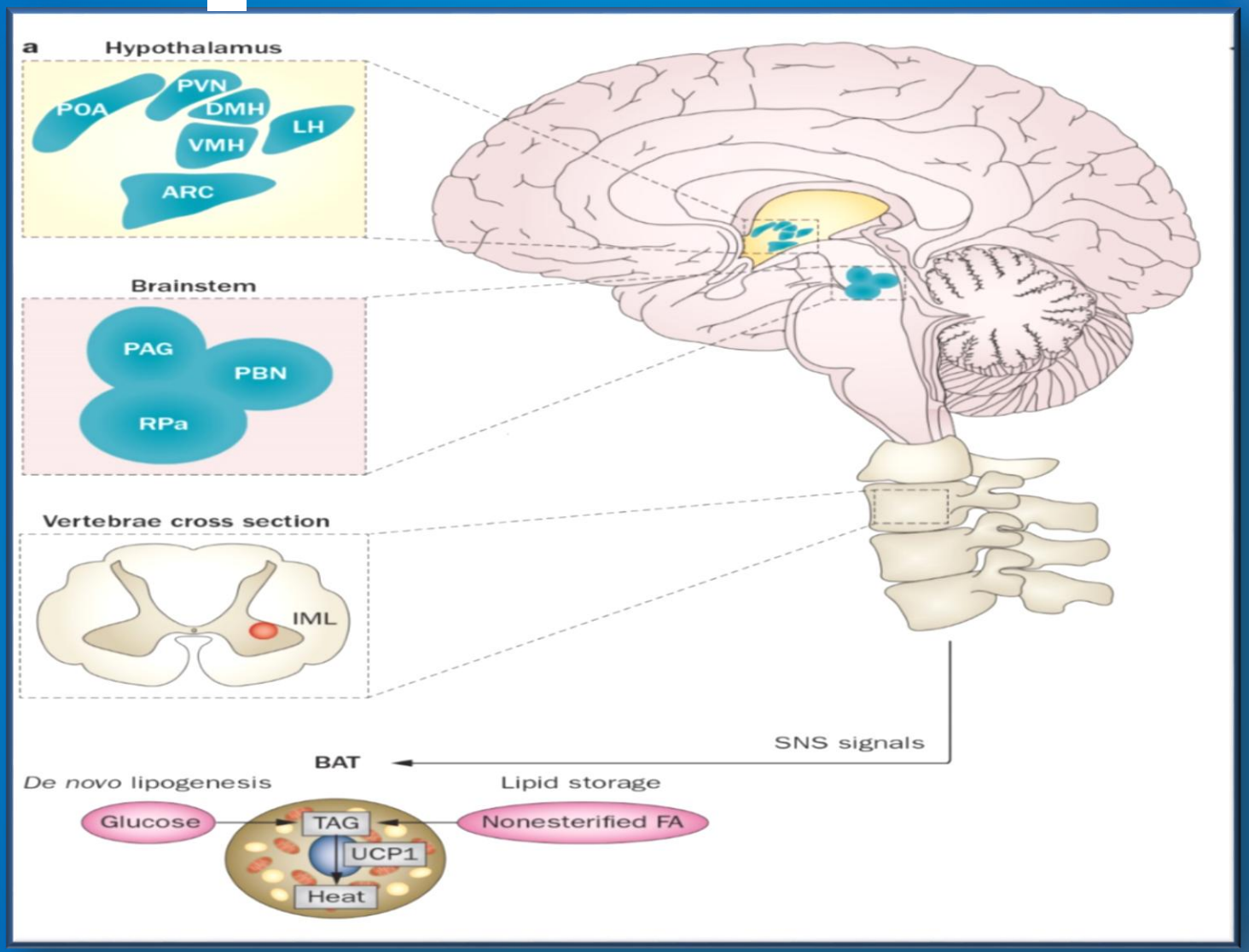
6. The complexity of appetite regulation

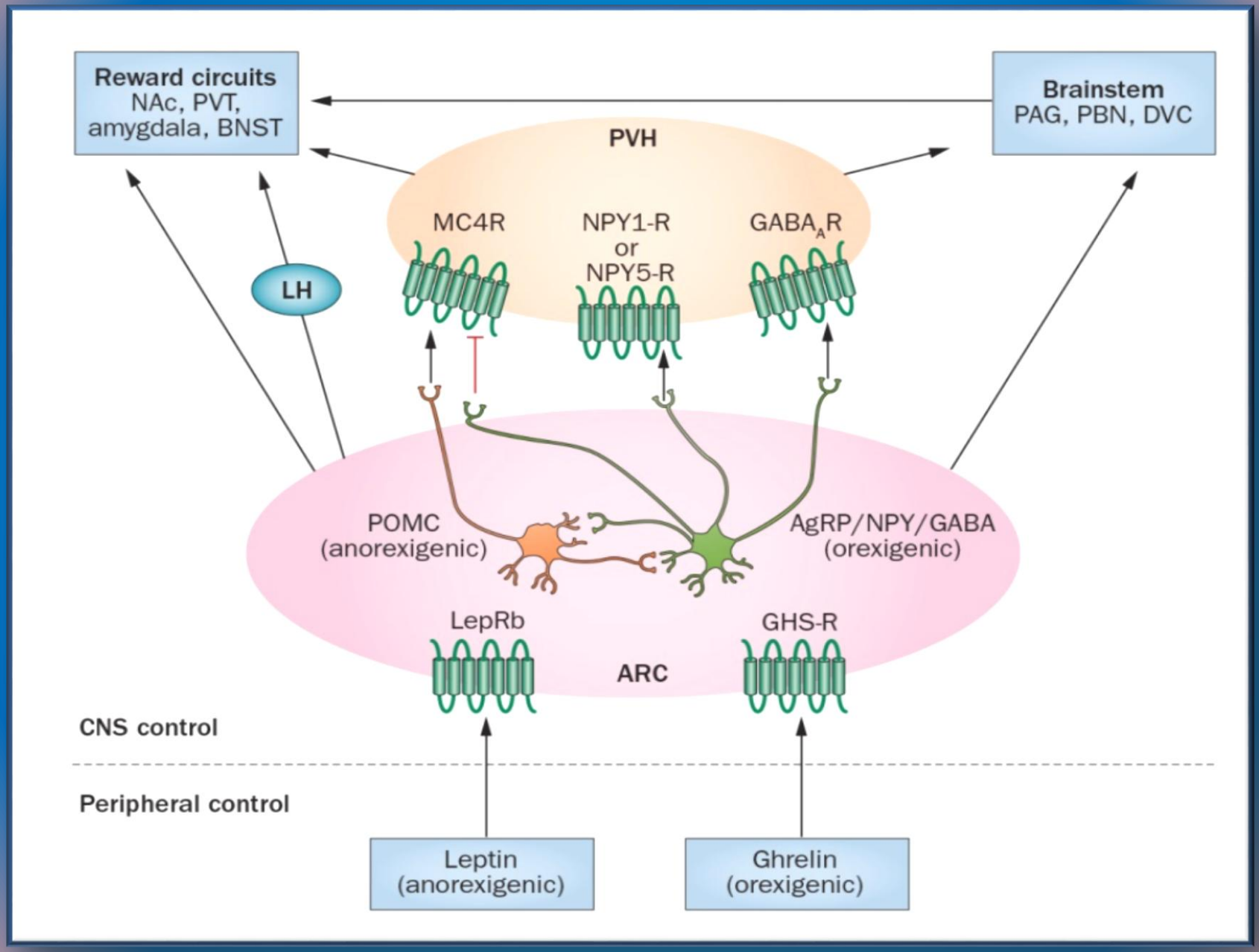


Schematic integration of the different levels of food intake and energy balance regulation




PFC
ACC
Insula
Executive
NAc
VTA
Amygdala
Hippocampus
Reward
Hypothalamus
Brainstem
Autonomic
Anabolic control
AgRP/NPY
GABA
Dopamine
Opioid
Endocannabinoids
Catabolic control
POMC
Glutamate
SF1
MC4R
CNS control
Peripheral control
Ghrelin
(anabolic)
Leptin
(catabolic)
Food
Energy stores
Muscle
BAT
Heat


a
Hypothalamus
PVN
POA
DMH
LH
VMH
ARC
Brainstem
PAG
PBN
RPa
Vertebrae cross section
IML
SNS signals
BAT
De novo lipogenesis
Lipid storage
Glucose
TAG
Nonesterified FA
UCP1
Heat


Reward circuits
NAc, PVT,
amygdala, BNST
Brainstem
PAG, PBN, DVC
PVH
MC4R
NPY1-R
or
NPY5-R
GABA$_A$R
LH
POMC
(anorexigenic)
AgRP/NPY/GABA
(orexigenic)
LepRb
GHS-R
ARC
CNS control
Peripheral control
Leptin
(anorexigenic)
Ghrelin
(orexigenic)

Key points

- Obesity results from genetic and environmental factors that interfere with the action of brain and peripheral networks involved in regulating energy balance
- The control of energy expenditure is, in part, exerted on the activity of brown adipose tissue, which might have a considerable thermogenic effect in the body
- The controls of energy intake and expenditure are insured by interrelated cortical executive, reward and autonomic circuits in the brain
- The dopamine mesolimbic circuit and the opioid, endocannabinoid and melanocortin systems are key central nervous system elements in energy homeostasis
- Leptin and ghrelin are peripheral homeostatic hormones that signal to the brain to provide information on energy balance and nutritional status

7. The difficulty in maintaining weight loss

The great thing in this world
is not so much where we are,
but in what direction we are moving

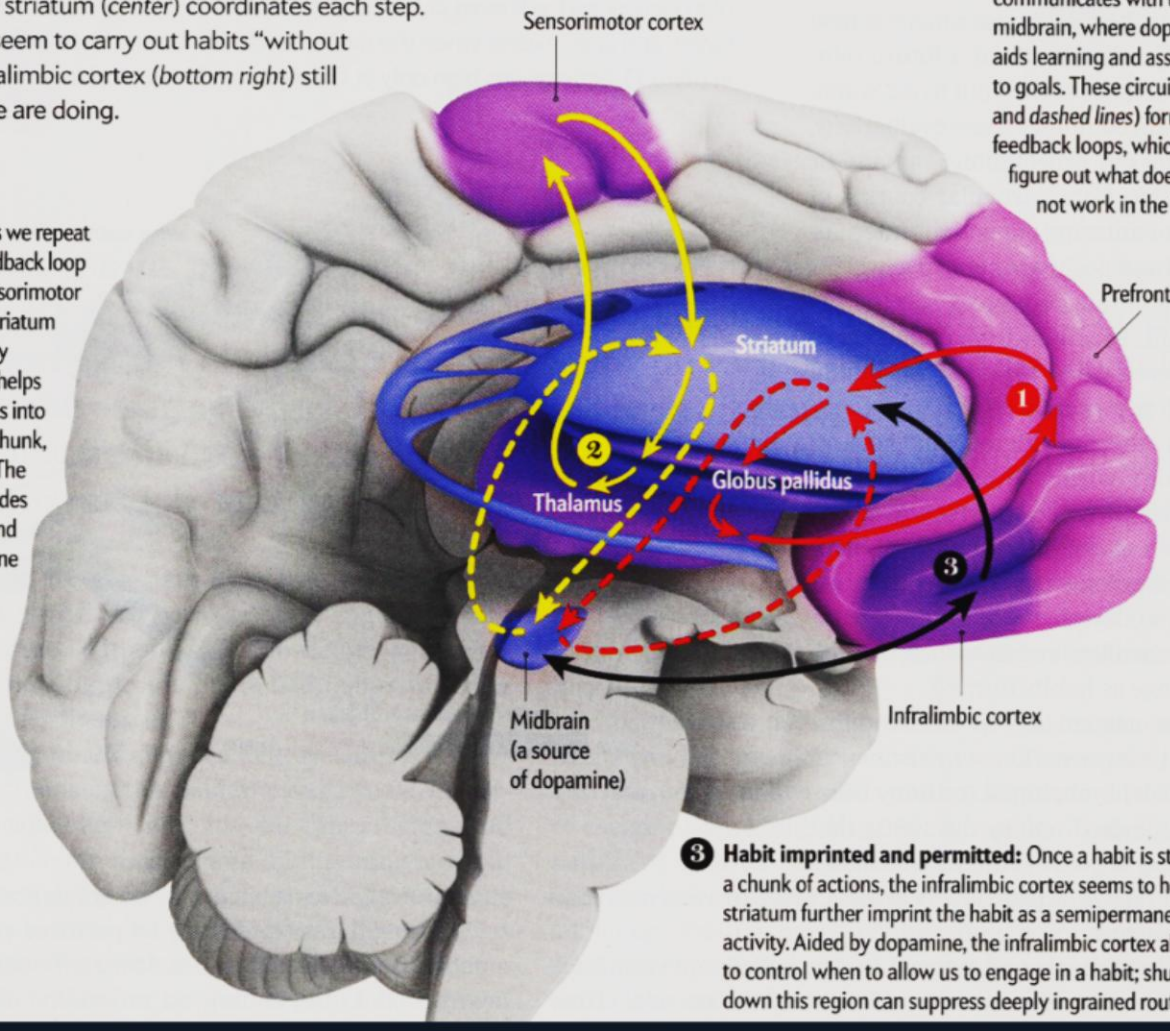
Oliver Wendell Holmes (1809-1894)

How Habits Form

We use three steps to learn and lock in habits: explore a new behavior, form a habit, then imprint it into the brain (*colored numbers*). Although scientists have not refined all the details, the striatum (*center*) coordinates each step. Even though we seem to carry out habits "without thinking," the infralimbic cortex (*bottom right*) still monitors what we are doing.

1 New behavior explored: The prefrontal cortex communicates with the striatum, and the striatum communicates with the midbrain, where dopamine aids learning and assigns value to goals. These circuits (*solid* and *dashed lines*) form positive feedback loops, which help us figure out what does and does not work in the behavior.

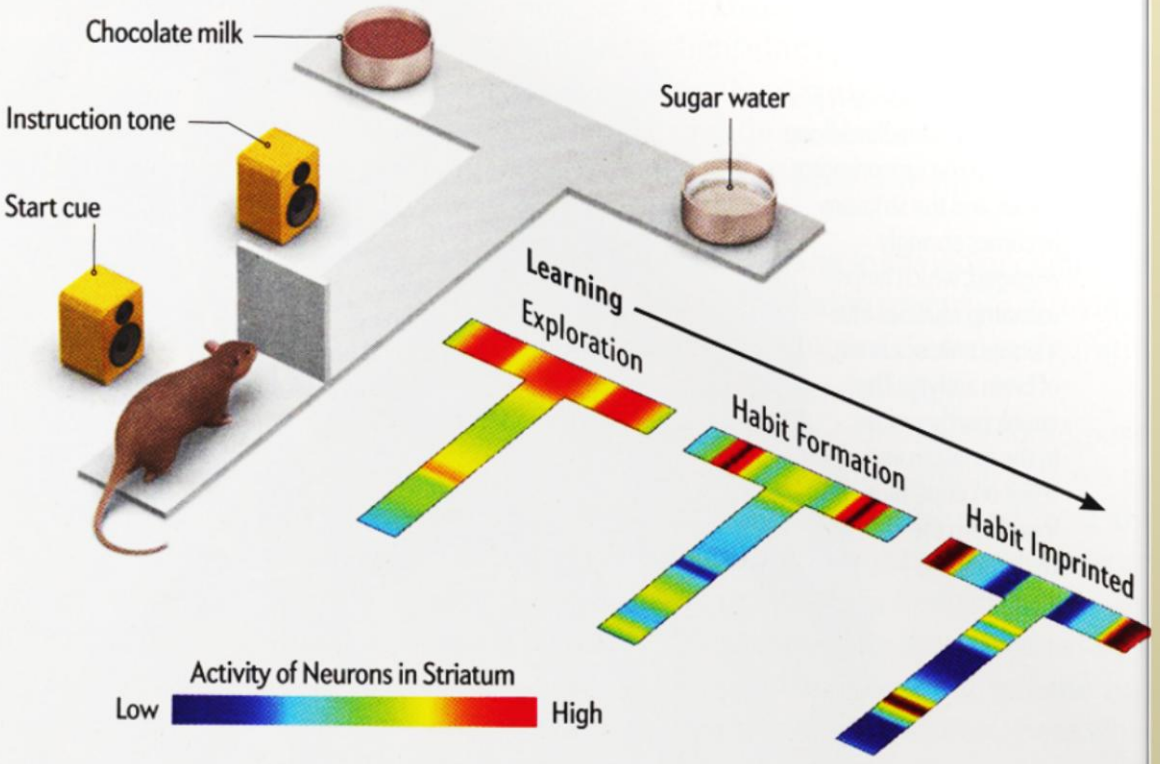
2 Habit forms: As we repeat a behavior, a feedback loop between the sensorimotor cortex and the striatum becomes strongly engaged, which helps us stamp routines into a single unit, or chunk, of brain activity. The chunk partly resides in the striatum and relies on dopamine input from the midbrain.



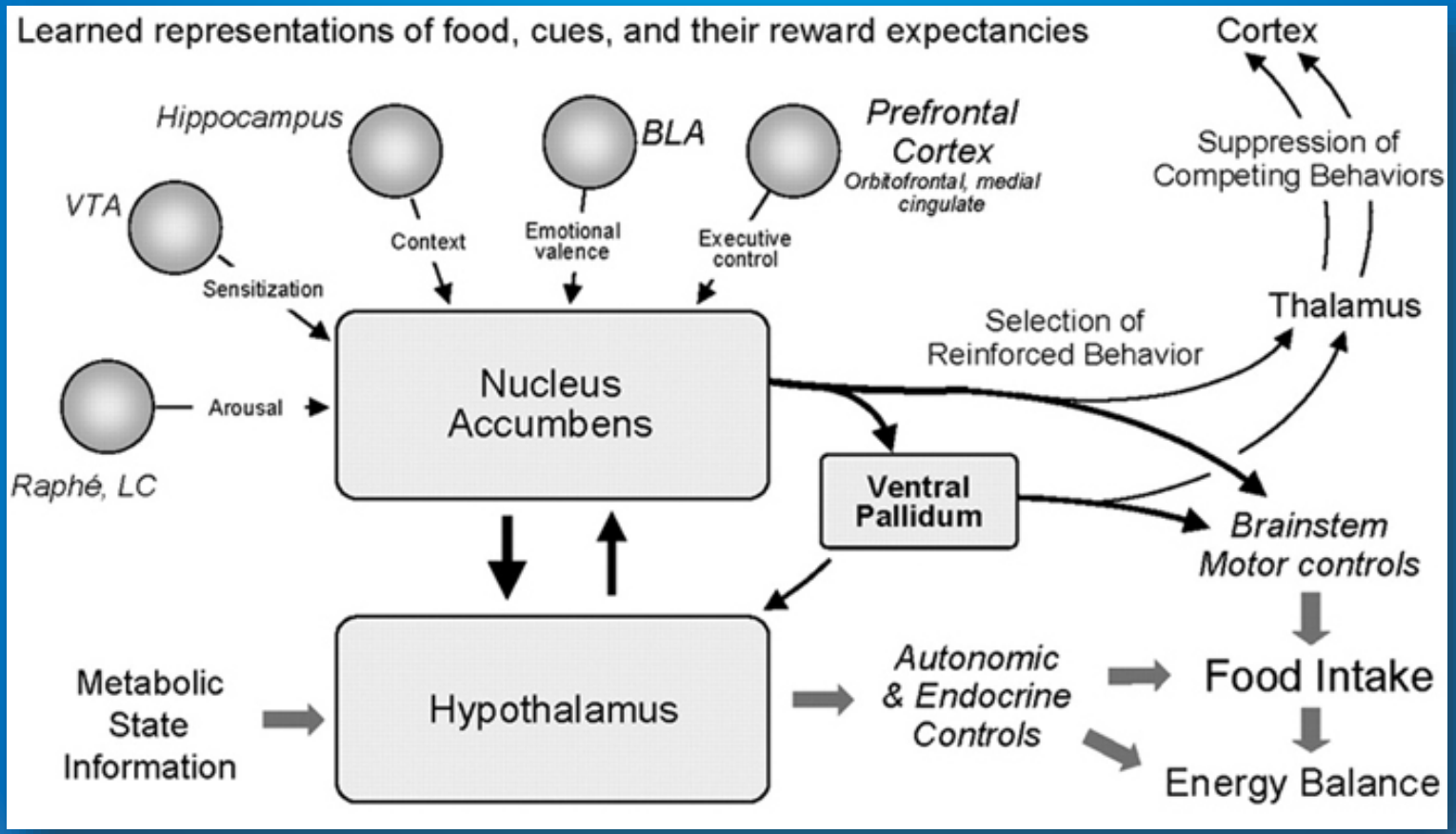
3 Habit imprinted and permitted: Once a habit is stored as a chunk of actions, the infralimbic cortex seems to help the striatum further imprint the habit as a semipermanent brain activity. Aided by dopamine, the infralimbic cortex also seems to control when to allow us to engage in a habit; shutting down this region can suppress deeply ingrained routines.

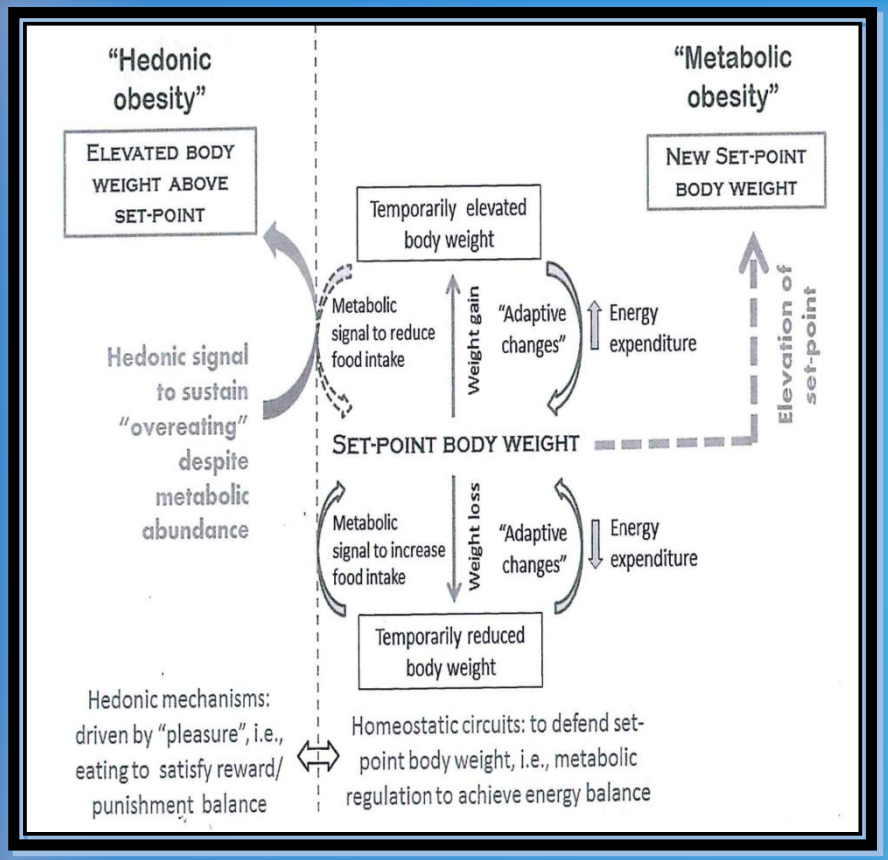
Acting without Thinking

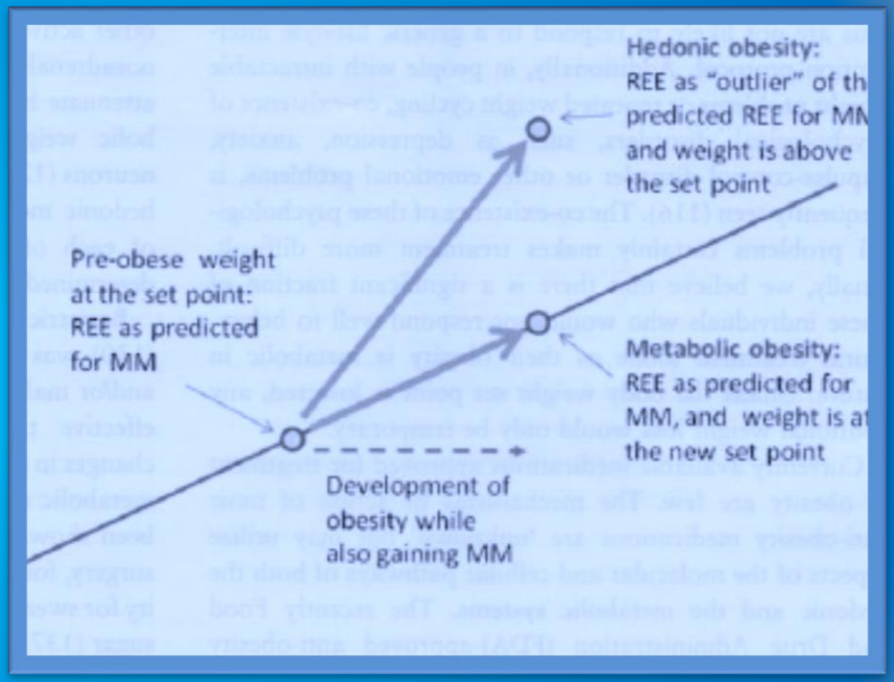
Tests on rats revealed that the brain treats a habit as a single unit of behavior. The rats learned to run down a T-maze and turn left or right toward a reward, depending on an instruction sound. During early runs (*first colored T*), activity in the brain's striatum was high (*yellow* and *red*) most of the time. As a habit formed (*second T*), activity quieted (*green* and *blue*) except when the rat had to decide to turn or to drink. Once a habit set in (*third T*), activity was high only at the start and finish, marking one unit of behavior.



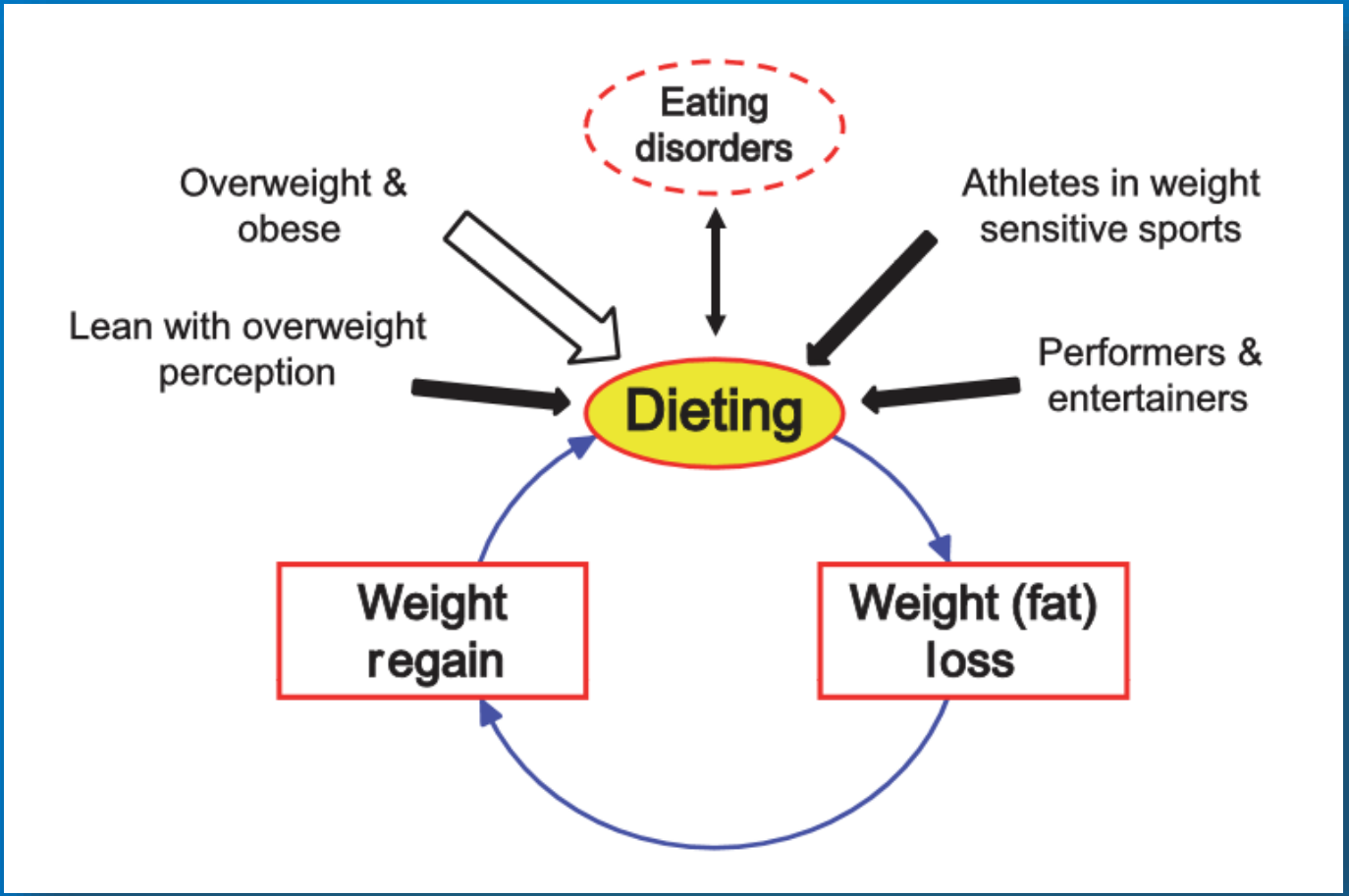
Homeostatic and Non-homeostatic Pathways Involved in the Control of Food Intake and Energy Balance



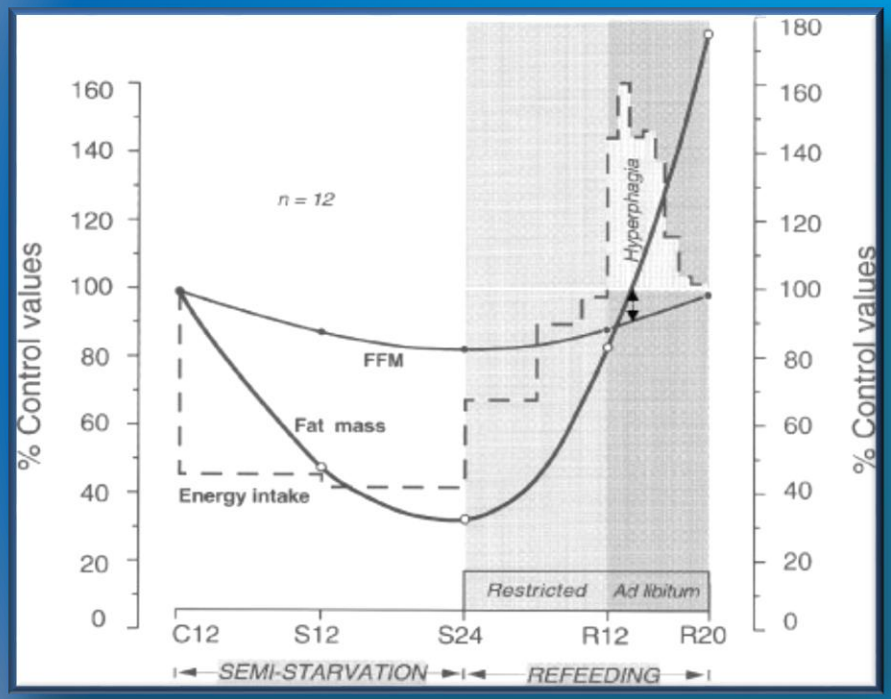

"Hedonic obesity"
"Metabolic obesity"
ELEVATED BODY WEIGHT ABOVE SET-POINT
NEW SET-POINT BODY WEIGHT
Temporarily elevated body weight
Metabolic signal to reduce food intake
Weight gain
"Adaptive changes"
Energy expenditure
Hedonic signal to sustain "overeating" despite metabolic abundance
Elevation of set-point
SET-POINT BODY WEIGHT
Metabolic signal to increase food intake
Weight loss
"Adaptive changes"
Energy expenditure
Temporarily reduced body weight
Hedonic mechanisms: driven by "pleasure", i.e., eating to satisfy reward/ punishment balance
Homeostatic circuits: to defend set-point body weight, i.e., metabolic regulation to achieve energy balance

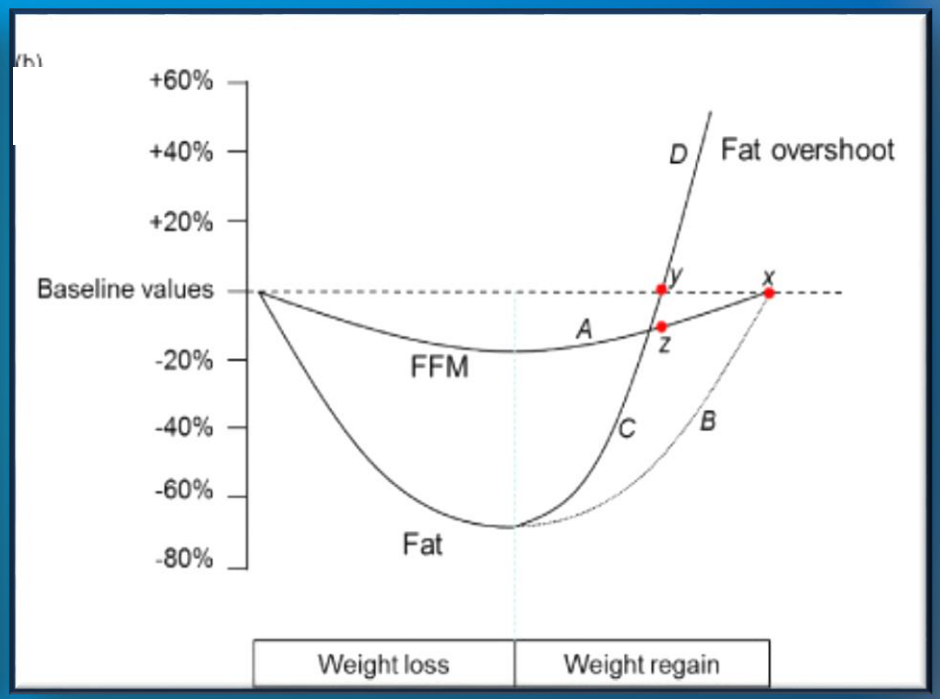

Hedonic obesity:
REE as "outlier" of th
predicted REE for MM
and weight is above
the set point
Pre-obese weight
at the set point:
REE as predicted
for MM
Metabolic obesity:
REE as predicted for
MM, and weight is a
the new set point
Development of
obesity while
also gaining MM

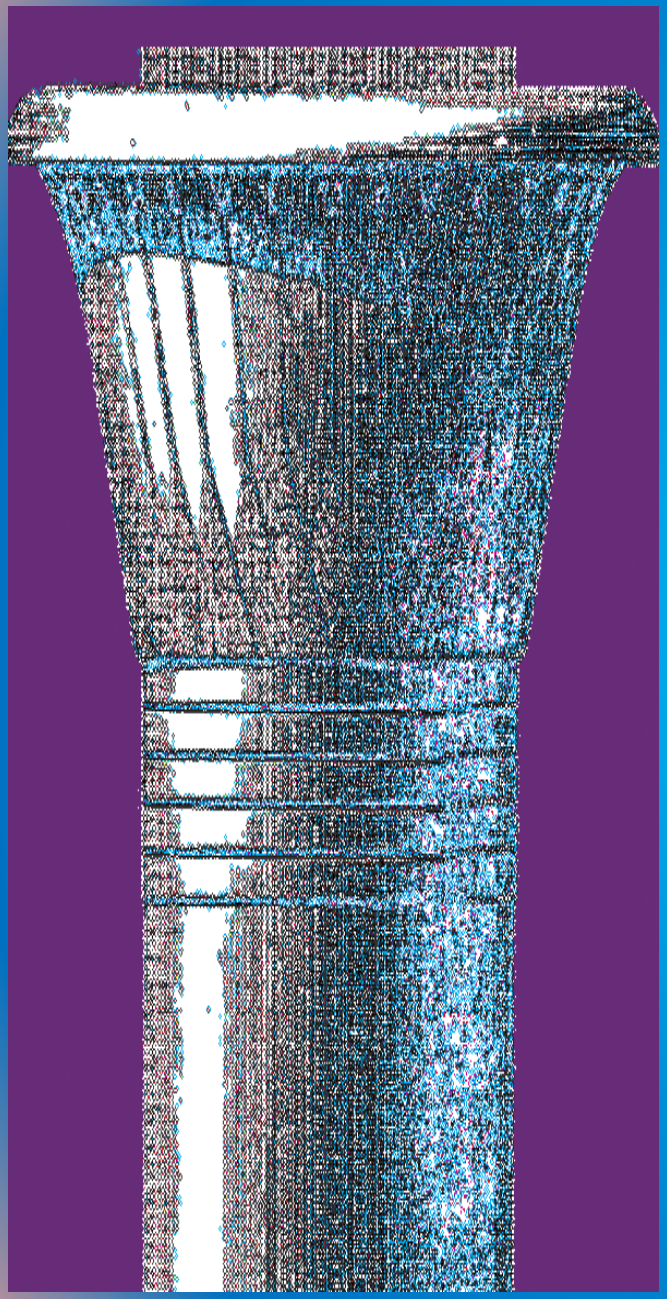
Dieting and weight cycling as risk factors for cardiometabolic diseases: who is really at risk?



How dieting makes the lean fatter: from a perspective of body composition autoregulation through adipostats and proteinstats awaiting discovery





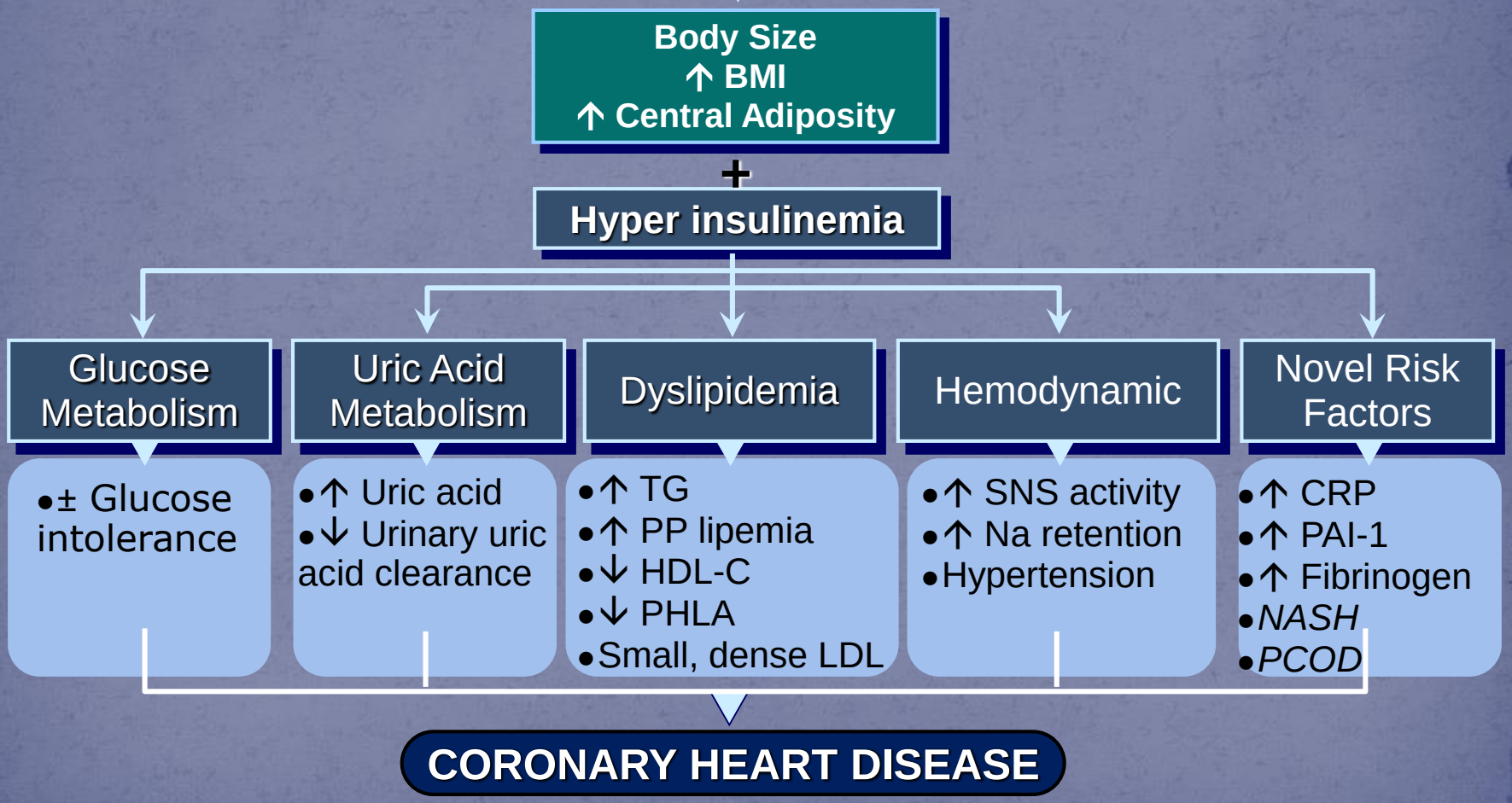


8. Understanding

Insulin

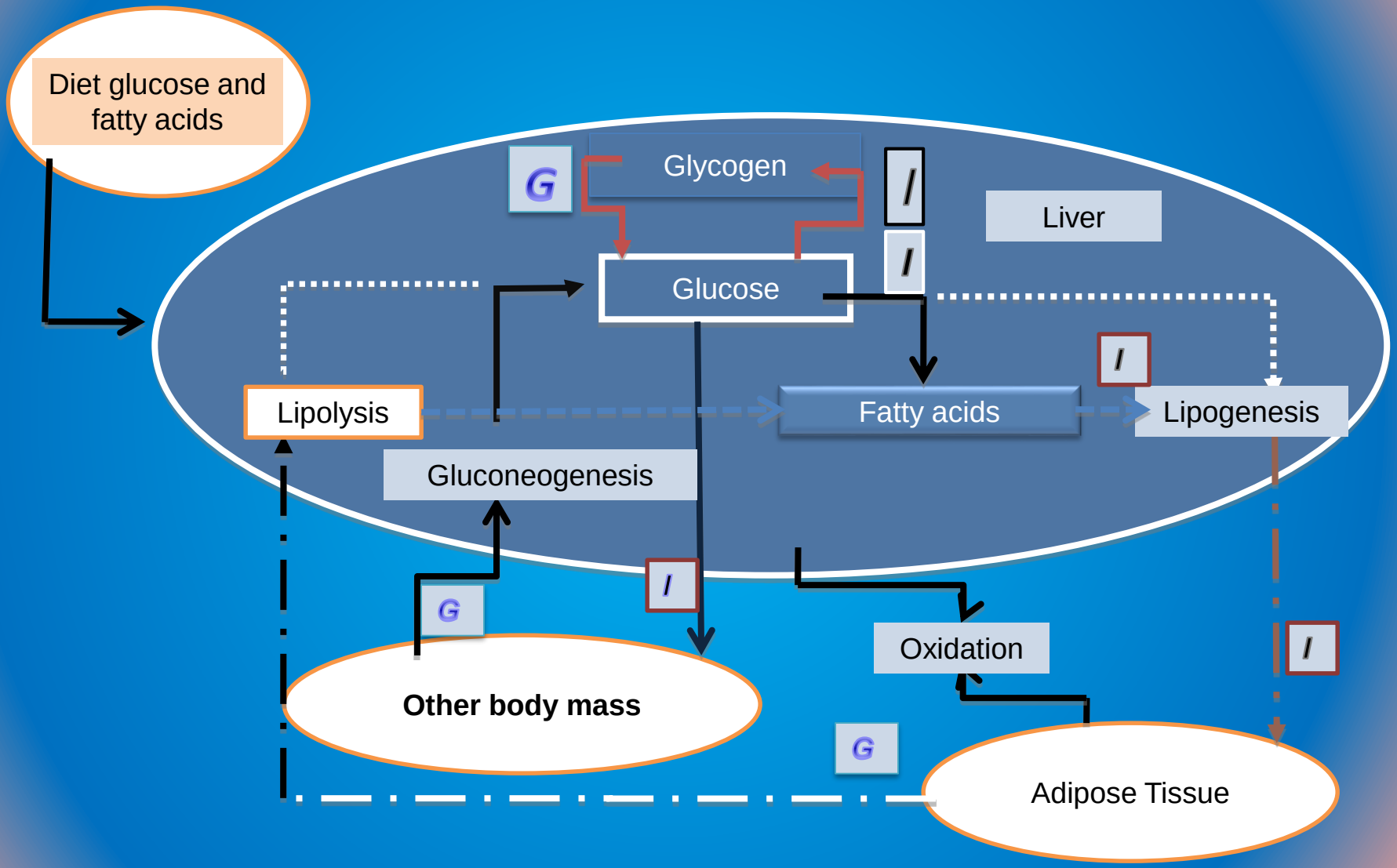
resistance

Metabolic Syndrome



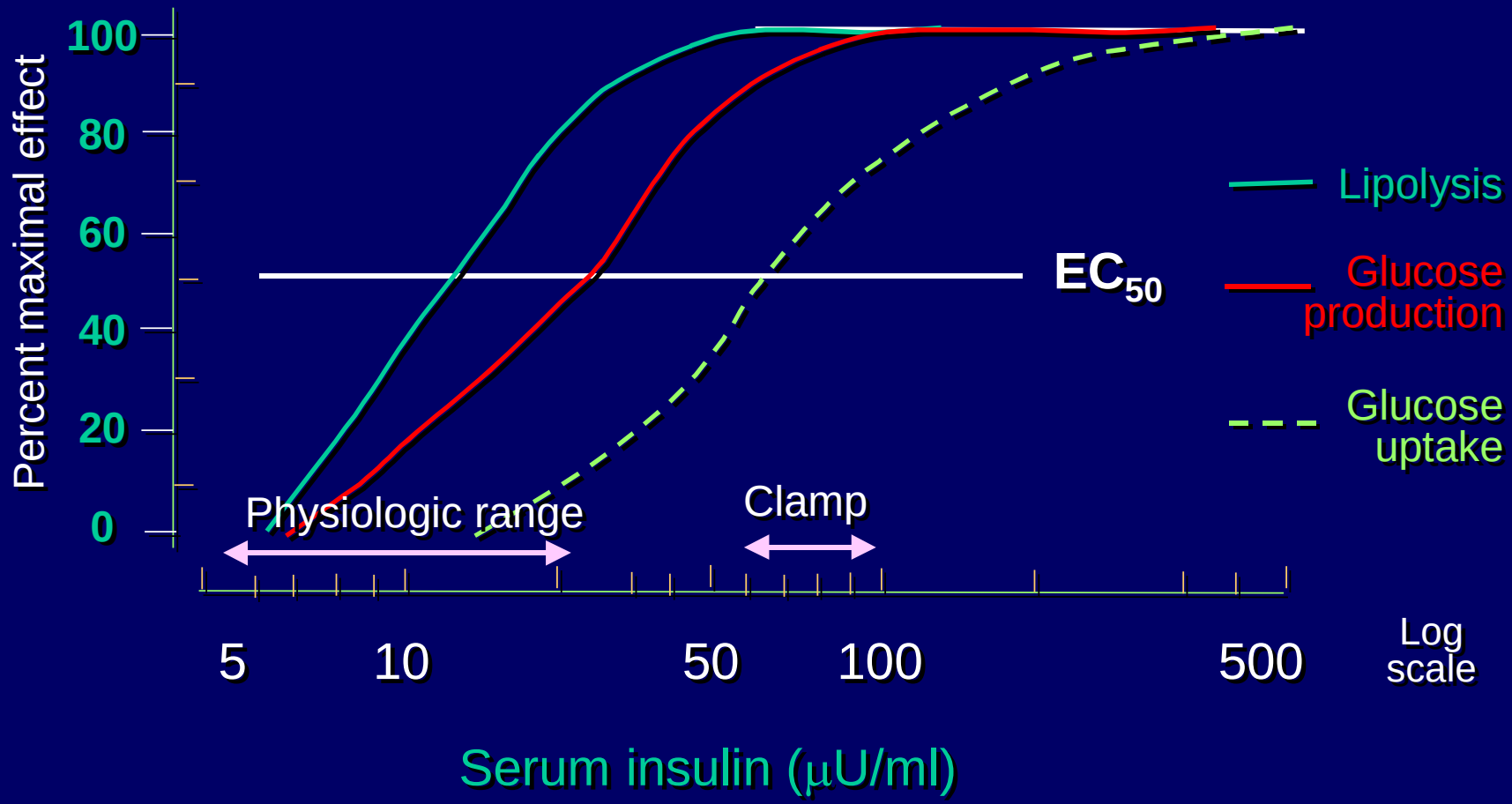
Adapted from Reaven G. Drugs. 1999;58 (suppl):19-20

MAJOR METABOLIC PATHWAYS INVOLVED IN GLUCOSE HOMEOSTASIS

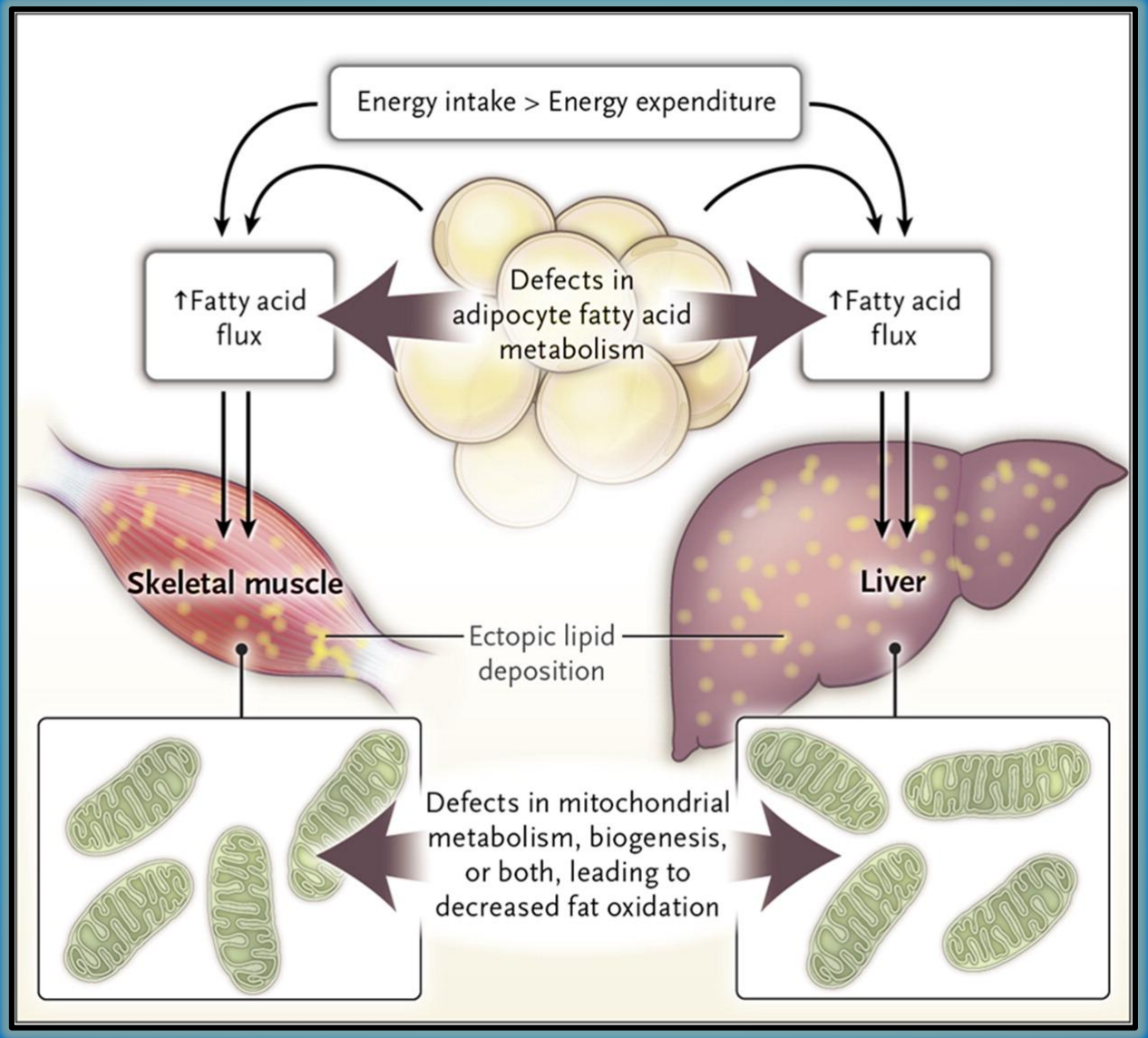


Smith JMD, Maas JA, Garnsworthy PC, Owen MR, Coombes S et al. Mathematical modeling of glucose homeostasis and its relationship with energy balance and body fat. Obesity 17 (4) April 2009;633

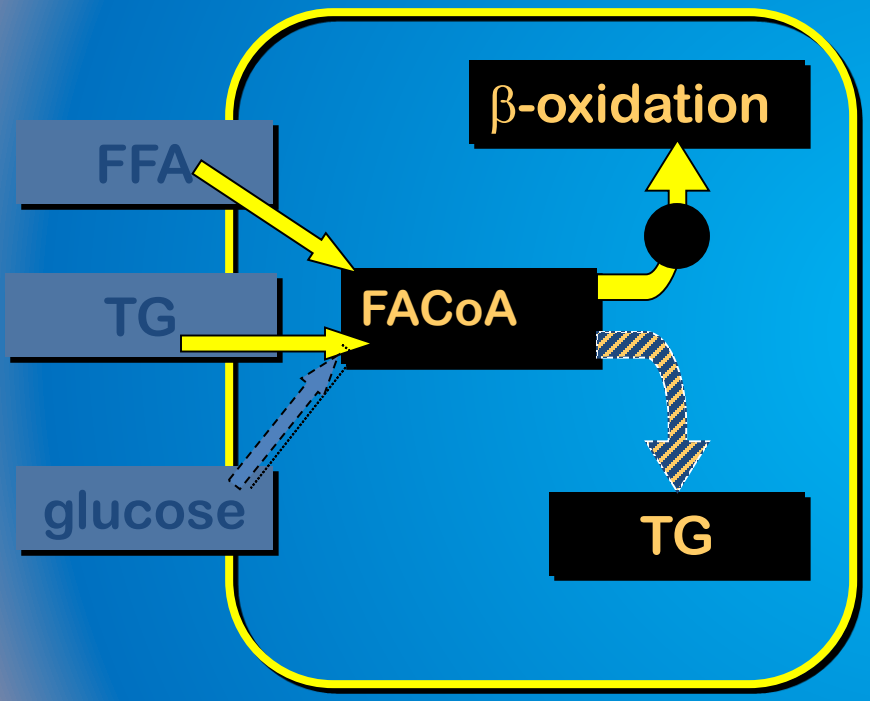
INSULIN DOSE RESPONSE CURVES FOR STIMULATION OF GLUCOSE UPTAKE, SUPPRESSION OF GLUCOSE PRODUCTION AND SUPPRESSION OF LIPOLYSIS



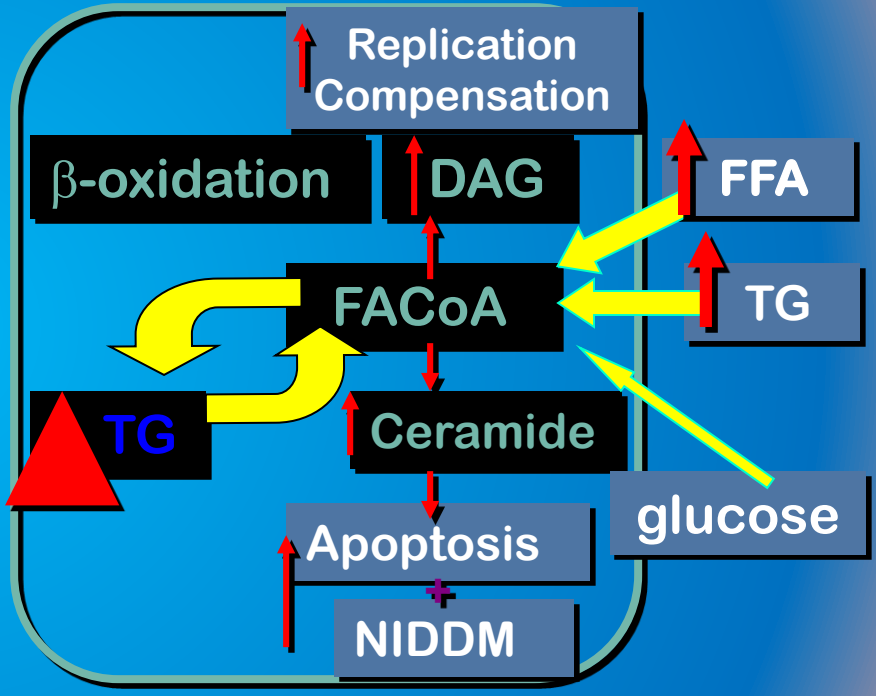
Mechanisms of Increased Ectopic Lipid Deposition in the Liver and Skeletal Muscle



Normal intracellular FA homeostasis



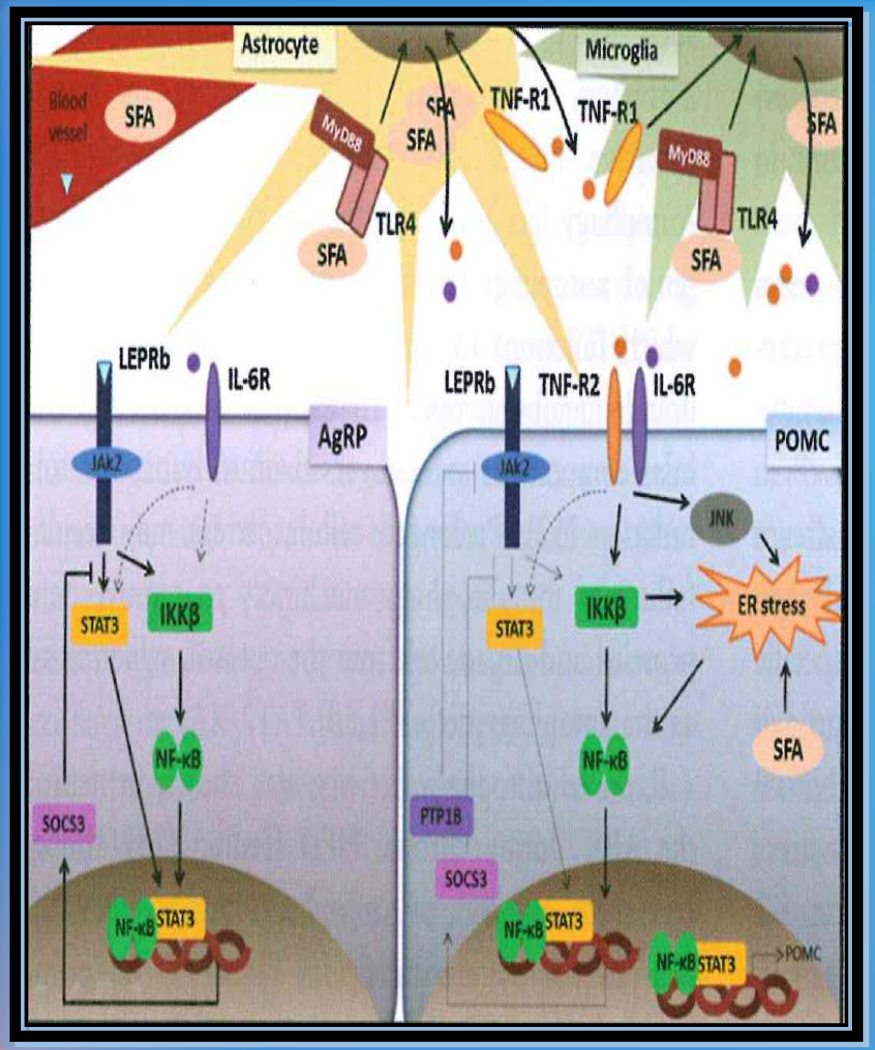
Obesity – impaired FA homeostasis with ß cell /cell death

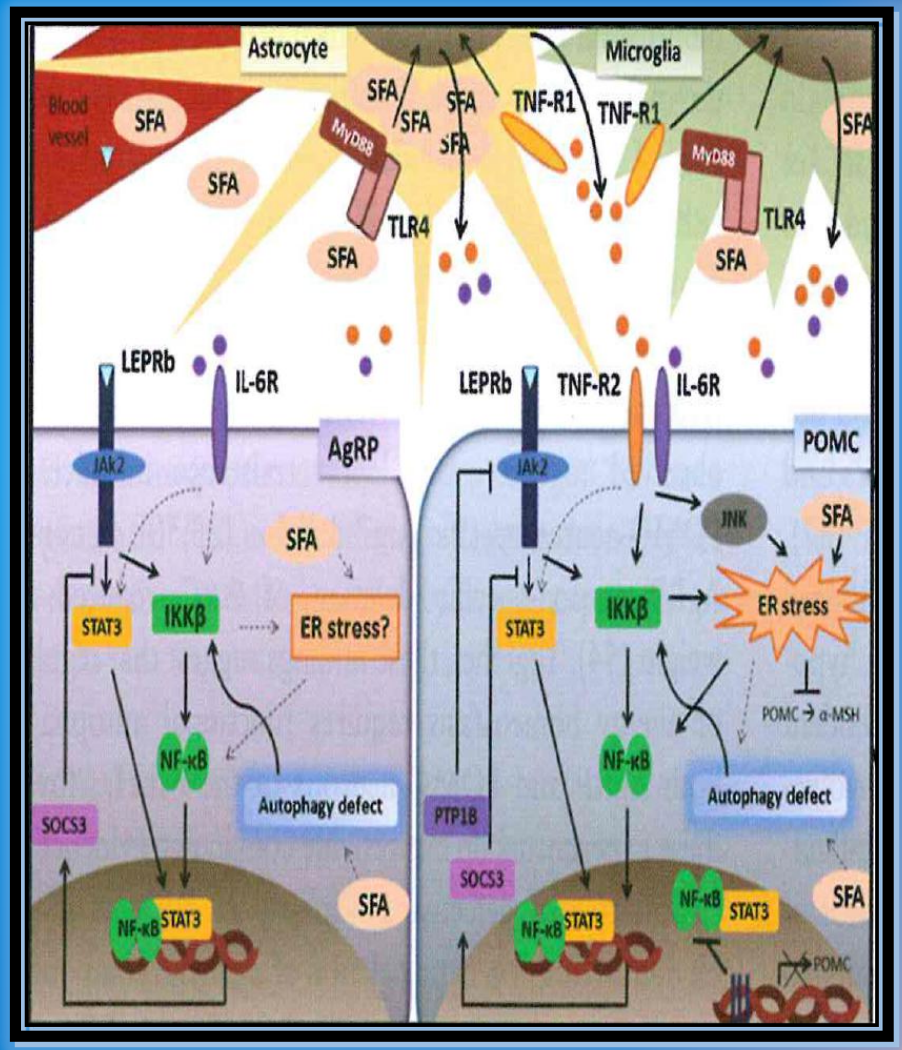


HFD-induced leptin resistance and obesity

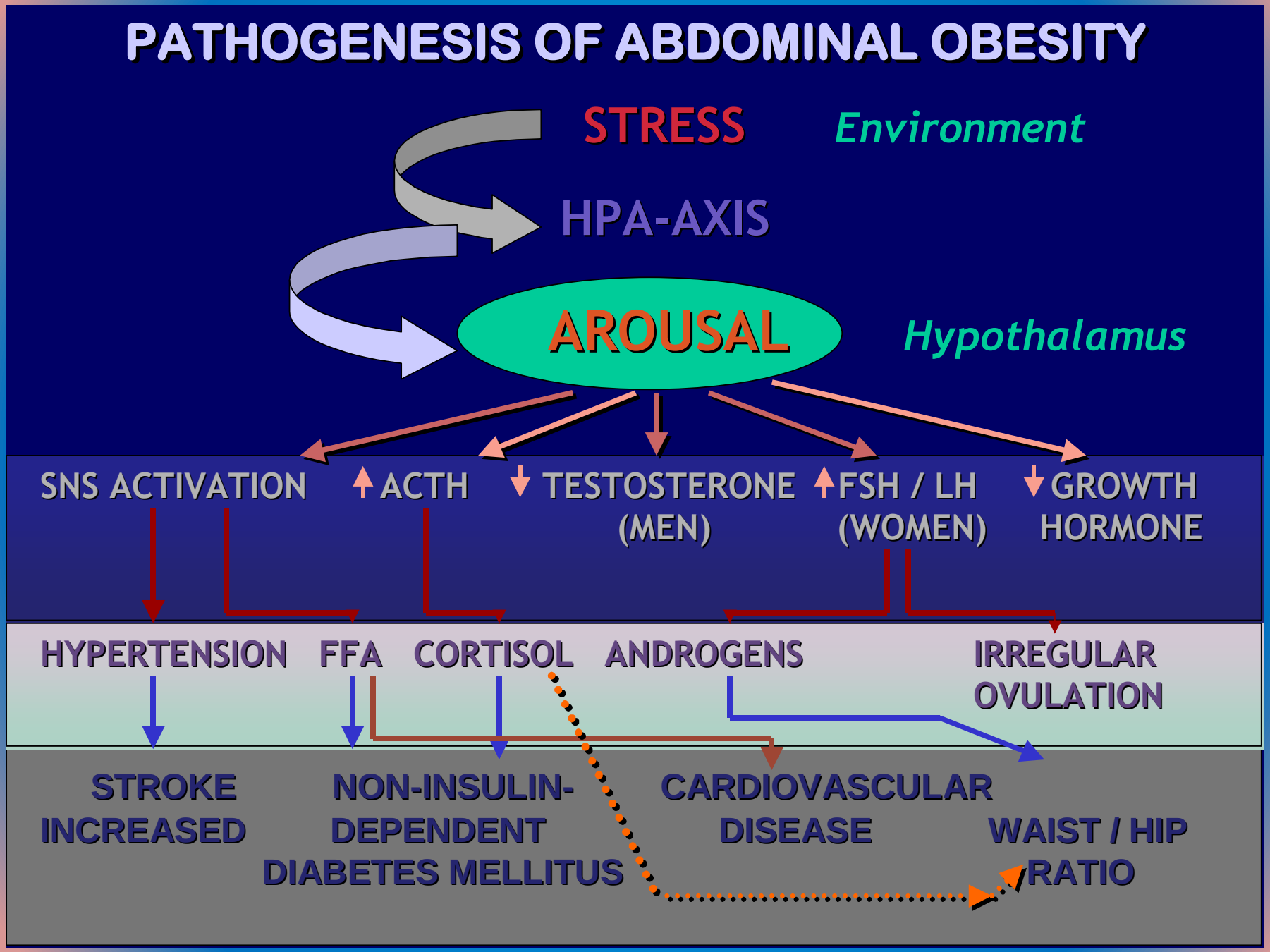
Short-term HFD-feeding

Chronic HFD-feeding

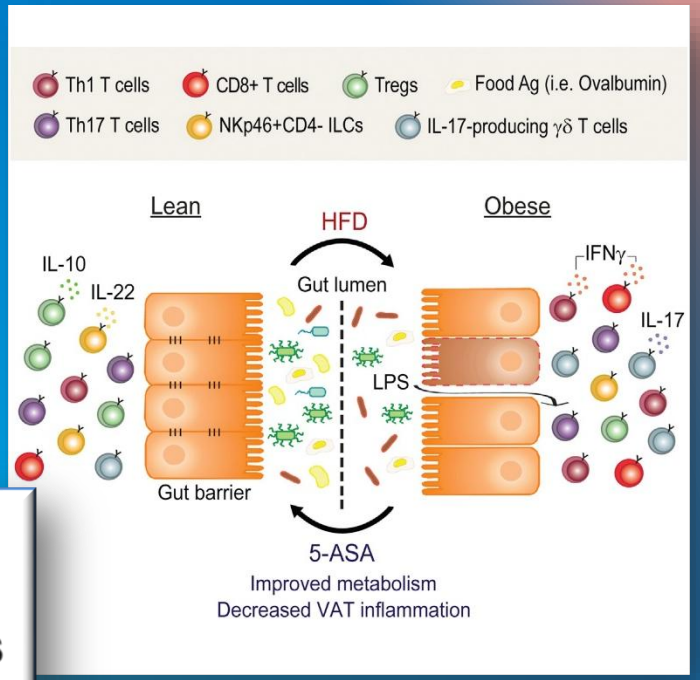




PATHOGENESIS OF ABDOMINAL OBESITY



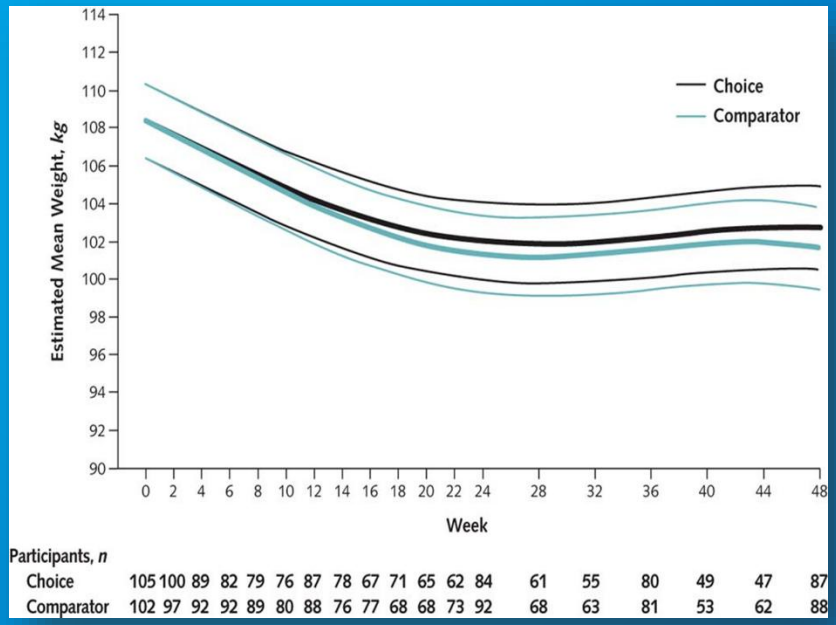
Regulation of Obesity-Related Insulin Resistance with Gut Anti-inflammatory Agents



Highlights

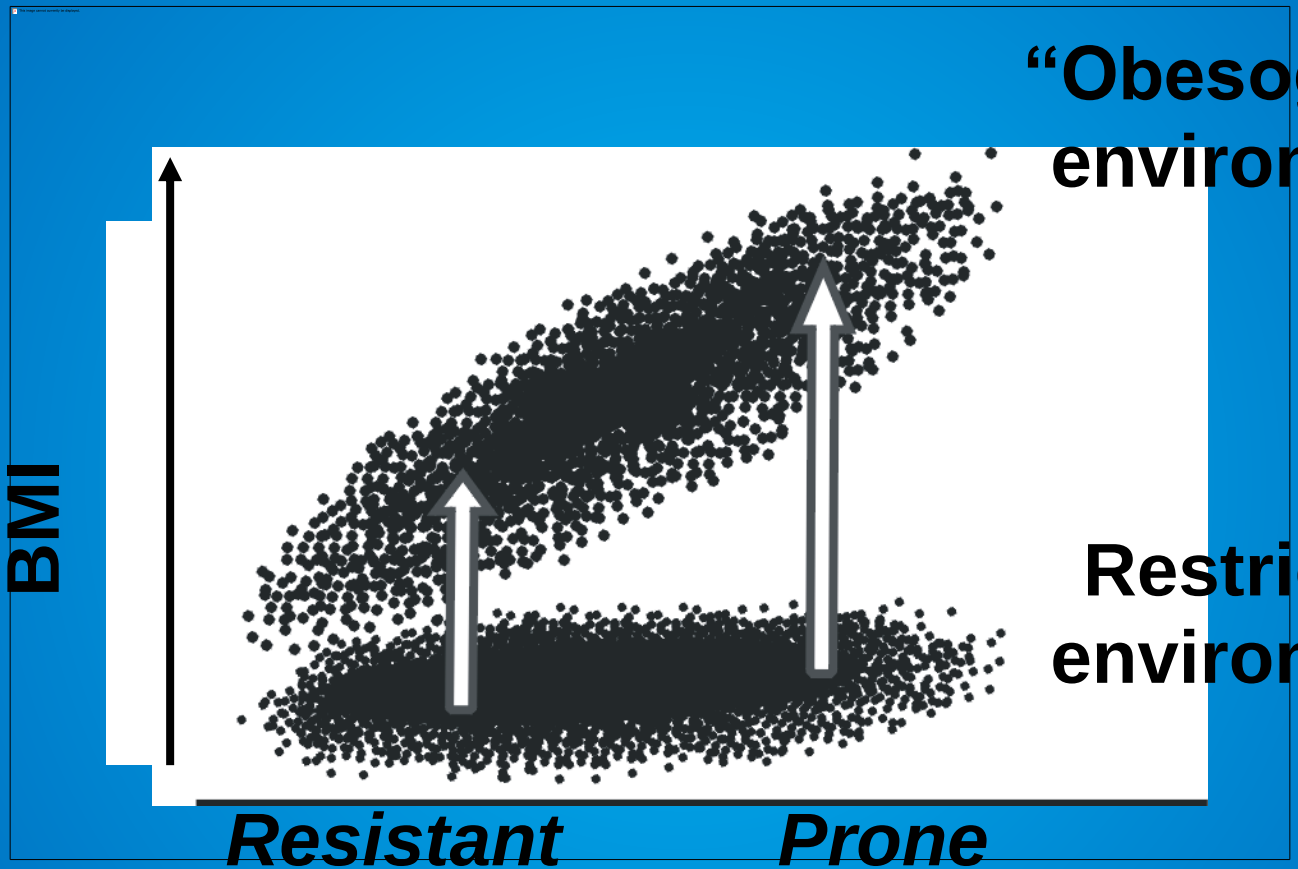
- High-fat diet induces low-grade bowel inflammatory changes in resident immune cells
- Altered gut immunity in obesity contributes to obesity-related insulin resistance
- Gut immunity alters gut barrier, fat inflammation, and oral tolerance in obesity
- Targeting gut inflammation is a novel treatment approach for metabolic disease

9. The principles of Nutrition



Effect of Allowing Choice of Diet on Weight Loss

Defective Biology

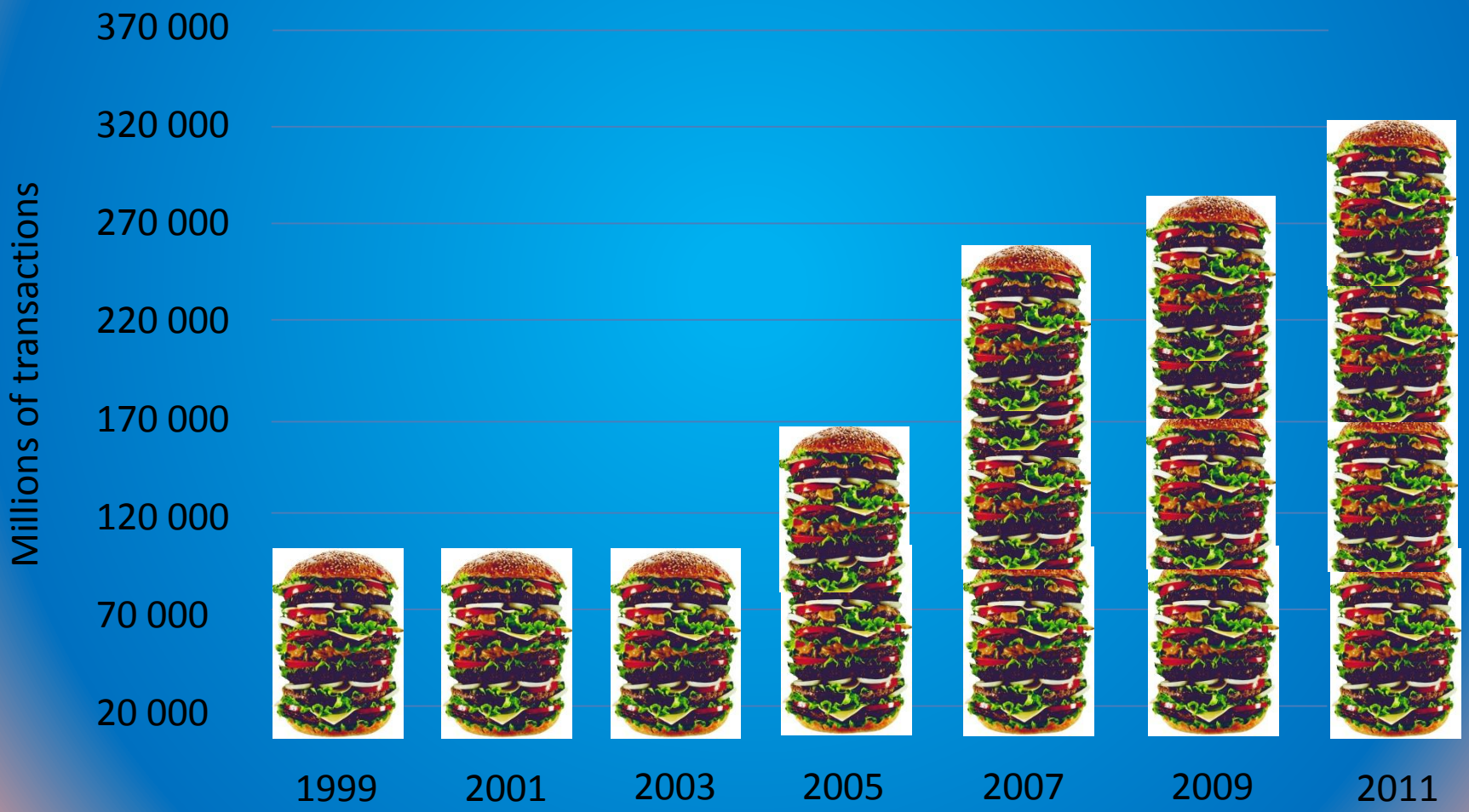


Adapted from Ravussin and Bouchard, 2000

Annual fast food

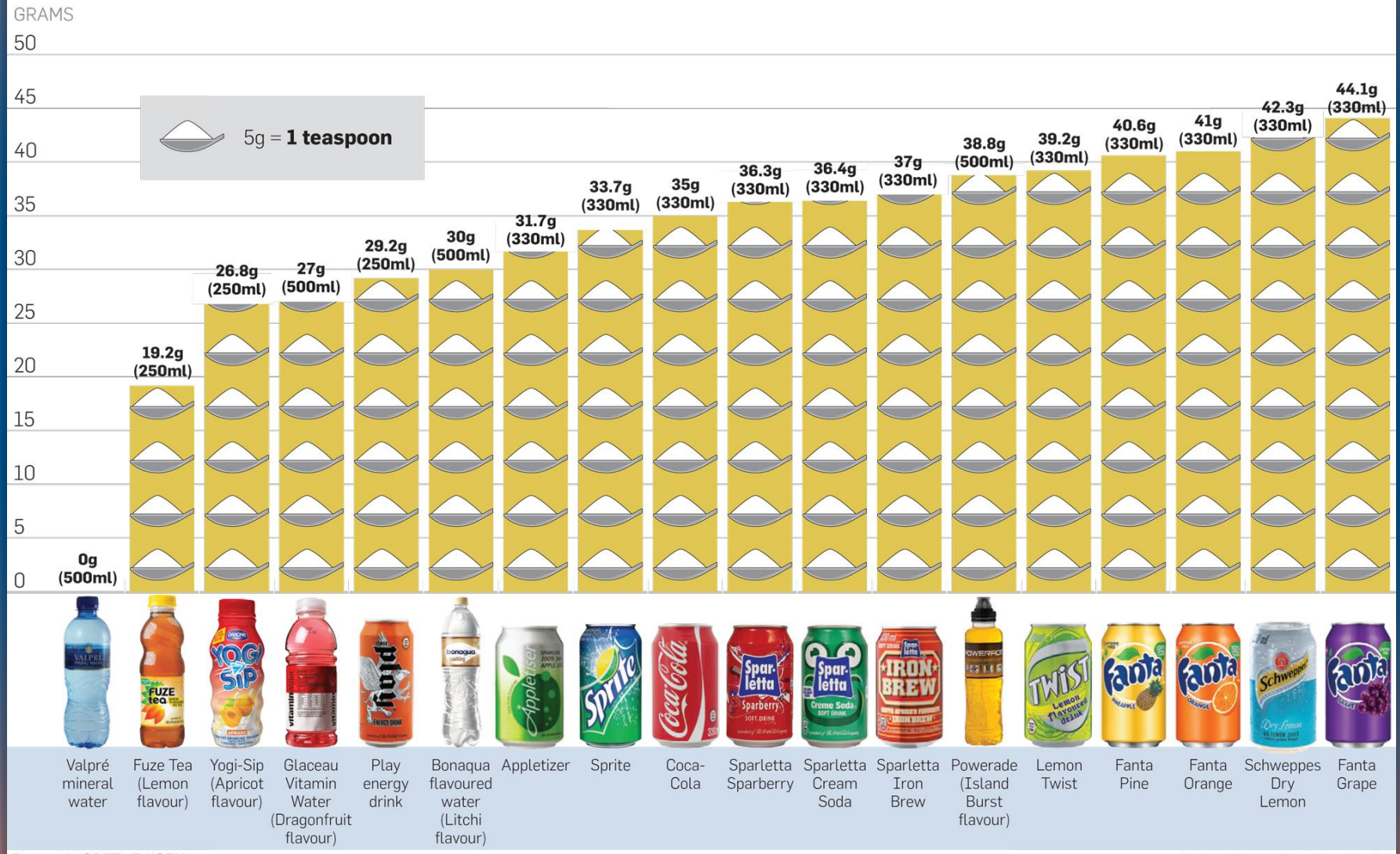
Burger and chicken transactions in South Africa

Euromonitor Report 2012



INFOGRAPHIC: WHAT'S SA'S MOST SUGARY DRINK?

We gathered a few popular drinks from our canteen's refrigerator to check how much sugar each one contains. The results were surprising. Even 'healthier' drinks such as flavoured mineral water and drinking yogurt contain a large amount of sugar



Research: GRETHE KOEN

THEUNS KRUGER, Graphics24

Drinking our kilojoules

Sweetened cool drinks are a major contributor to our high sugar intake. Each soft drink contains up to 55g of sugar.

By 2012 South Africans were drinking 260 cans of Coke per person per year, that's almost three times the global average. ***Coca-Cola Annual Report.***

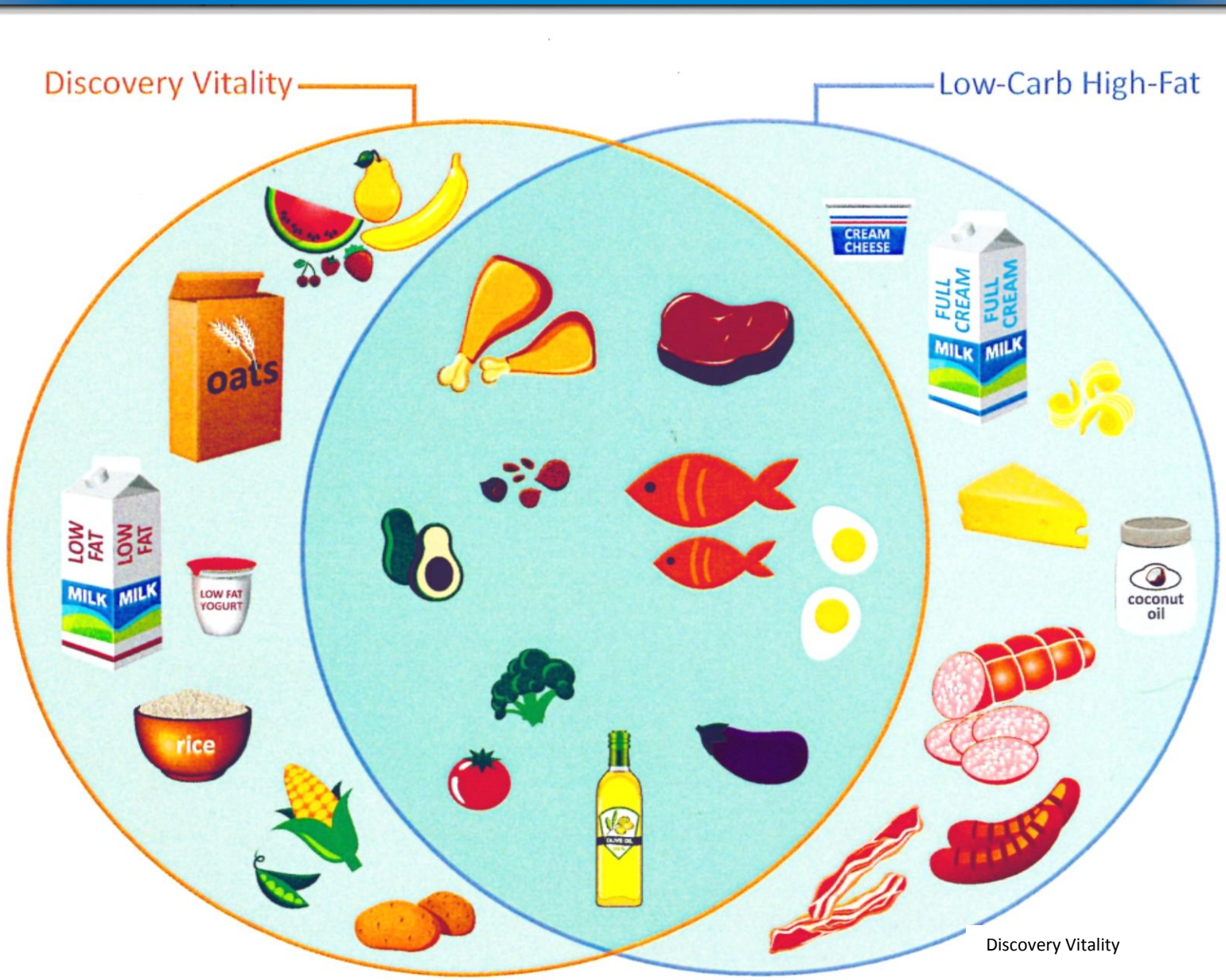
One or more soft drink per day increases risk of diabetes by 26%. Sugar also increases risk for obesity. Risk increases when sugar is consumed in the form of a sugar-sweetened beverages.

Sugar limits

25g: The recommended maximum daily limit for adults. ***World Health Organization***.

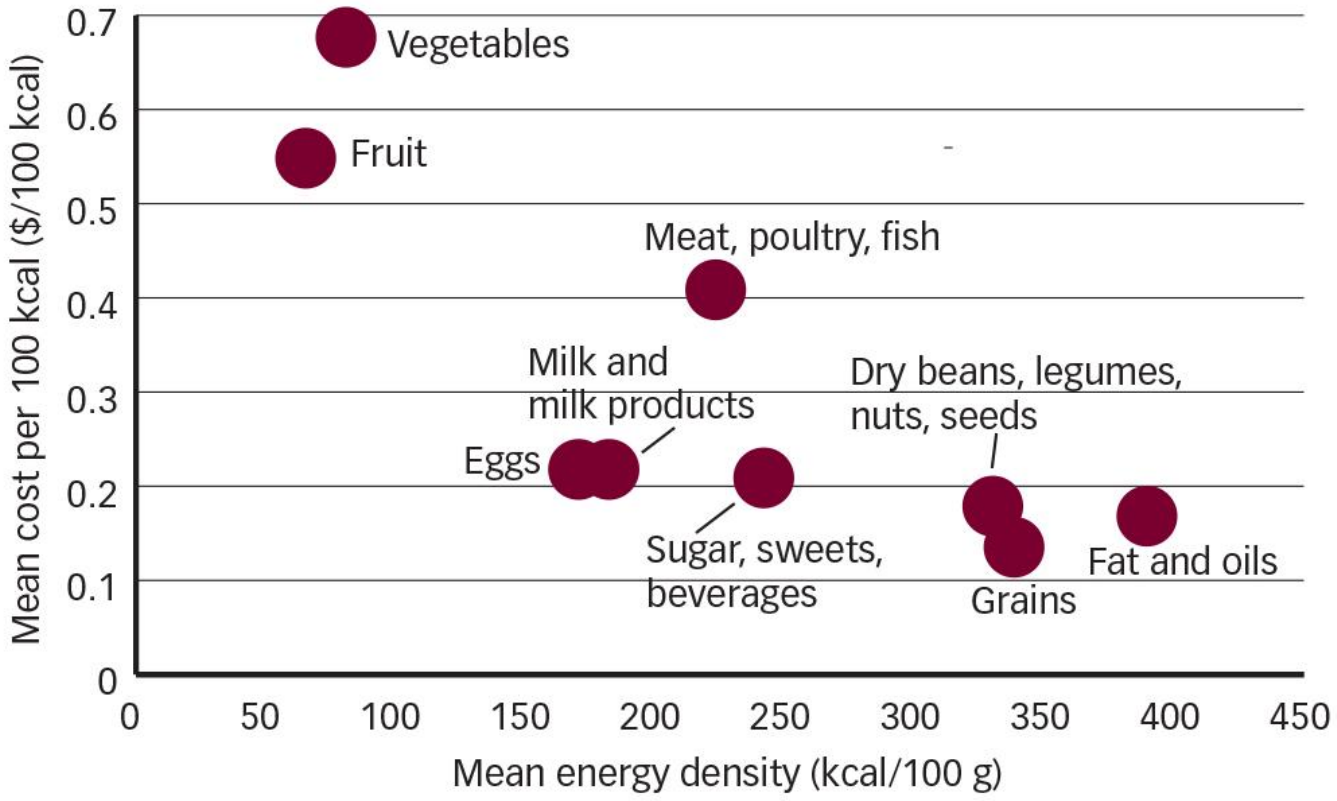
Up to 100g+: The amount of sugar some adults have per day. This includes sugar added to foods such as cereal and tea as well as what is already in processed foods.

How much sugar is in a …

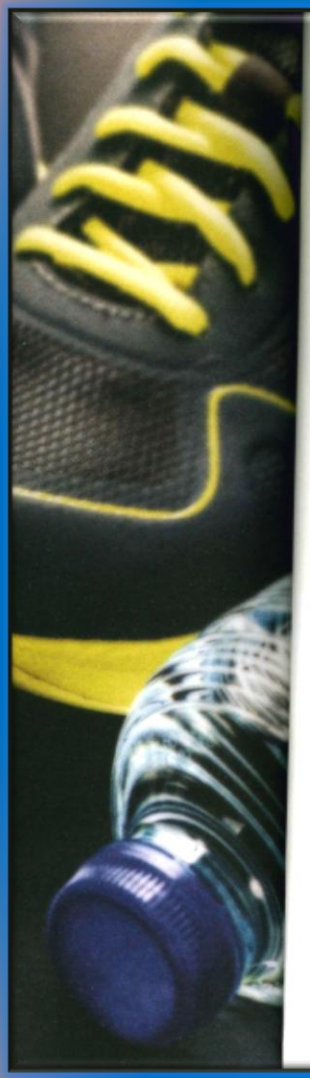


Discovery Vitality

The Relationship between Energy Density (Kcal/100g) and Energy Cost ($/100 kcal) in Nine Major Food Groups



10. The Role of Physical Activity



Sedentary living

A typical day

It's not difficult to spend 90% of your day sitting down: from breakfast at the table, to the commute to work, a day in the office, the drive home, dinner time, and a few hours in front of the TV, much of modern living happens in a seated position. 'Sitting is the new smoking' as they say, and too much chair time can be detrimental to your health, particularly if you are prone to metabolic illnesses like diabetes.

Discovery Vitality

The genetics of physical activity(Bouchard *et al)*

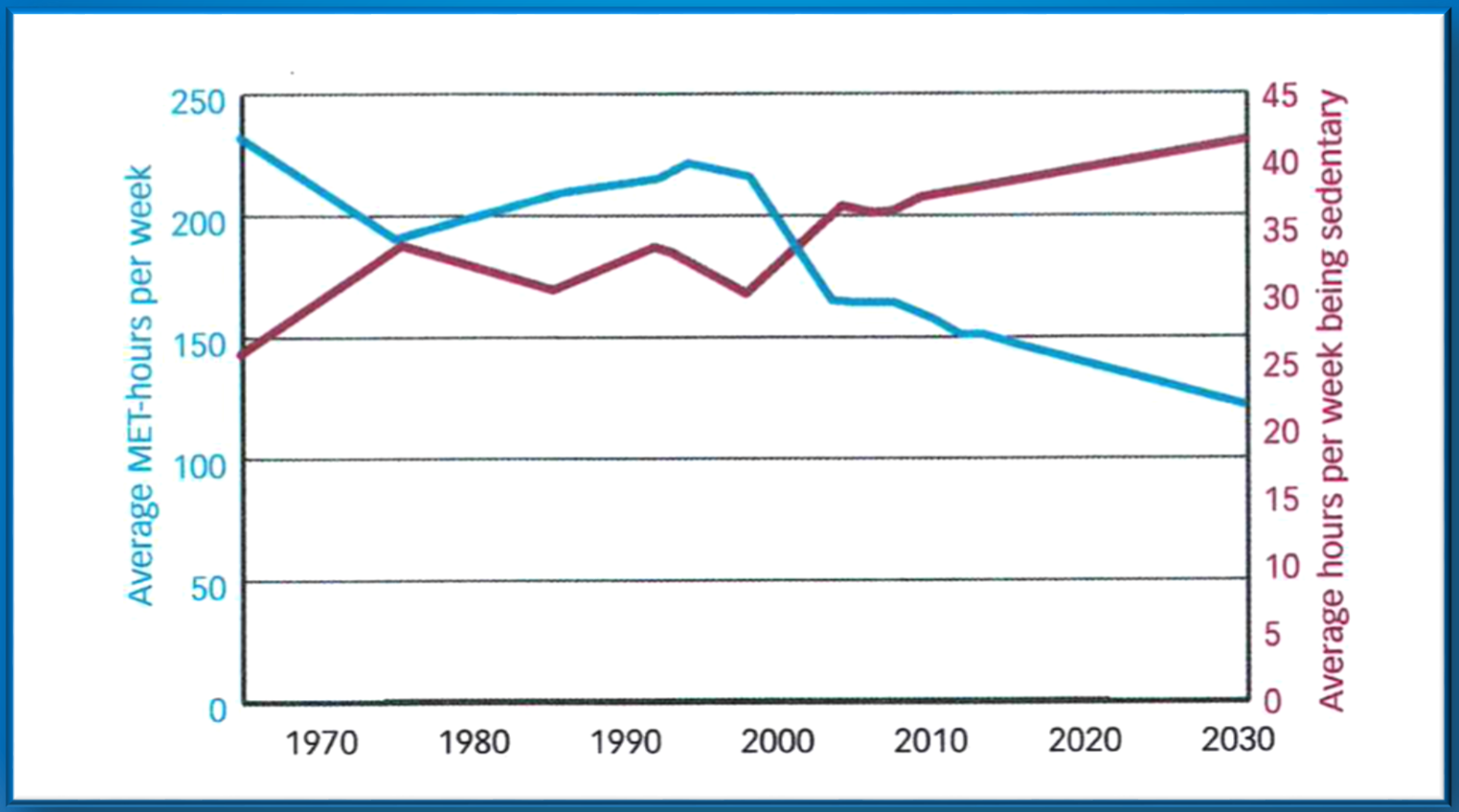
Remarkable genetic variability in response to training and exercise

While factors such as baseline fitness, age, sex, weight, or ethnicity, each explain only around **2-5 %** of the variability to exercise response, familial aggregation can explain around **50%** of the variability

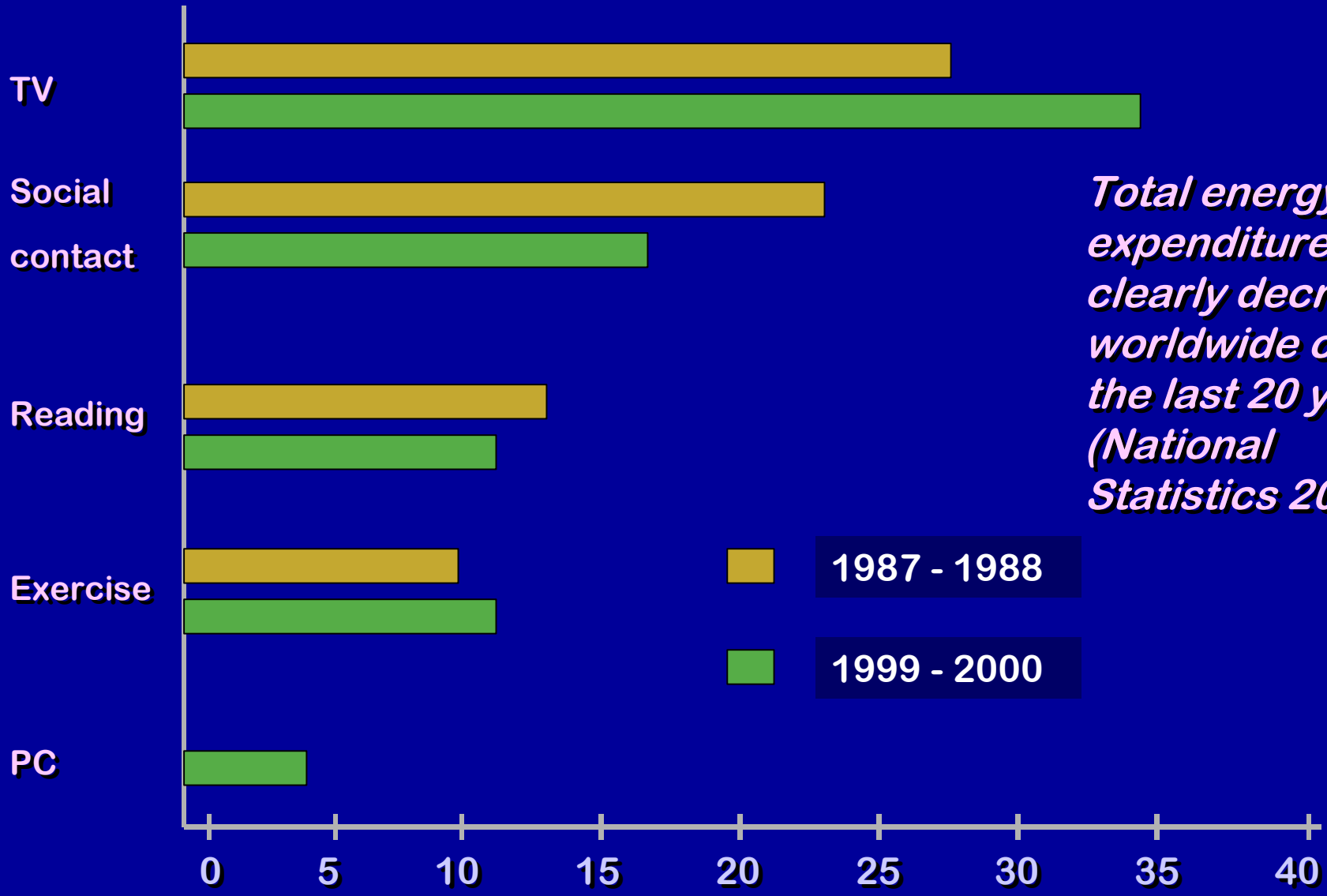
This has important implication for the risk of weight gain

Being "genetically-programmed" for high levels of spontaneous physical activity (e.g. fidgeting) and having genes that allow the body to rapidly adapt to and benefit from regular exercise will make you much less likely to gain weight than if your genetic program reads "sedentariness"

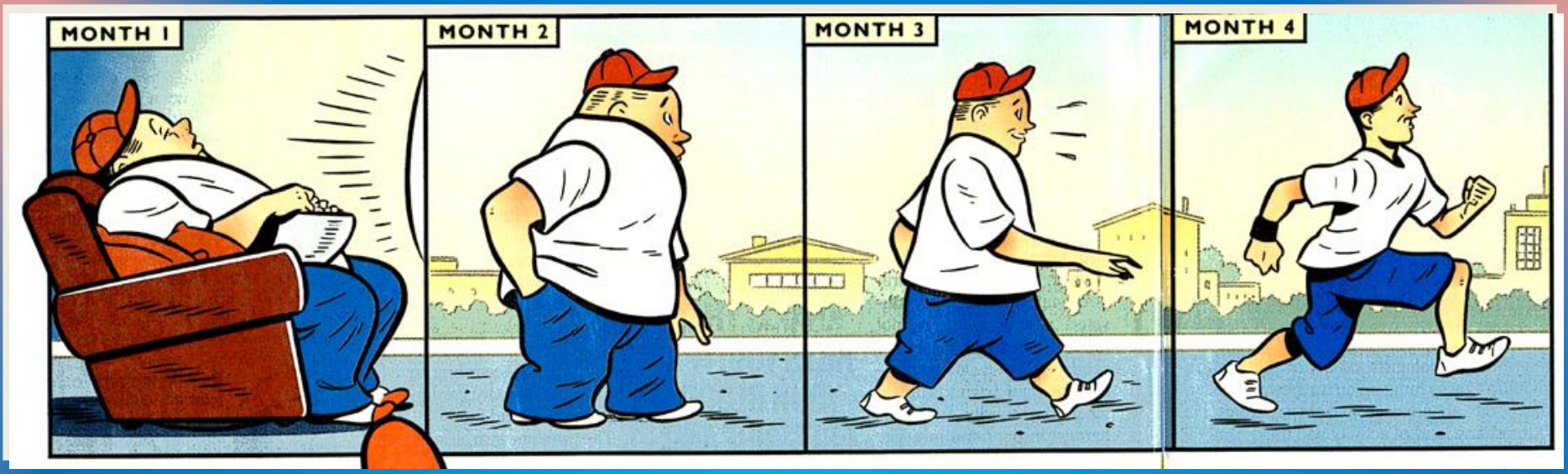
Changes in Levels of Physical Activity and Time Spent in Sedentary Behaviour in the US



Leisure activities in Finland 1987 – 1988 and 1999 - 2000



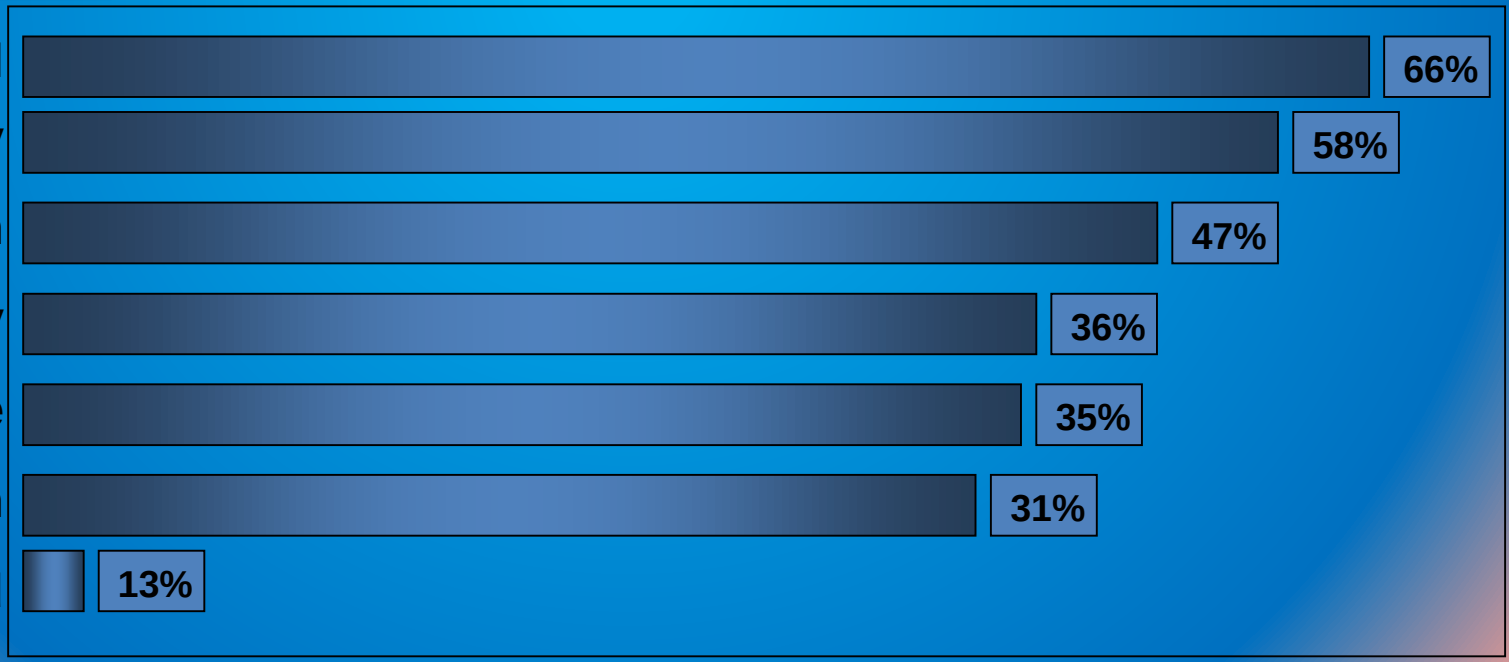
Total energy expenditure has clearly decreased worldwide over the last 20 years (National Statistics 2002)



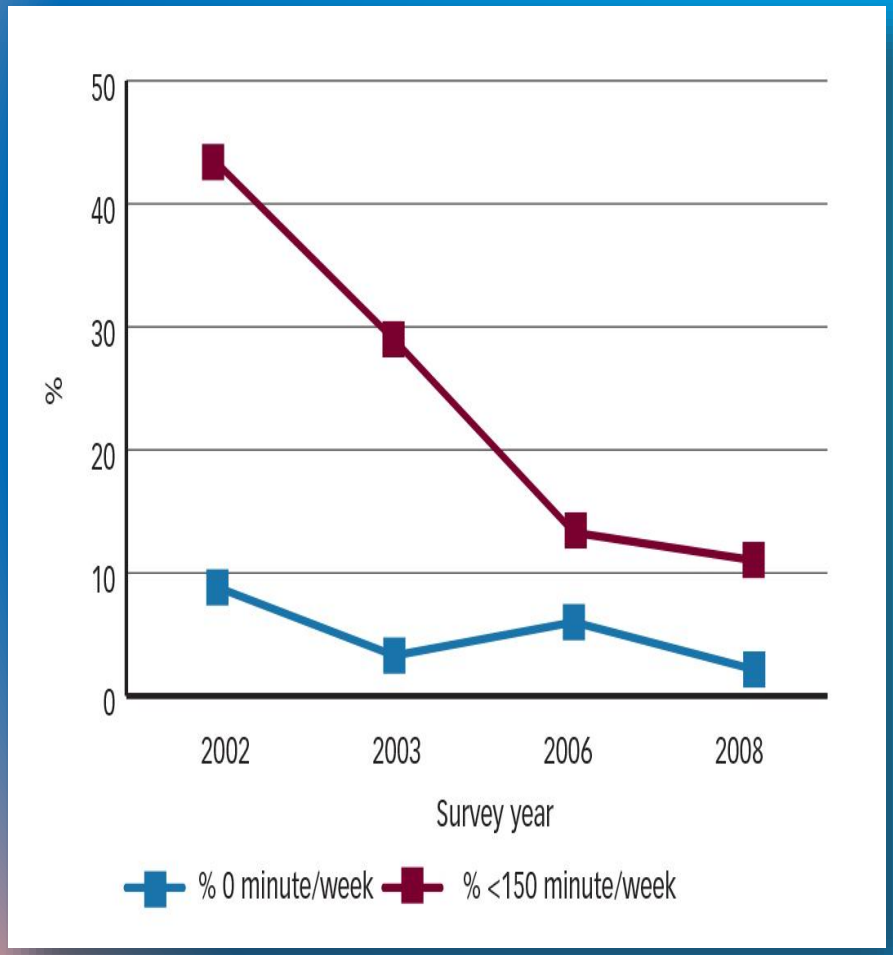
Fit for Life?

The Number of Europeans who never exercise or play sport:

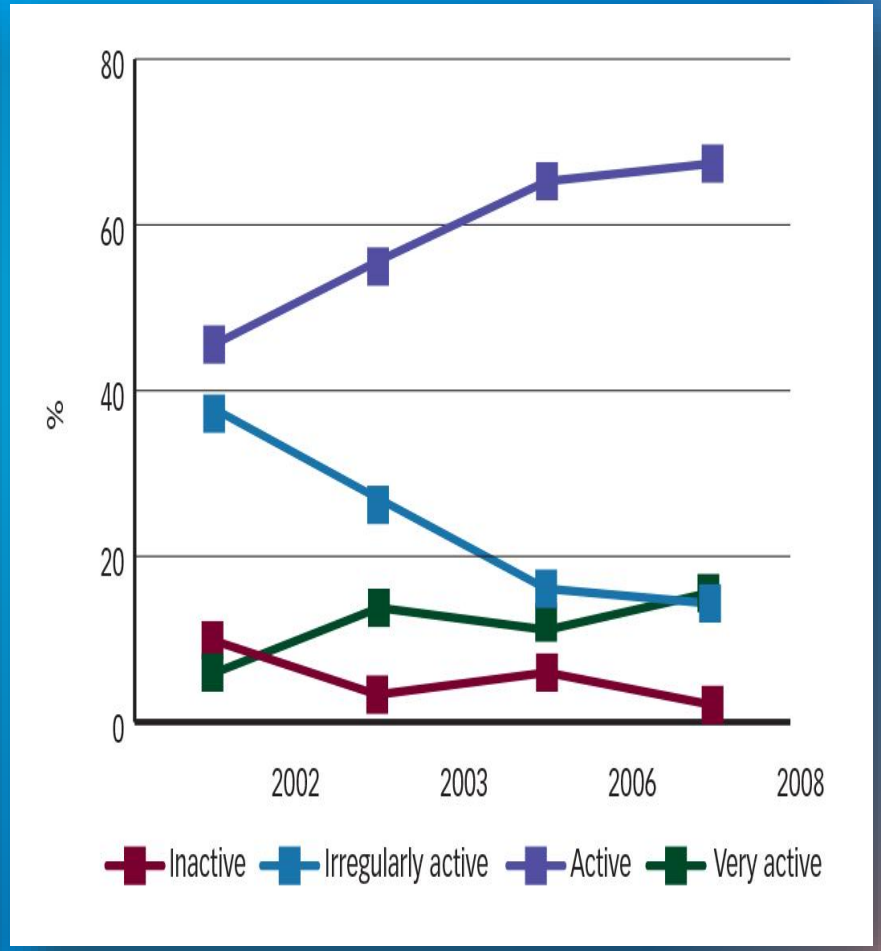
Time, August 8, 2005



Trends in People Inactive or Insufficiently Active in the State of Sao Paulo, Brazil during the Years 2002, 2003, 2006 and 2008



Trends in Physical Activity in the State of Sao Paulo, Brazil during the Years 2002, 2003, 2006 and 2008



ACHIEVING AND MAINTAINING 7KG WEIGHT LOSS

	24h energy expenditure	Energy stores
77kg man	2657 kcal per day	17kg
70kg man	2535 kcal per day	10kg
Difference	122 kcal per day	50 000 kcal
Walking for 30min /day	150 kcal per day	
Chocolate biscuit / day	150 kcal per day	

Characteristics of individuals maintaining a weight loss of at least 13.6 kg (McGiure *et al)*

Body weight loss 13.6kg

Period of maintenance 5.7 years

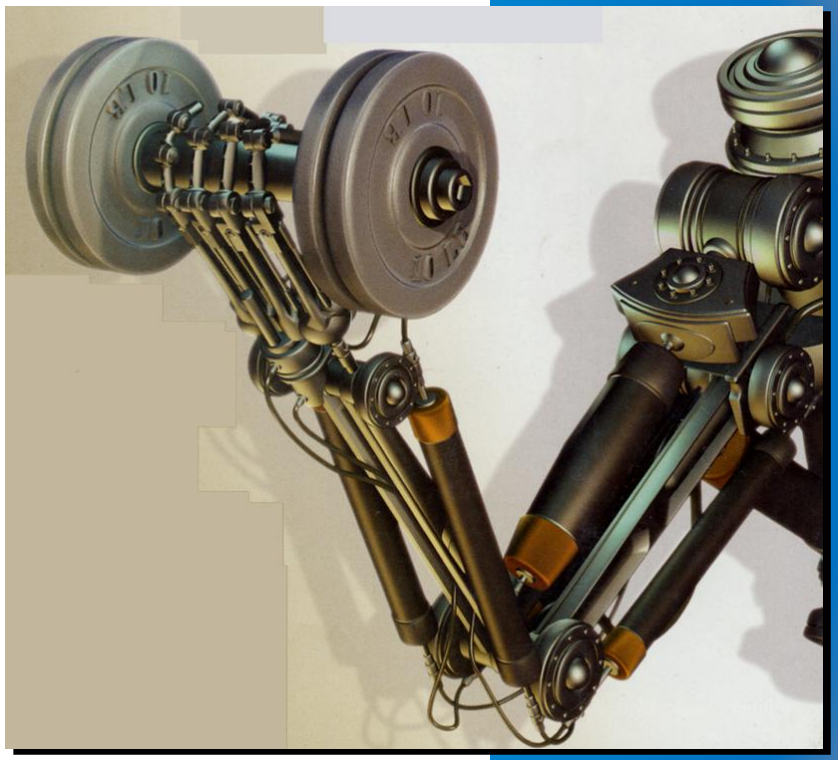
Relative fat intake 25% of total energy intake

Physical activity 11 847KJ (or 2820 cal)/week

Changes that occur with endurance training

Cardiovascular and whole-body

- Increased cardiac output, and ability to increase this during exercise
- Improved respiratory function
- Increased lean body mass (mainly muscle bulk)
- Decreased body fat
- Increased bone strength



Structural changes in muscle

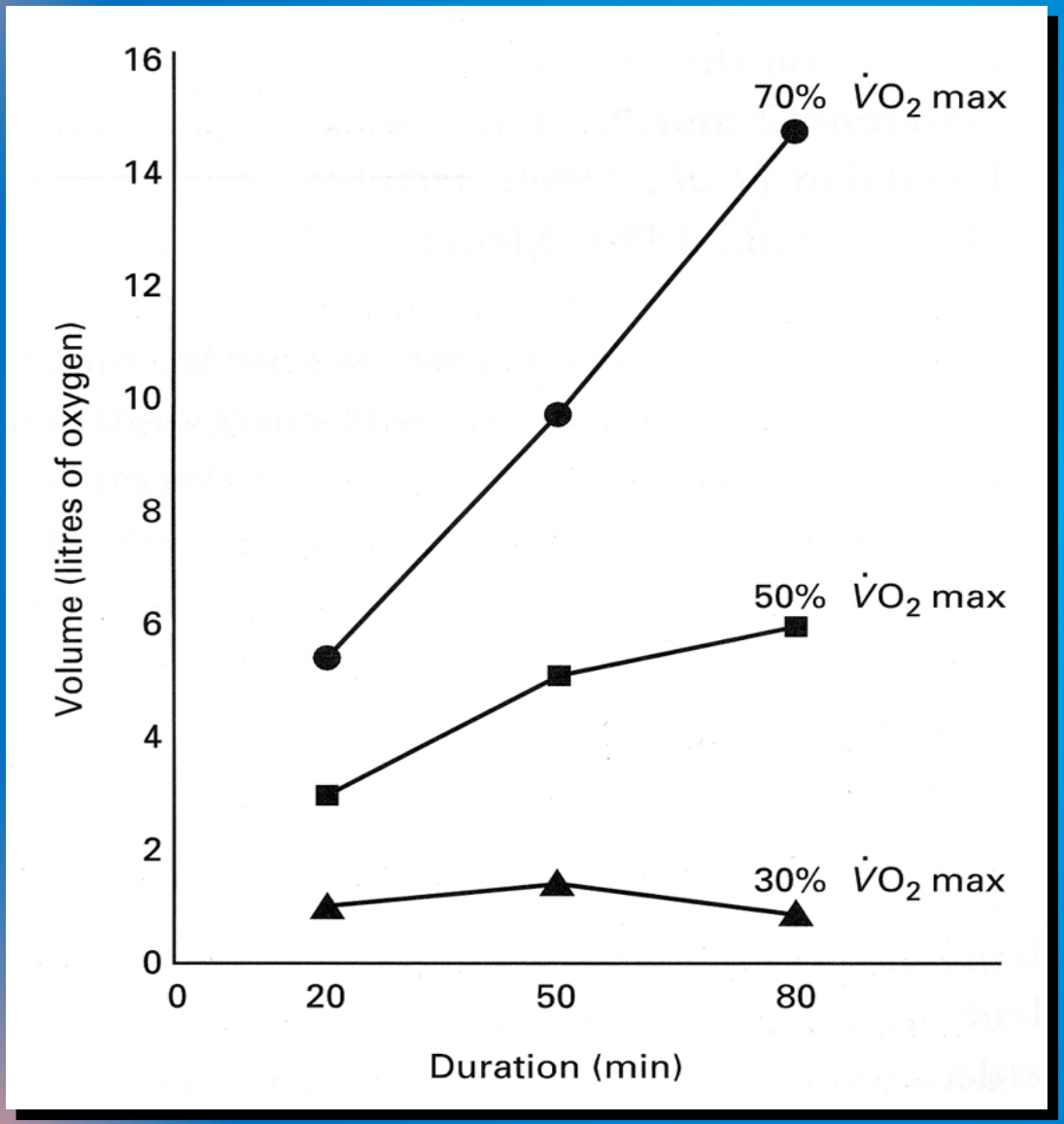
- Increased density of capillaries
- Increased number of mitochondria
- Increased size of mitochondria
- Increased myoglobin concentration

Metabolic changes in muscle

- Increased expression of GLUT4
- Increased sensitivity to insulin
- Increased activity of lipoprotein lipase
- Increased activity of oxidative enzymes in mitochondria (tricarboxylic acid cycle and β-oxidation)
- Increased glycogen synthase activity

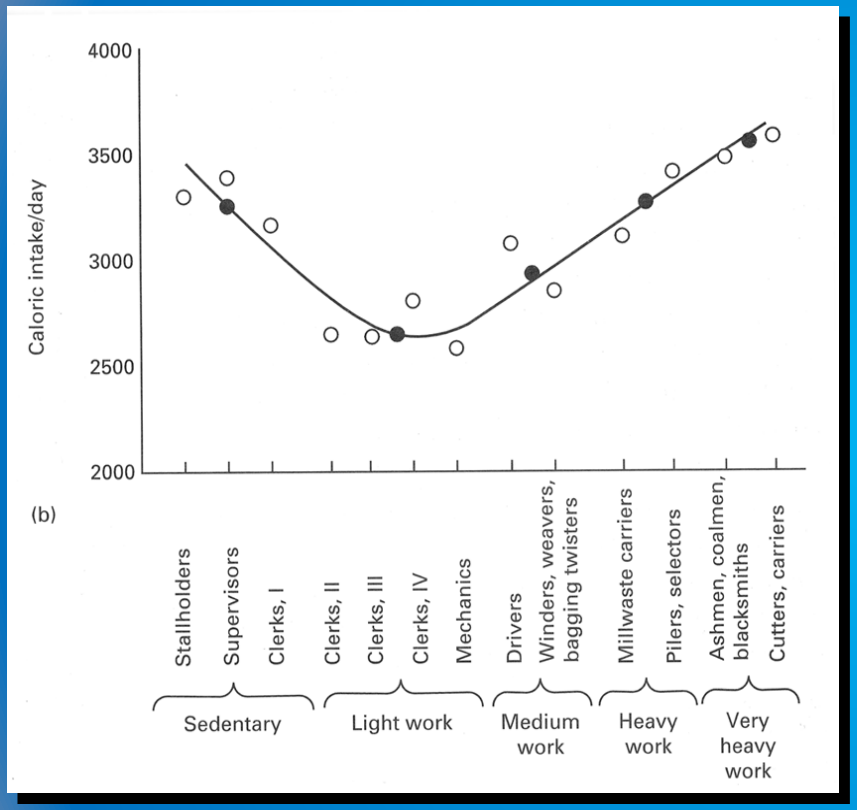
Metabolic Regulation. A human perspective. J.N. Frayn. Portland Press 1996, chapter 7

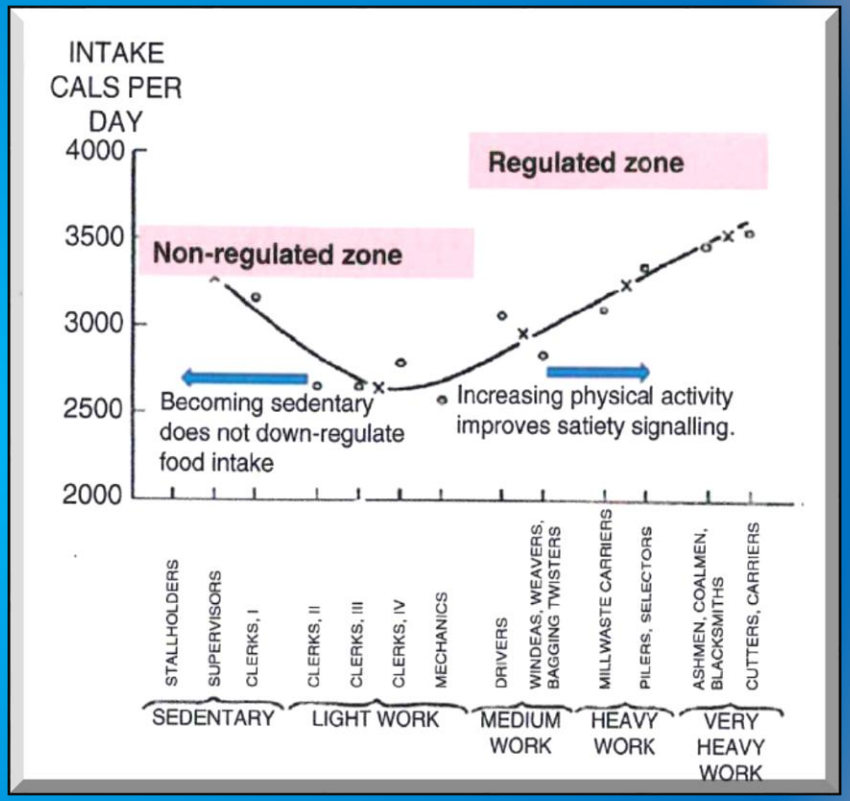
POSTEXERCISE ENERGY EXPENDITURE



Excess postexercise oxygen consumption (EPOC) resulting from 20, 50 and 80 minutes of treadmill exercise at 30, 50 and 70% VO_2 max. Adapted from Gore and Withers (1990).

Appetite control and energy balance: impact of exercise





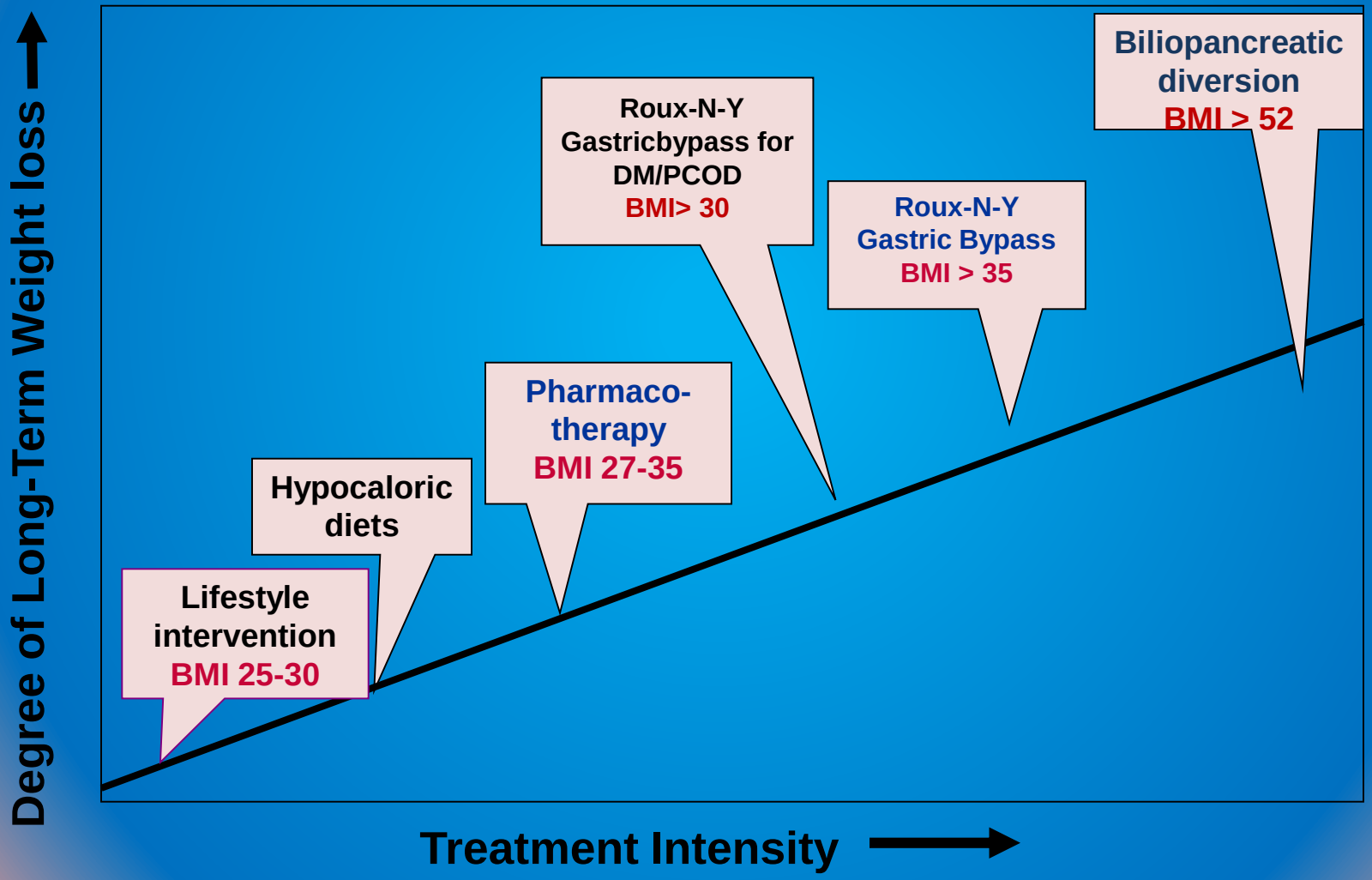
Caloric intake as a function of level of physical activity at work, in an industrial male population in West Bengal. From Mayer *et al.* (1956)

11. Medical Management

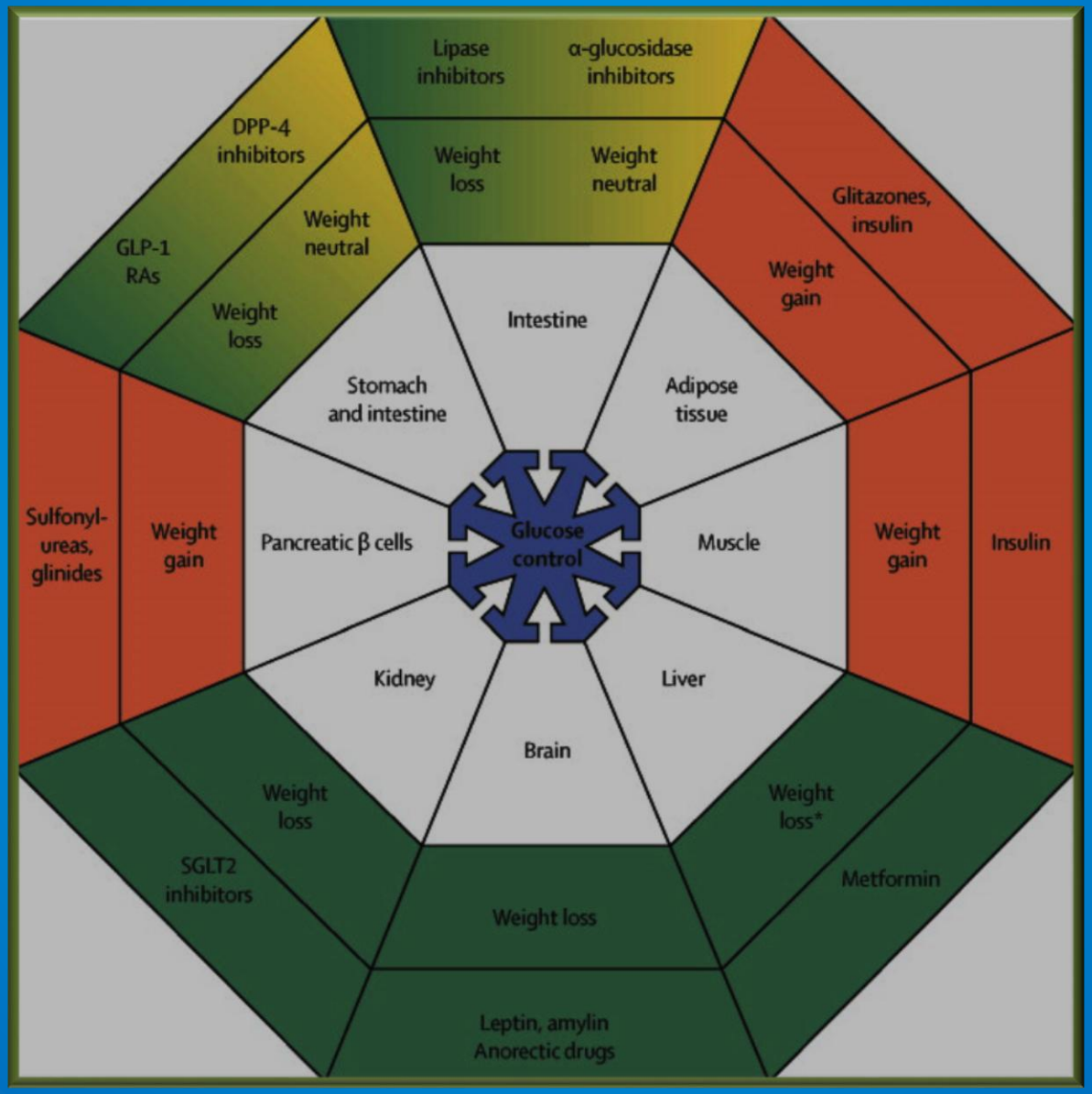
To have a purpose that is worthwhile, and that is steadily being accomplished, that is one of the secrets of a life that is worth living

Herbert Casson (1869-1951)

Stepped **Care Approach to Obesity Management**



Several pharmacological approaches used to control hyperglycaemia in type 2 Diabetes, with a focus on the drugs' effects on bodyweight

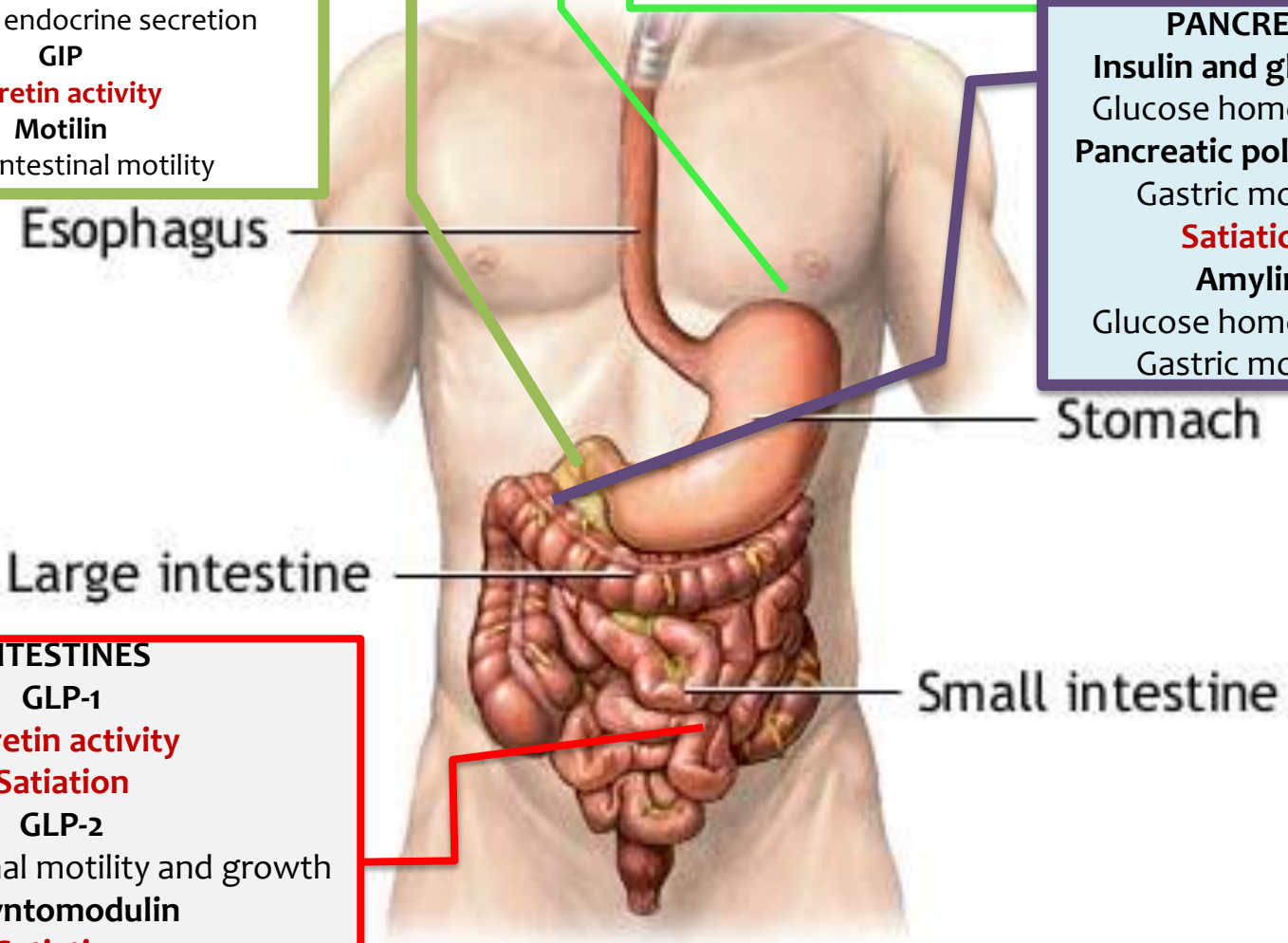


LANCET Diabetes & Endocrinology 2014

DUODENUM
Cholecystokinin
Gall bladder contraction
Gastrointestinal motility
Pancreatic endocrine secretion
Secretin
Pancreatic endocrine secretion
GIP
Incretin activity
Motilin
Gastrointestinal motility

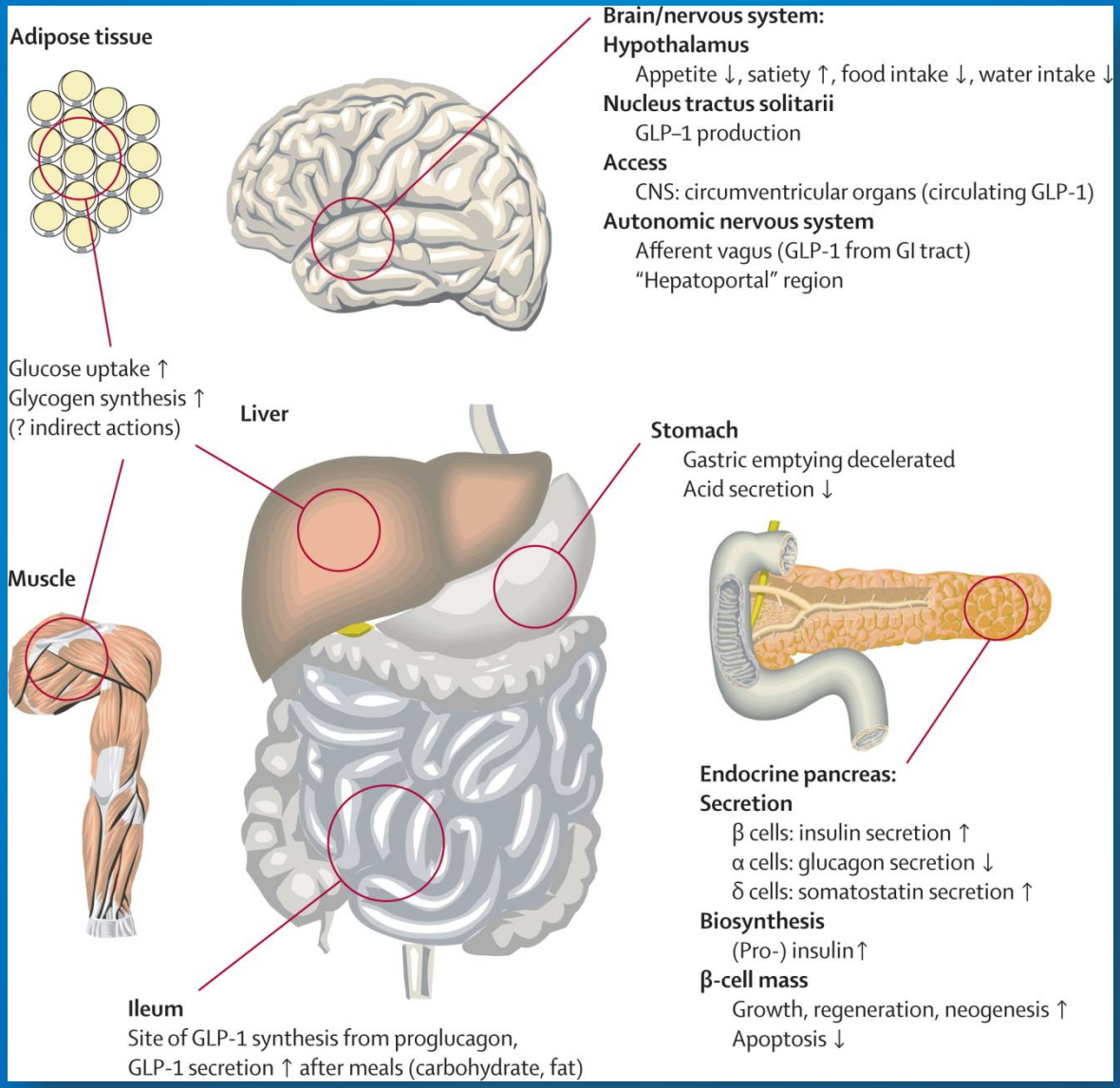
STOMACH
Ghrelin
Hunger
Growth hormone release
Gastrin
Acid secretion

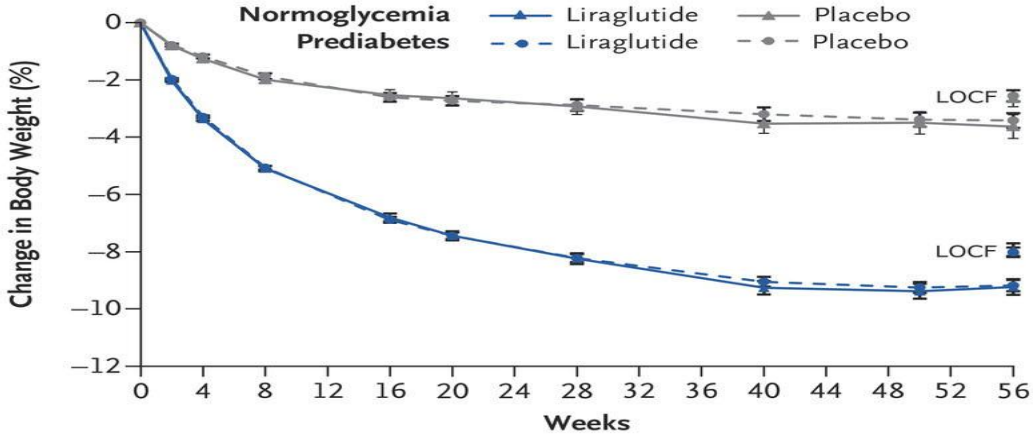
PANCREAS
Insulin and glucagon
Glucose homeostasis
Pancreatic polypeptide
Gastric motility
Satiation
Amylin
Glucose homeostasis
Gastric motility

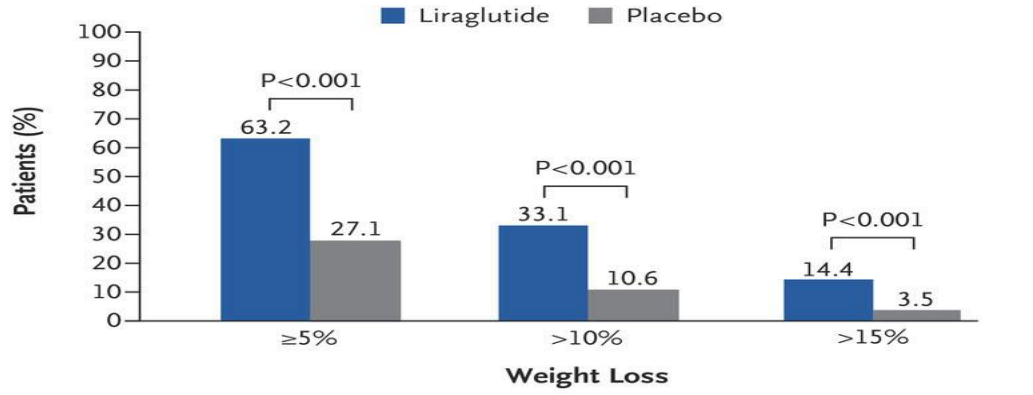


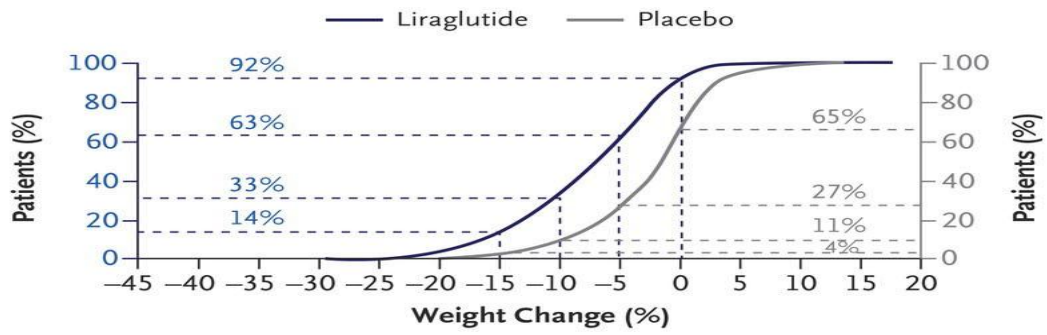
INTESTINES
GLP-1
Incretin activity
Satiation
GLP-2
Gastrointestinal motility and growth
Oxyntomodulin
Satiation
Acid secretion
PYY_{3-34}
Satiation

The incretin system: glucagon-like peptide-1 receptor agonists and dipeptidyl peptidase-4 inhibitors in type 2 diabetes

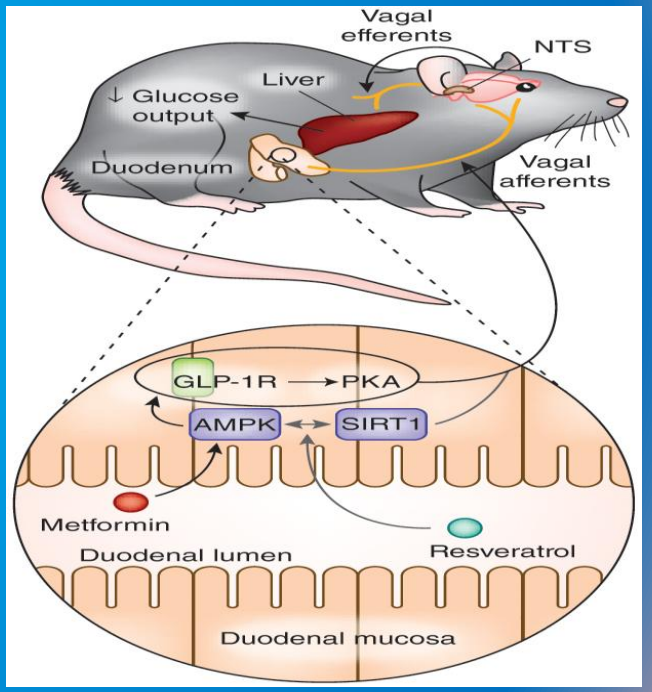



Normoglycemia
Liraglutide
Placebo
Prediabetes
Liraglutide
Placebo
LOCF
LOCF
Change in Body Weight (%)
0
−2
−4
−6
−8
−10
−12
0 4 8 12 16 20 24 28 32 36 40 44 48 52 56
Weeks


Liraglutide
Placebo
Patients (%)
100
90
80
70
60
50
40
30
20
10
0
P<0.001
63.2
27.1
P<0.001
33.1
10.6
P<0.001
14.4
3.5
≥5%
>10%
>15%
Weight Loss

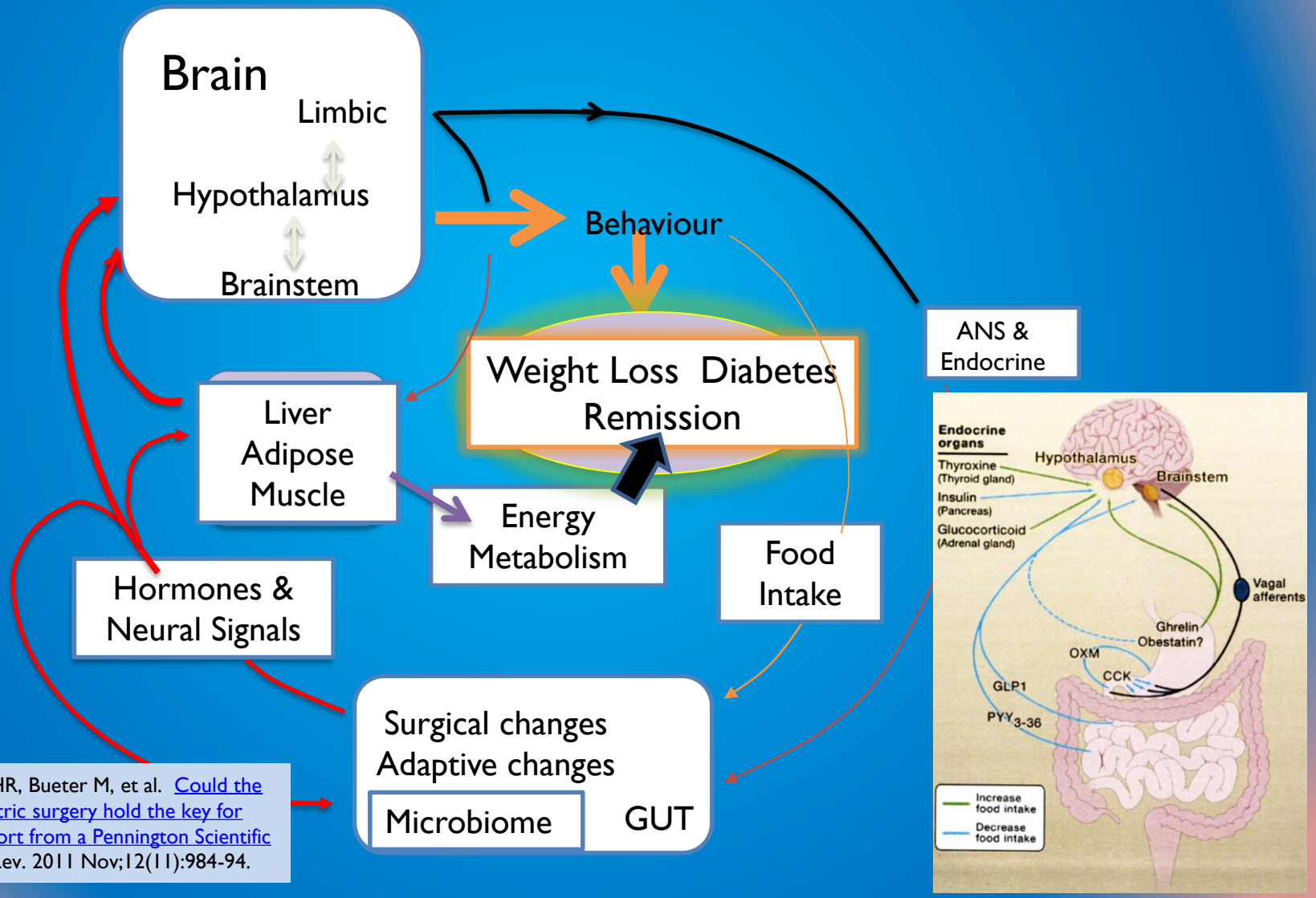

Liraglutide
Placebo
Patients (%)
100
80
60
40
20
0
92%
63%
33%
14%
65%
27%
11%
4%
−45 −40 −35 −30 −25 −20 −15 −10 −5 0 5 10 15 20
Weight Change (%)

12. Surgical Management



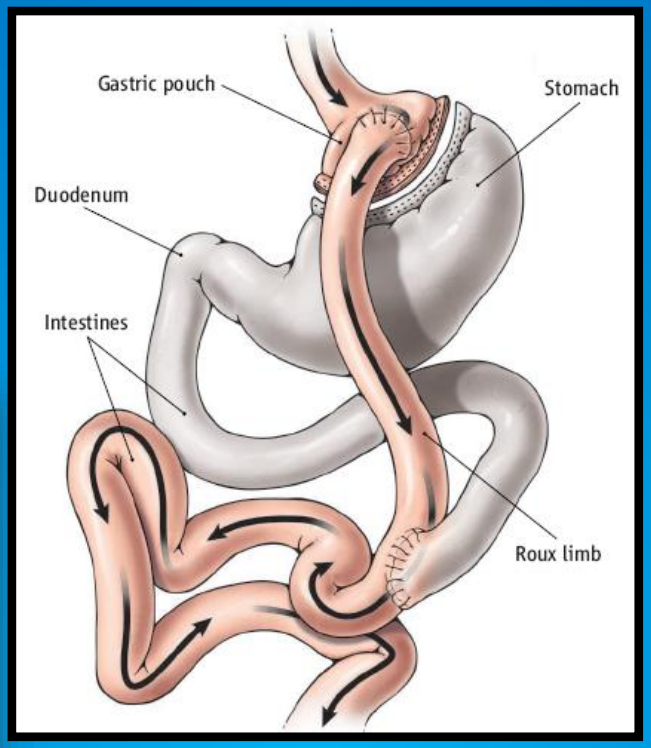
Duodenal energy sensing regulates hepatic glucose output
Nature 21: 5; May 2015

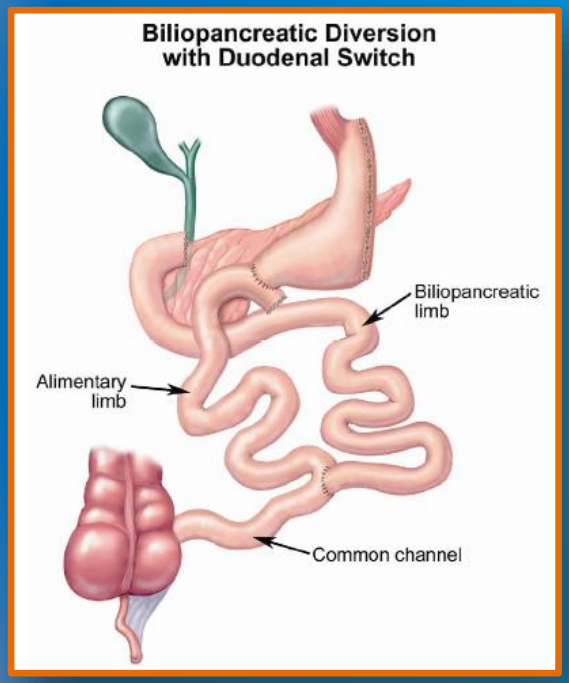
MECHANISMS OF BARIATRIC SURGERY – KEY FOR NOVEL THERAPIES?

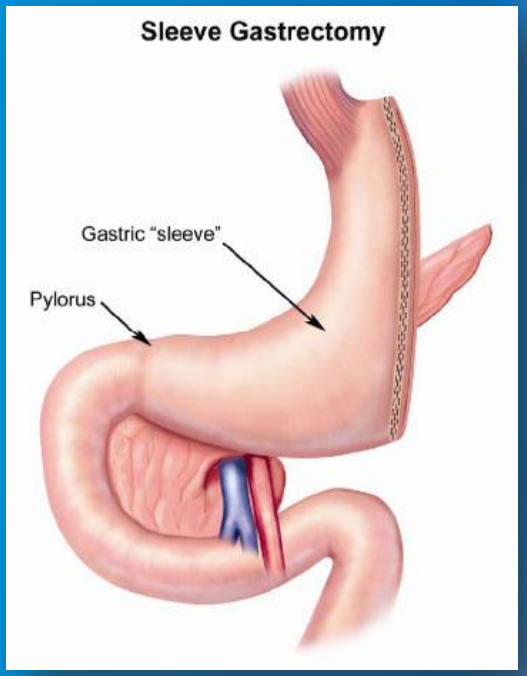


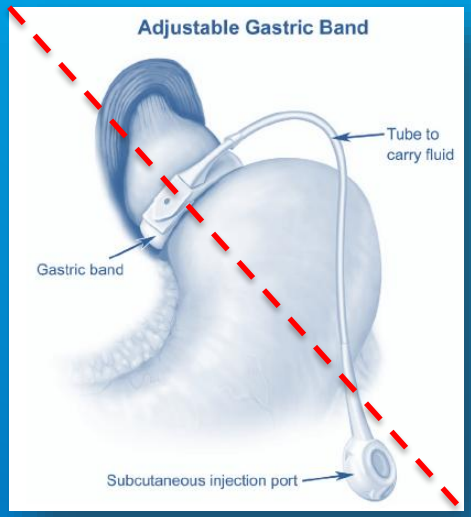
Tam CS, Berthoud HR, Bueter M, et al. Could the mechanisms of bariatric surgery hold the key for novel therapies? report from a Pennington Scientific Symposium. Obes Rev. 2011 Nov;12(11):984-94.

Surgical Procedures





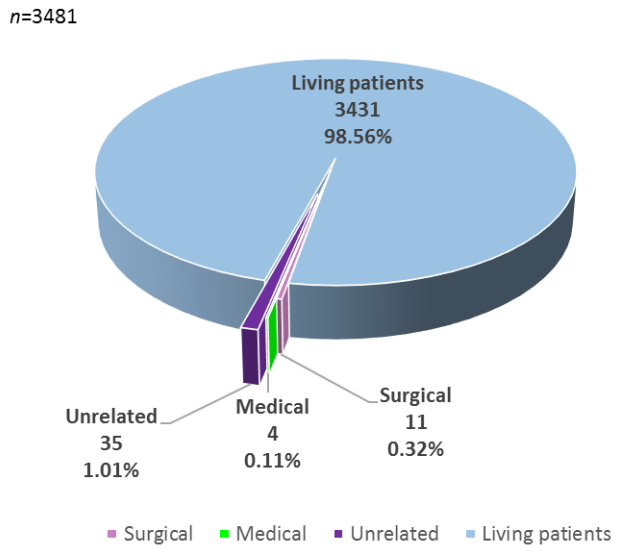




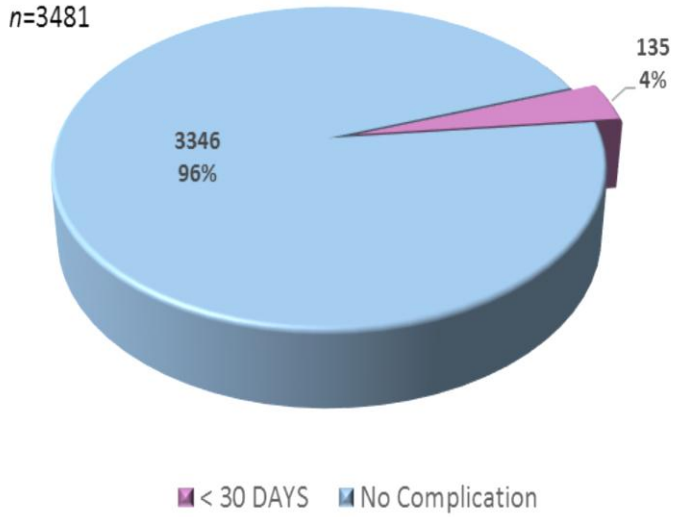
Tallies up to the end of July 2015

HOSPITAL	SURGEON	TOTALS
George Mediclinic	Dr. Folscher	90
Durbanville Mediclinic	Dr. Swanepoel	267
Sunwardpark Hospital	Dr. Schutte	245
St. Augustine's Hosp	Dr. Du Toit	168
St. Augustine's Hosp	Dr. Campbell	2
Waterfall City Hosp	Dr. Fetter	1468
Waterfall City Hosp	Dr. Naidoo	29
Kingsbury Hosp	Dr. Stapleton	105
Bloemfontein M-Clinic	Dr. Heyns	68
St. Augustine's Hosp	Dr. Funnel	91
Kingsbury Hosp	Dr. I. Marr	20
Kingsbury Hosp	Dr. J. Marr	77
N1 City Hosp	Dr. Potgieter	275
Rosepark Hosp	Dr. Fichardt	142
Green acres Hosp	Dr. Van Niekerk	118
Green acres Hosp	Dr. Wasserman	4
St. Anne's Hosp	Dr. Brombacher	176
Union Hosp	Dr. WMJ Van Vuuren	89
TOTALS		**3434**

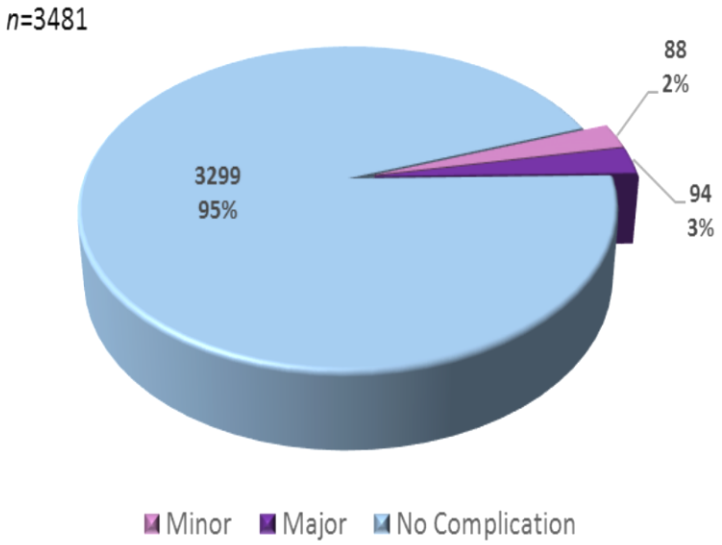
NATIONAL - MORTALITIES



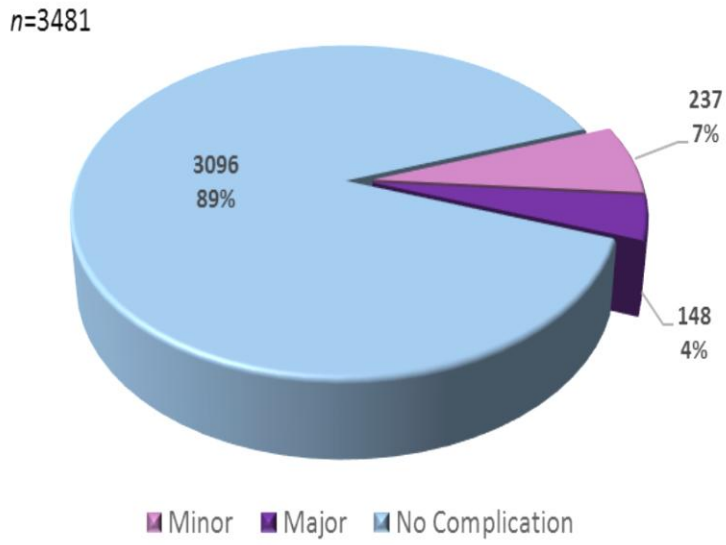
NATIONAL - MEDICAL COMPLICATIONS < 30 DAYS



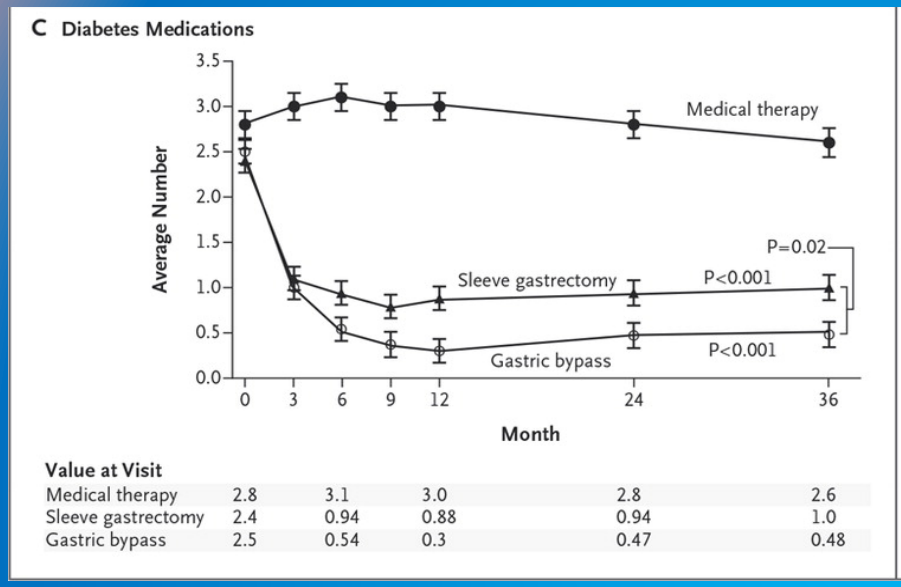
NATIONAL - SURG COMP <30 DAYS



NATIONAL - SURG COMP >30 DAYS

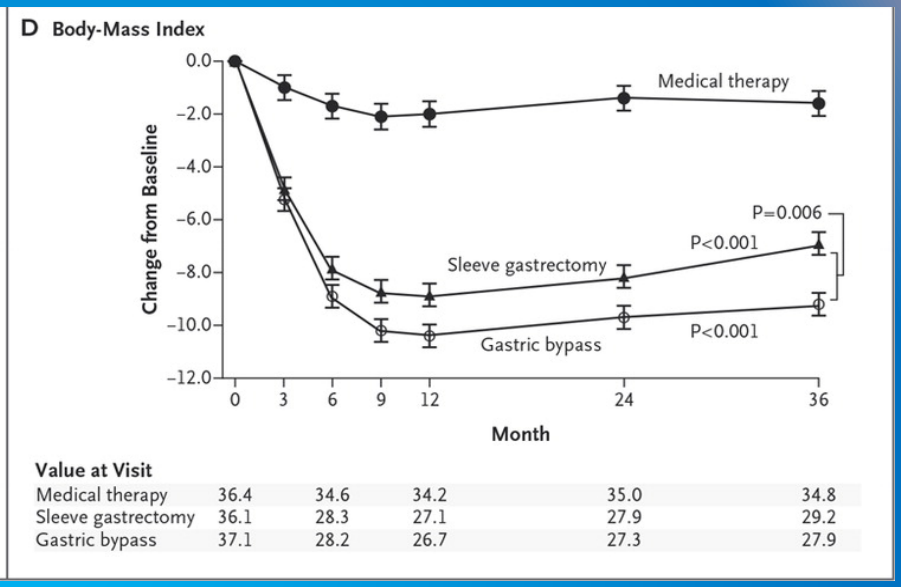


Bariatric Surgery *vs* Intensive Medical Therapy: Three year outcome data New Engl J Medicine 2014; 370: 2002-2013 *Schauer et al*



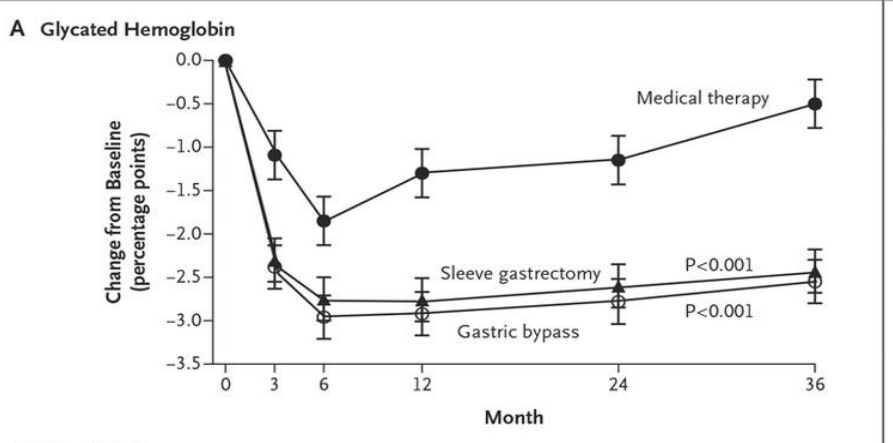
Value at Visit

Medical therapy	2.8	3.1	3.0	2.8	2.6
Sleeve gastrectomy	2.4	0.94	0.88	0.94	1.0
Gastric bypass	2.5	0.54	0.3	0.47	0.48



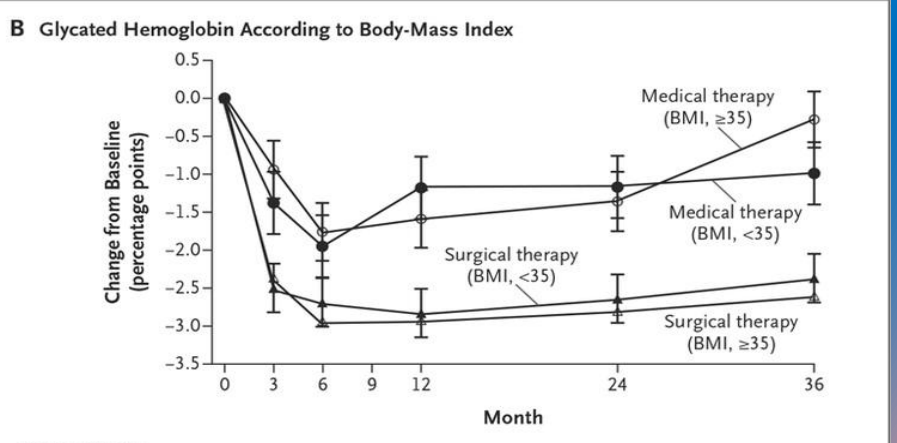
Value at Visit

Medical therapy	36.4	34.6	34.2	35.0	34.8
Sleeve gastrectomy	36.1	28.3	27.1	27.9	29.2
Gastric bypass	37.1	28.2	26.7	27.3	27.9



Value at Visit

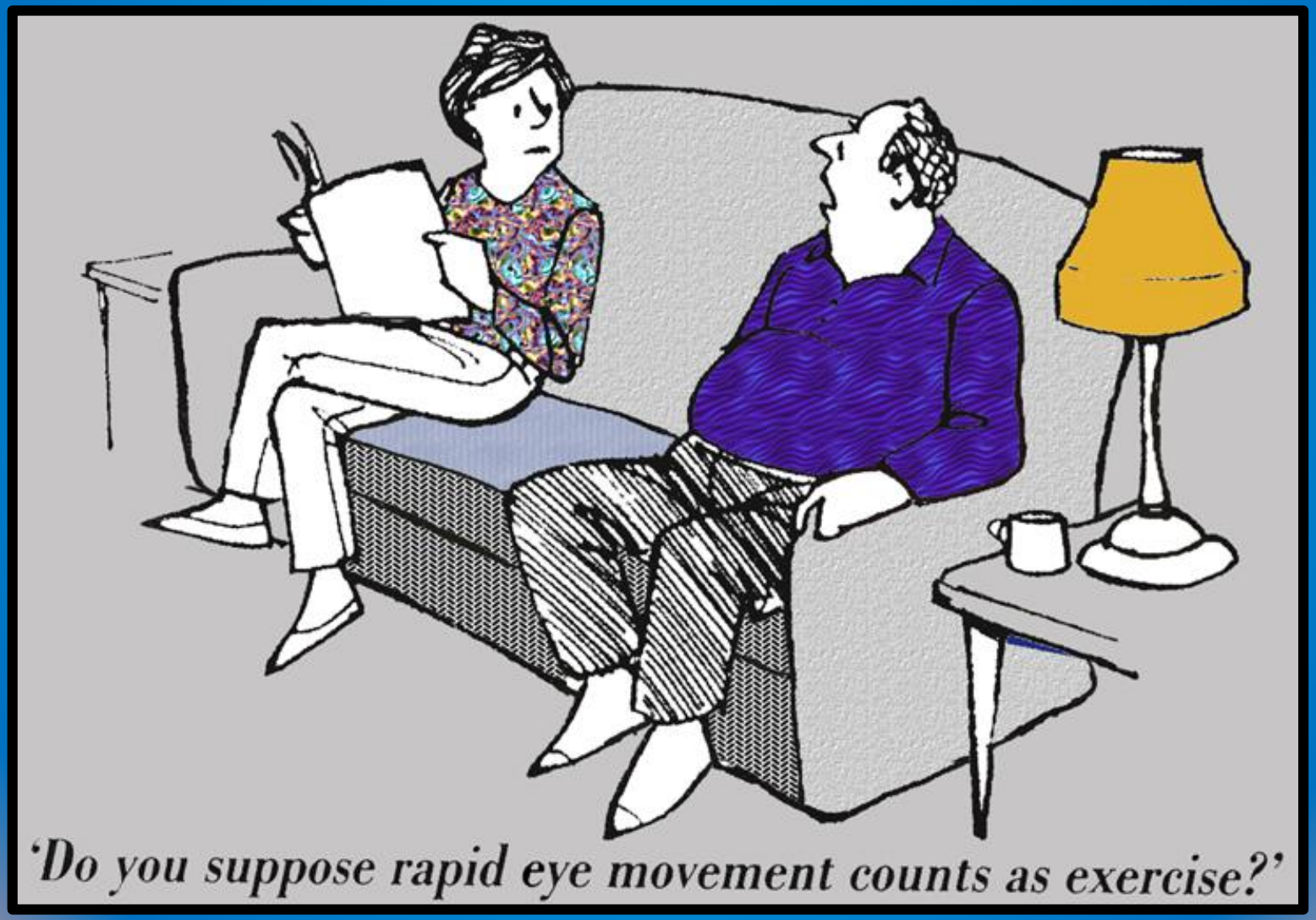
Medical therapy	9.0 (8.5)	7.1 (6.8)	7.5 (6.9)	7.7 (7.3)	8.4 (7.6)
Sleeve gastrectomy	9.5 (8.9)	6.7 (6.4)	6.6 (6.4)	6.8 (6.8)	7.0 (6.6)
Gastric bypass	9.3 (9.2)	6.3 (6.2)	6.3 (6.1)	6.5 (6.4)	6.7 (6.6)



Value at Visit

Medical <35 BMI	9.1 (8.9)	7.2 (6.8)	7.9 (6.9)	8.0 (7.4)	8.1 (7.8)
Medical ≥35 BMI	8.8 (8.5)	7.1 (6.8)	7.2 (6.7)	7.4 (6.9)	8.5 (7.3)
Surgical <35 BMI	9.4 (9.1)	6.7 (6.9)	6.6 (6.6)	6.8 (6.8)	7.1 (6.7)
Surgical ≥35 BMI	9.3 (9.2)	6.4 (6.2)	6.4 (6.1)	6.6 (6.4)	6.7 (6.4)

Conclusions



Health Cost Saving

- 5% weight reduction:
- BMI 30: R700/y
- BMI 35: R5280/y
- BMI 40: R20137/y

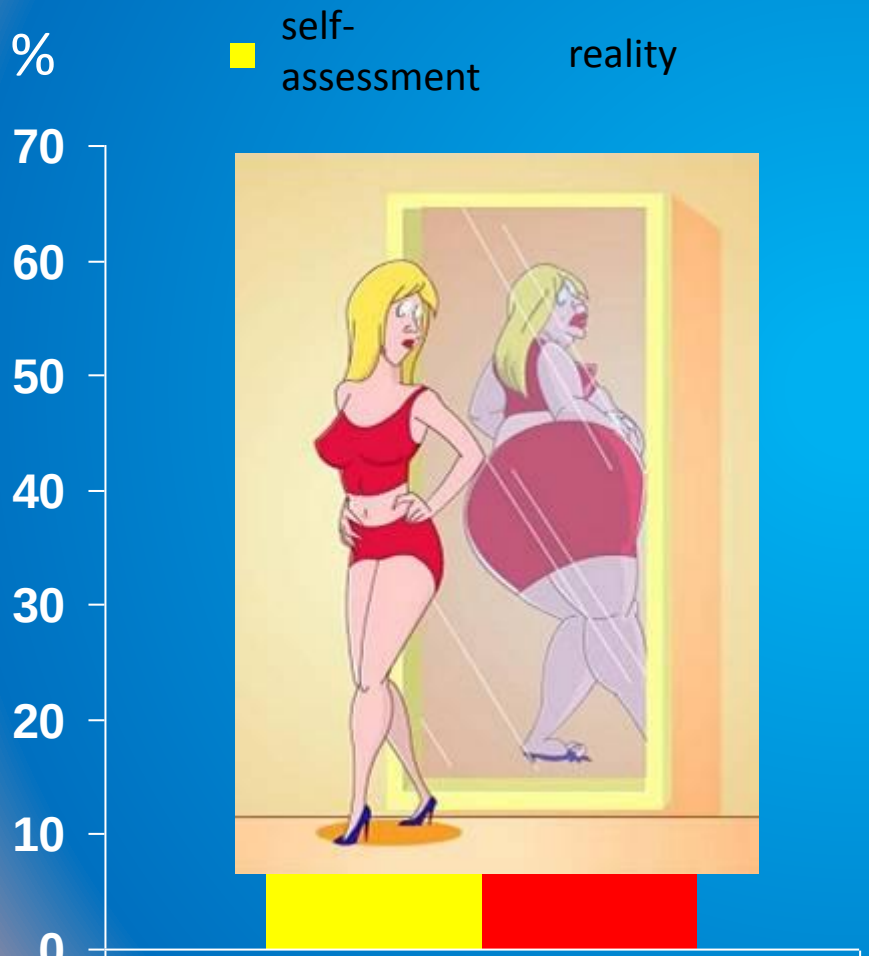
- To save $ 10 000 000/y
- Need to lower BMI by 5% in **14 500** people living with BMI at 30
- Need to lower BMI by 5% in only **100** people living with BMI at 40

OBESITY is very complex, and requires understanding

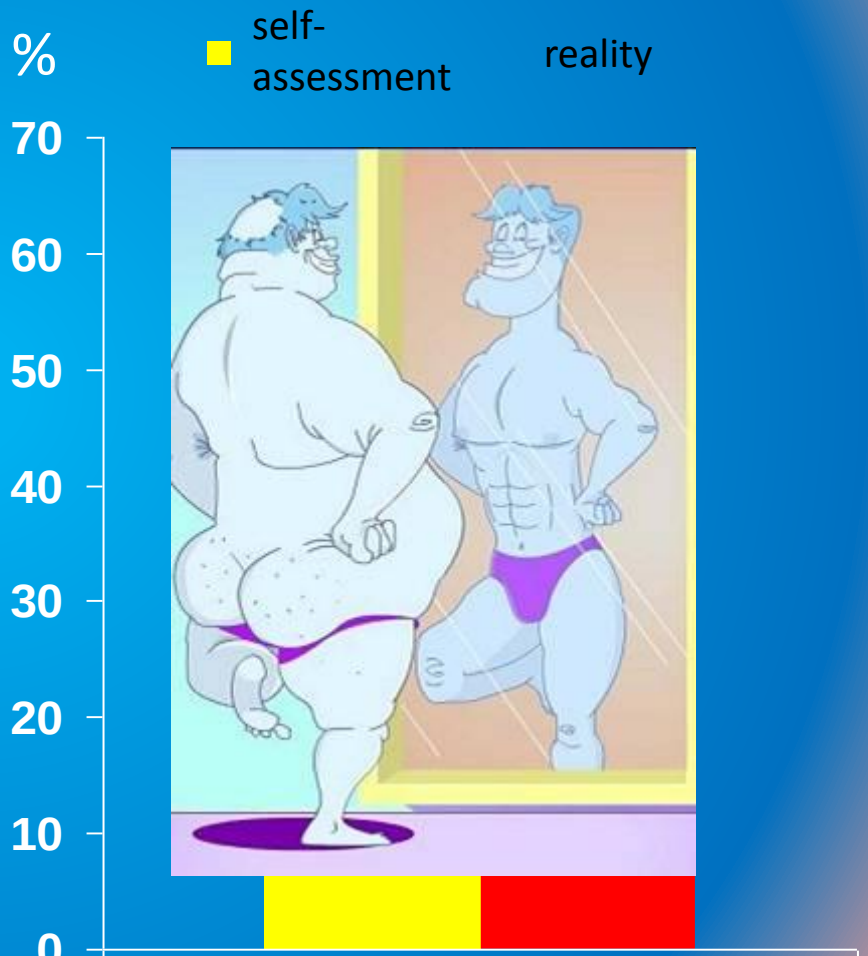
More needs to be done to debunk the myths and stereotypes that shape the attitudes of health professionals

Prevalence of overweight and obesity
Self-assessment vs. reality

NAT POL PLUS
2002



Females



Males