Childhood obesity

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Obesity as a Disease

Today, the AMA adopted policy that recognizes obesity as a disease requiring a range of medical interventions to advance obesity treatment and prevention.

"Recognizing obesity as a disease will help change the way the medical community tackles this complex issue that affects approximately one in three Americans," said AMA board member Patrice Harris, M.D. "The AMA is committed to improving health outcomes and is working to reduce the incidence of cardiovascular disease and type 2 diabetes, which are often linked to obesity."

Childhood obesity

1. Definition
2. Differential
3. Idiopathic obesity
   3.1 Epidemiology
4. Complications
5. Therapy
6. Society

Definition: Obese: BMI>95 pc
Overweight: BMI> 85 pc

Wall
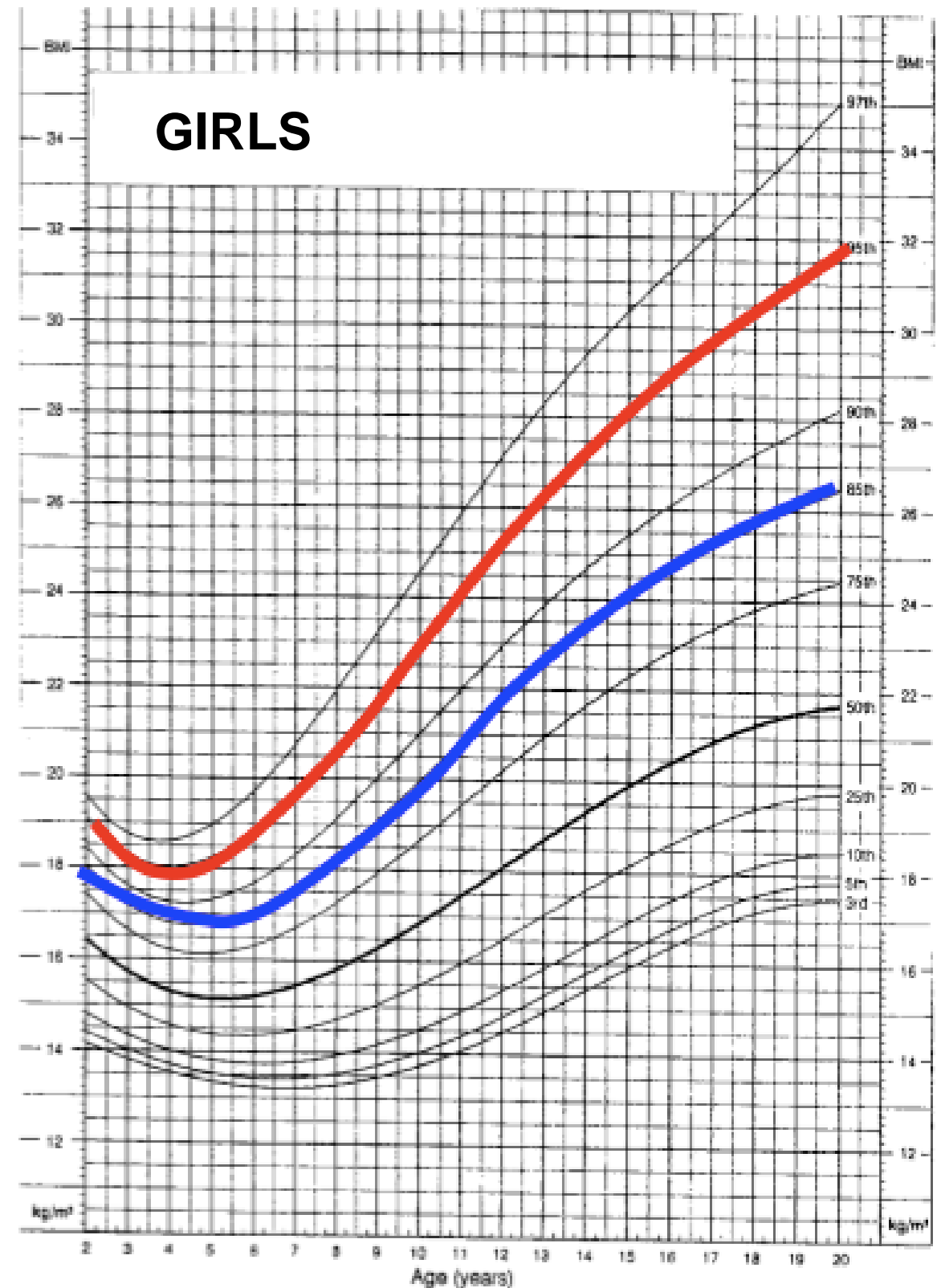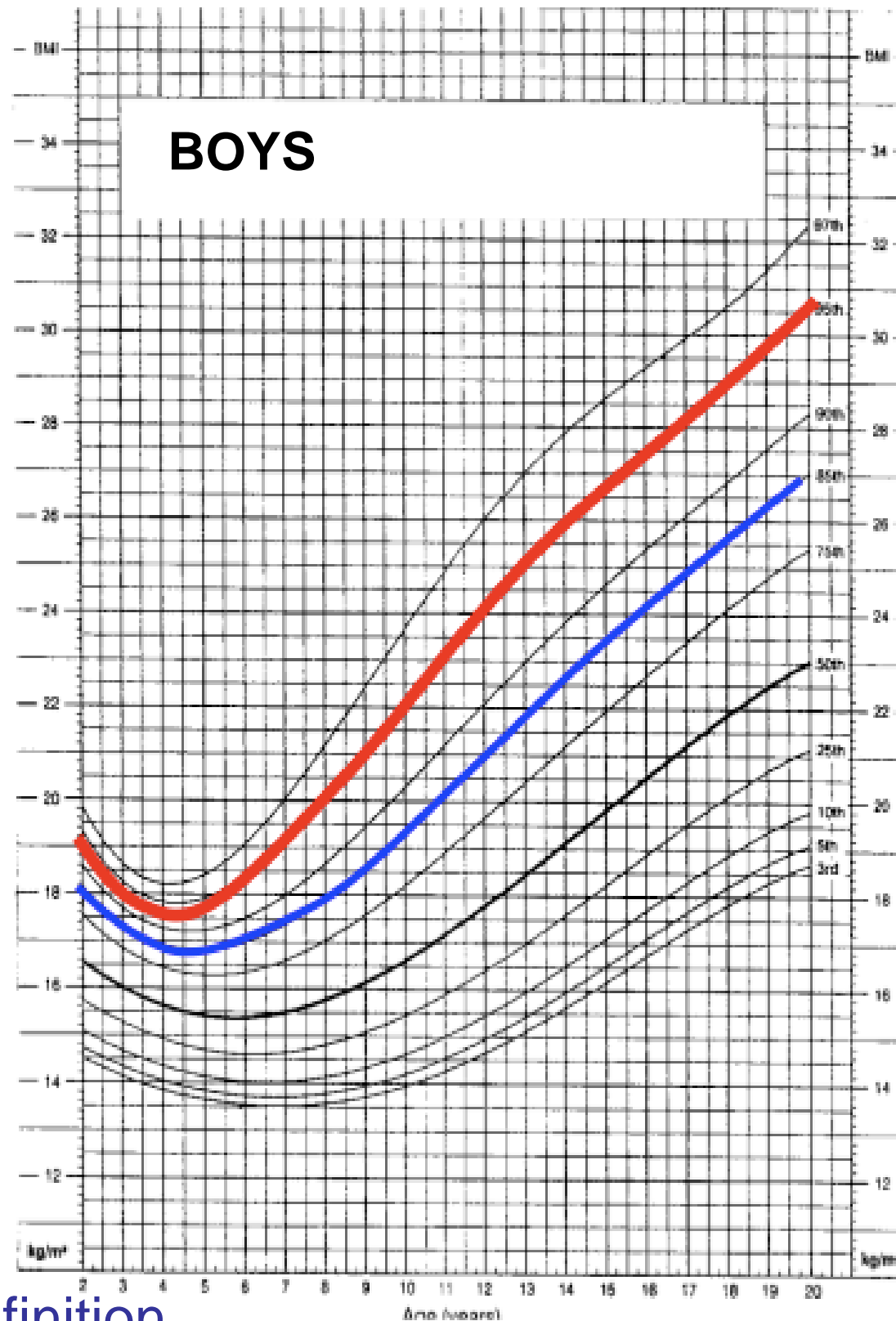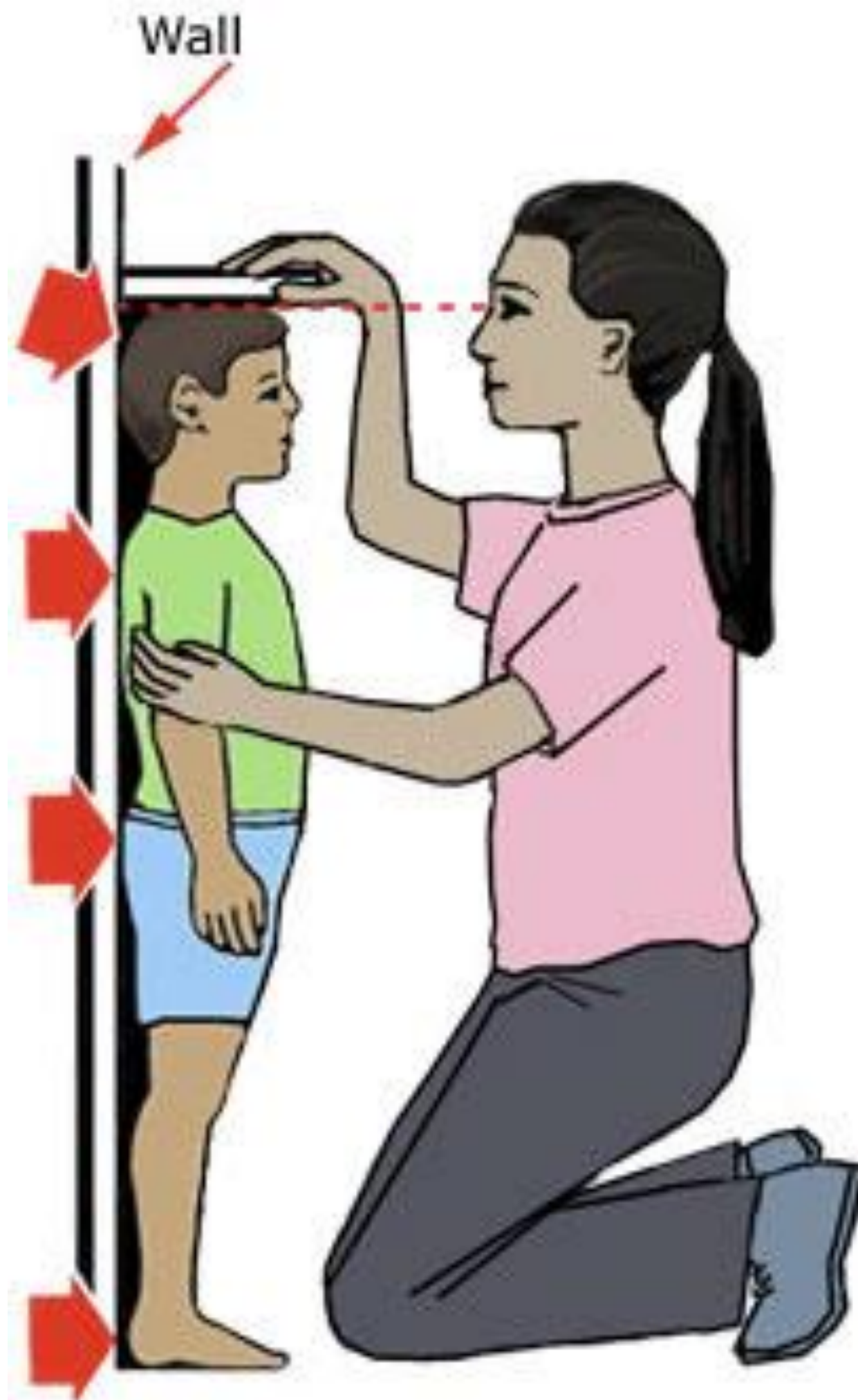
1. Definition

What causes obesity?

1. Genetics (extremely rare)

leptin gene
leptin receptor gene
POMC gene
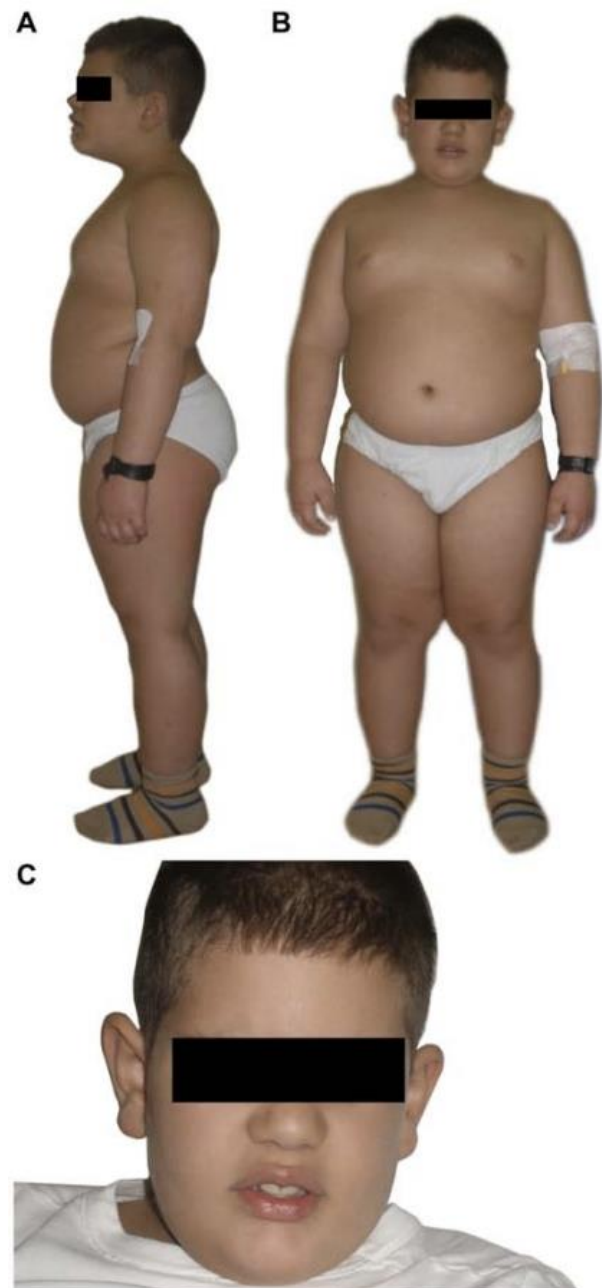melanocortin 4 receptor gene

2. Endocrine disorders (rare)

Hypothyroidism
Cushing syndrome
GH deficiency

3. Idiopathic (common)

2. Differential

Fig. 26.5. a Carpenter syndrome with lambdoidal craniosynostosis, low-set ears, short neck, obesity, down-slanting palpebral fissures and epicanthal folds. b Carpenter syndrome, with brachydactyly of the phalanges and partial soft tissue syndactyly. c Carpenter syndrome, with polysyndactyly of the feet.

Alström syndrome
Hypogonadism
Retinal degeneration
Deafness
Diabetes mellitus

Carpenter syndrome
Mental retadation
Polydactily, syndactily
Cranial synostosis

Laurence-Moon-Bardet-Biedl syndrome
Retinal degenaration
Syndactyly
Hypogonadism
Mental retardation

2. Differential

Down syndrome

Turner syndrome

Fröhlich syndrome
Disorder of hypothalamic
regulation
hypogonadism

2. Differential

Prader-Willi syndrome

Chromosome 15, uniparental dysomy
Complex hypothalamic disregulation: instable body
temperature, tantrums, apnoe, hypogonadism, overeating

Newborn:
Low birth weight
Low muscle tone ⟶ *Growth hormone treatment*
Failure to thrive *from 6 mo* increases
Cryptorchidism muscle tone and muscle/fat
Small hands and feet ratio

Rare causes: endocrine disorders

HYPOTHYROIDISM

Intolerance to Cold
Receding Hairline
Facial & Eyelid Edema
Dull-Blank Expression
Extreme Fatigue
Thick Tongue - Slow Speech
Anorexia
Brittle Nails & Hair
Menstrual Disturbances

Hair Loss
Apathy
Lethargy
Dry Skin (Coarse & Scaly)
Muscle Aches & Weakness
Constipation

Late Clinical Manifestations
Subnormal Temp
Bradycardia
Weight Gain
↓LOC
Thickened Skin
Cardiac Complications

Hypothyroidism:
small weight gain
growth retardation
cold intolerance, fatigue, jaundice, constipation, etc

Signs and symptoms of Cushing syndrome
Round face
Hump
Hirsutism
Easy bruising
inal weight gain
Stretch marks

Cushing syndrome (cortisol overproduction):
sudden, significant weight gain
growth retardation
hypertension, diabetes, hypokalemia, etc

GHD
Children of the same age
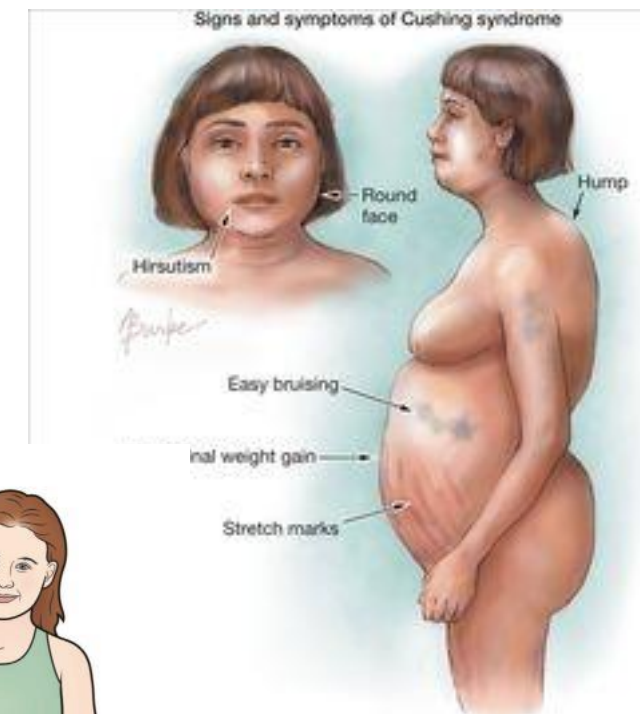
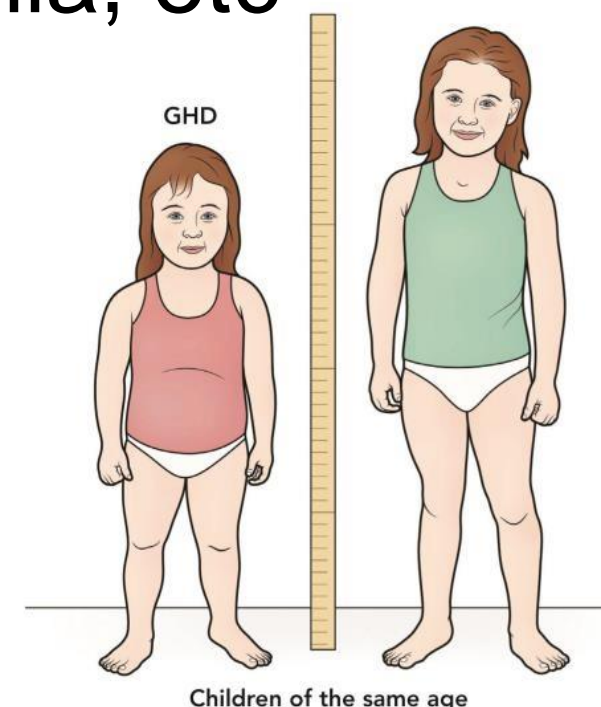**Growth hormone deficiency:**
growth retardation
minimal weight gain,
decreased muscle/fat ratio

Common: idiopathic obesity

"EVOLUTION"
©2004 CAROL LAY

3. Idiopathic
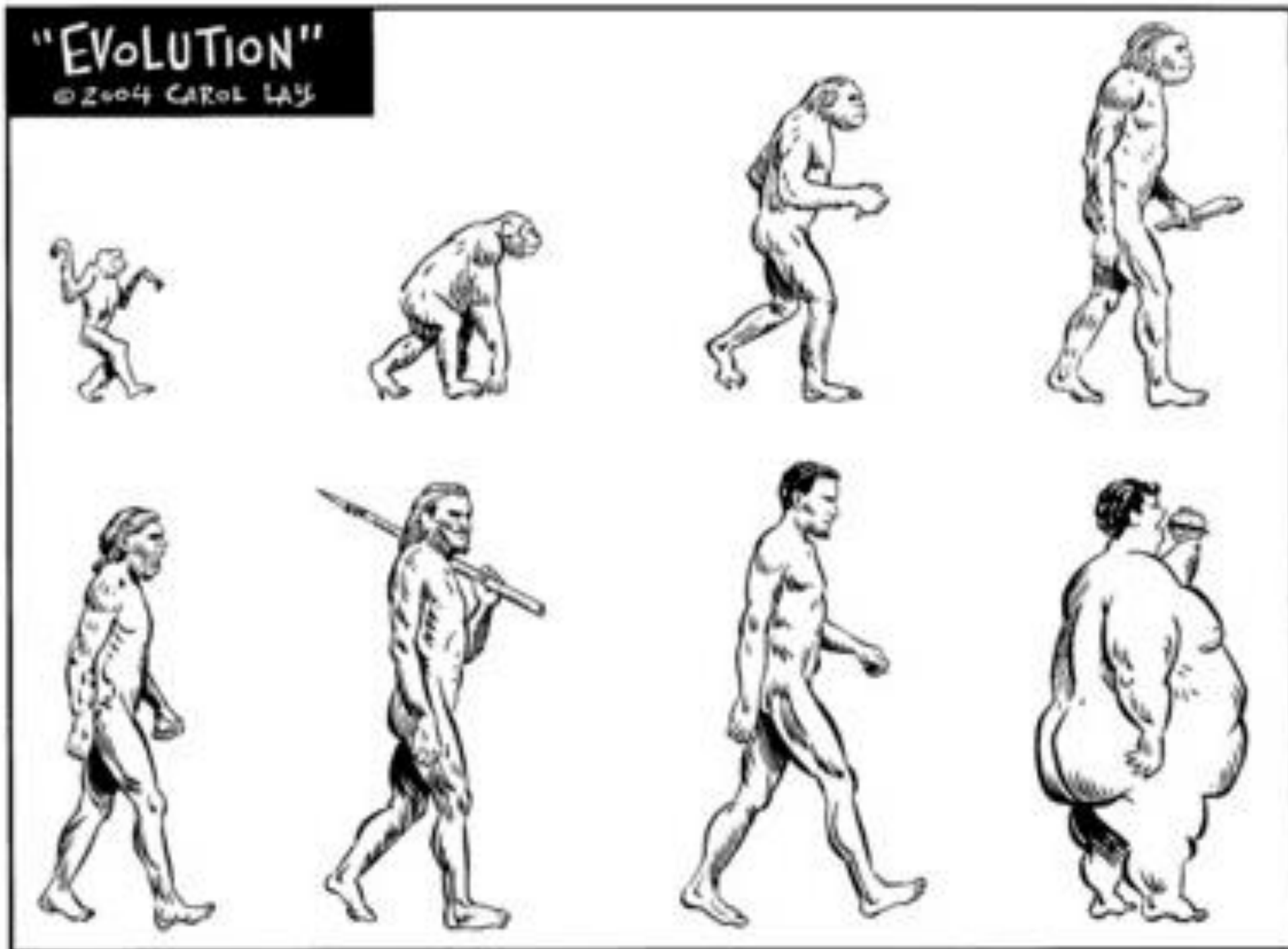
How frequent?

- Worldwide 1,5 billion overweight adults
- Younger than 5 five years: *42 million*, 35 million in developing countries

World Health Organization

Figure 1. Obesity among adults, 2012 or nearest year

3.1Epidemiology
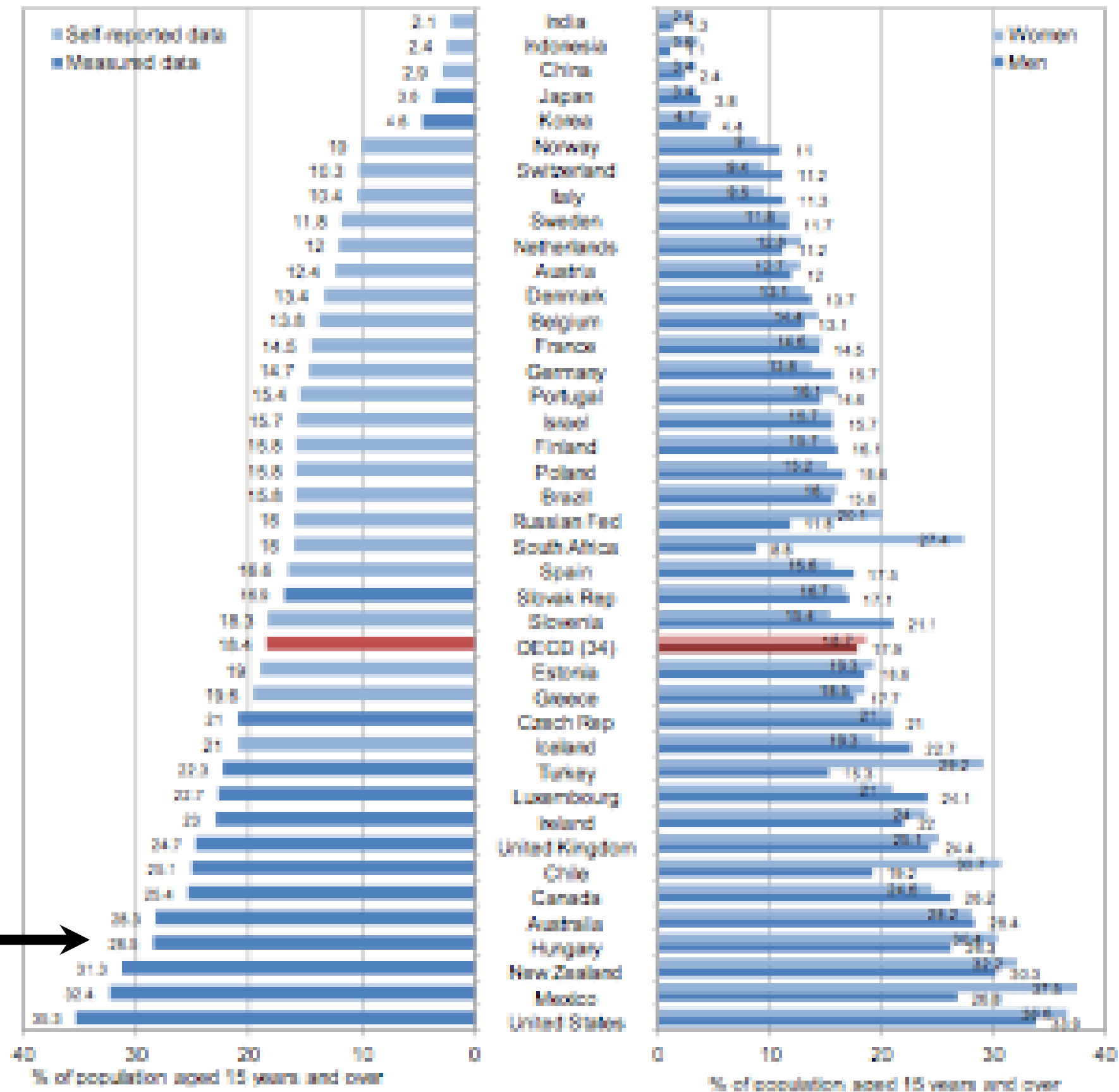
Figure 2.4.1 **Children aged 5-17 years who are overweight (including obese), latest available estimates**

	Girls		Boys
Greece	37.0	45.0	
United States	35.9	35.0	
Italy	30.9	32.4	
Mexico	29.0	28.1	
New Zealand	28.8	28.2	
Chile	27.1	28.6	
United Kingdom	26.6	22.7	
Canada	26.1	28.9	
Hungary	25.9	25.5	
Iceland	25.5	22.0	
Slovenia	24.4	28.7	
Australia	24.0	22.0	
Spain	22.9	32.9	
Portugal	21.6	23.5	
OECD	21.4	22.9	
Brazil	21.1	23.1	
Russian Federation	19.8	24.2	
Sweden	19.5	17.0	
Finland	19.1	23.6	
India	18.3	20.6	
Netherlands	17.9	14.7	
South Africa	17.7	13.6	
Germany	17.6	22.6	
Czech Republic	16.9	24.6	
Slovak Republic	16.2	17.5	
Denmark	15.2	14.1	
France	14.9	13.1	
Norway	14.7	12.9	
Japan	14.4	16.2	
Switzerland	13.1	16.7	
Poland	12.4	16.3	
Turkey	10.3	11.3	
Korea	9.9	16.2	
China	4.5	5.9	

% of children aged 5-17 years

Source: International Association for the Study of Obesity (2011).

3.1 Epidemiology
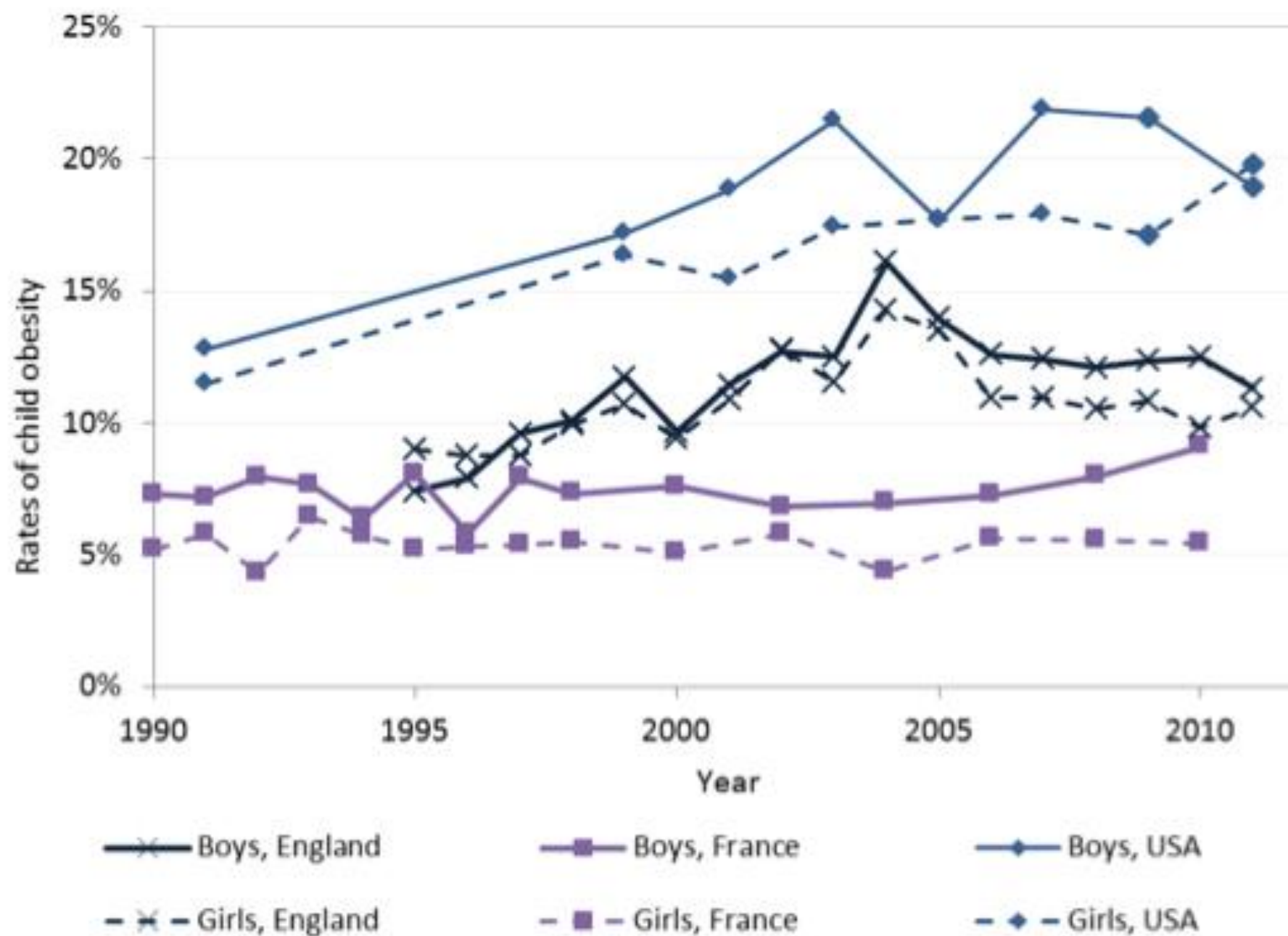
Worldwide trends in childhood obesity
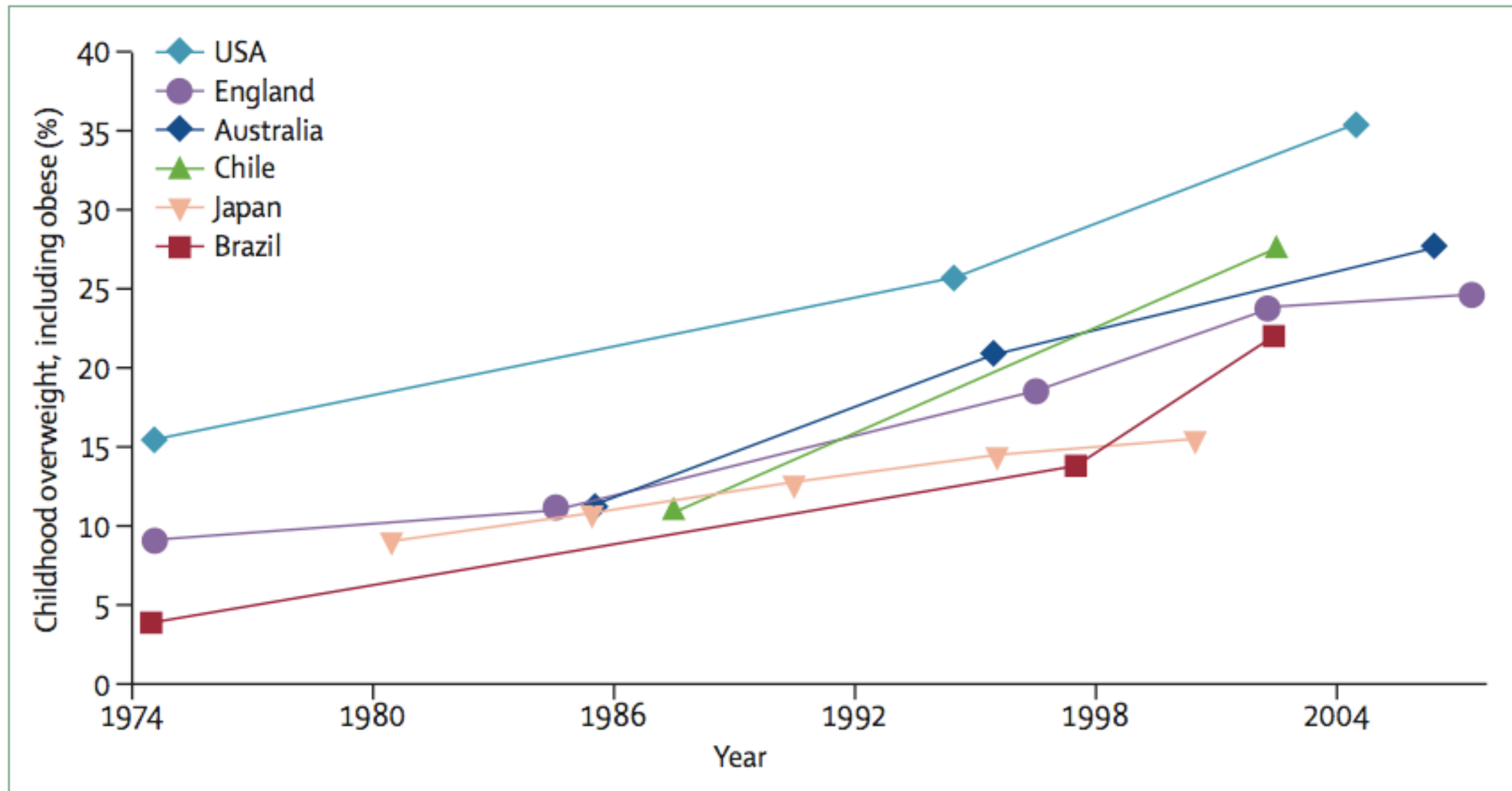
Figure 1: Estimates of percentage of childhood population overweight, including obese (with use of International Obesity Taskforce cutoffs) in a selection of countries

Based on data from Wang and Lobstein,[11] International Association for the Study of Obesity,[12] and Matsushita and colleagues.[13]
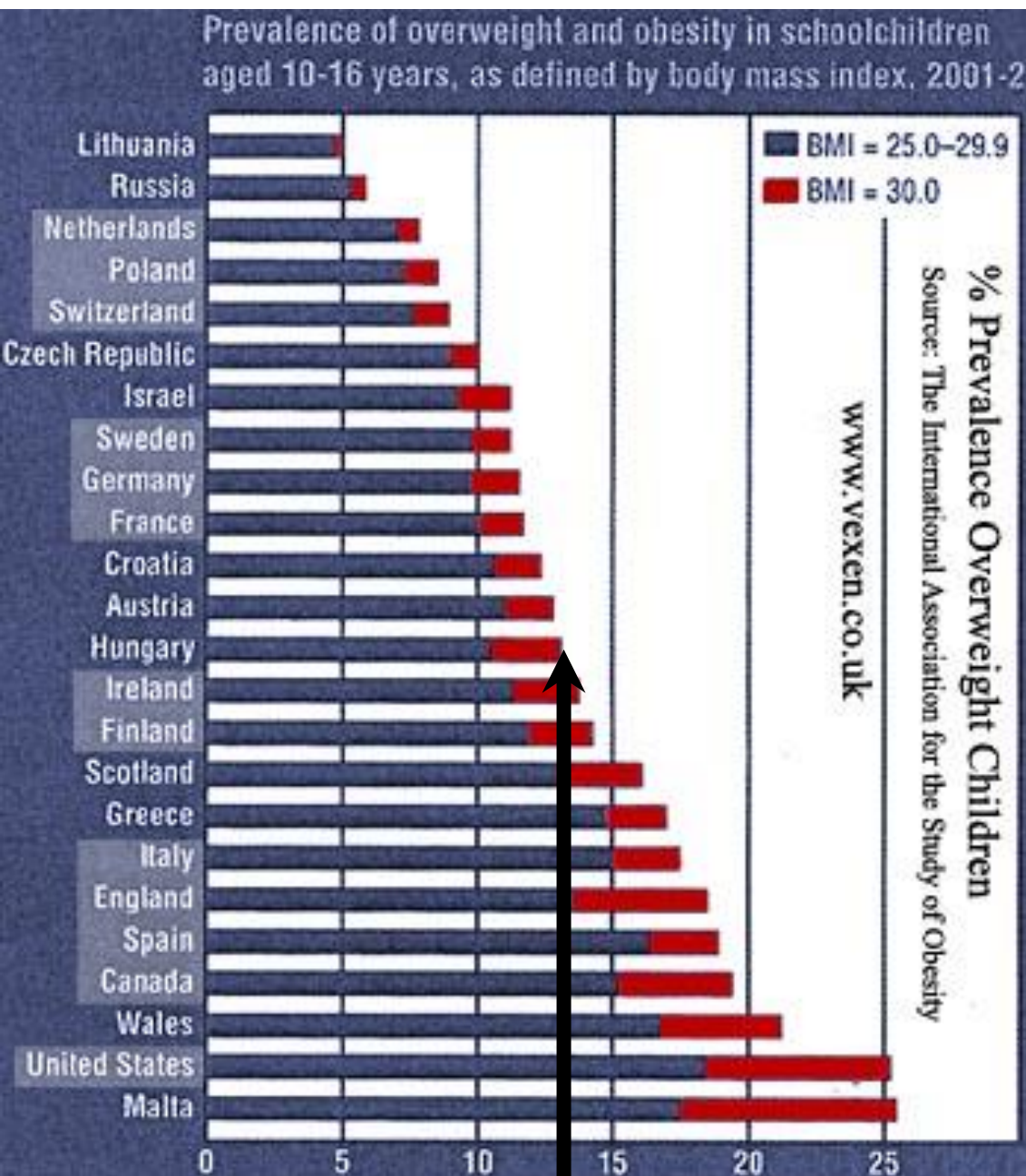
3.1Epidemiology

Figure 3. Trends in child obesity, age 3-17
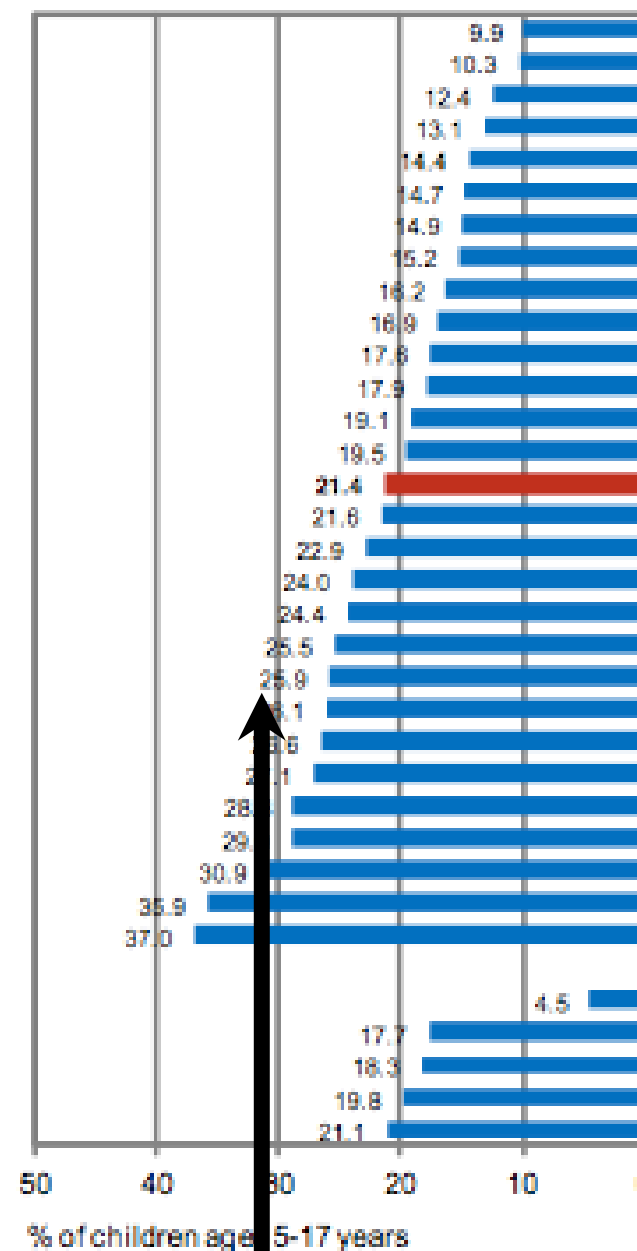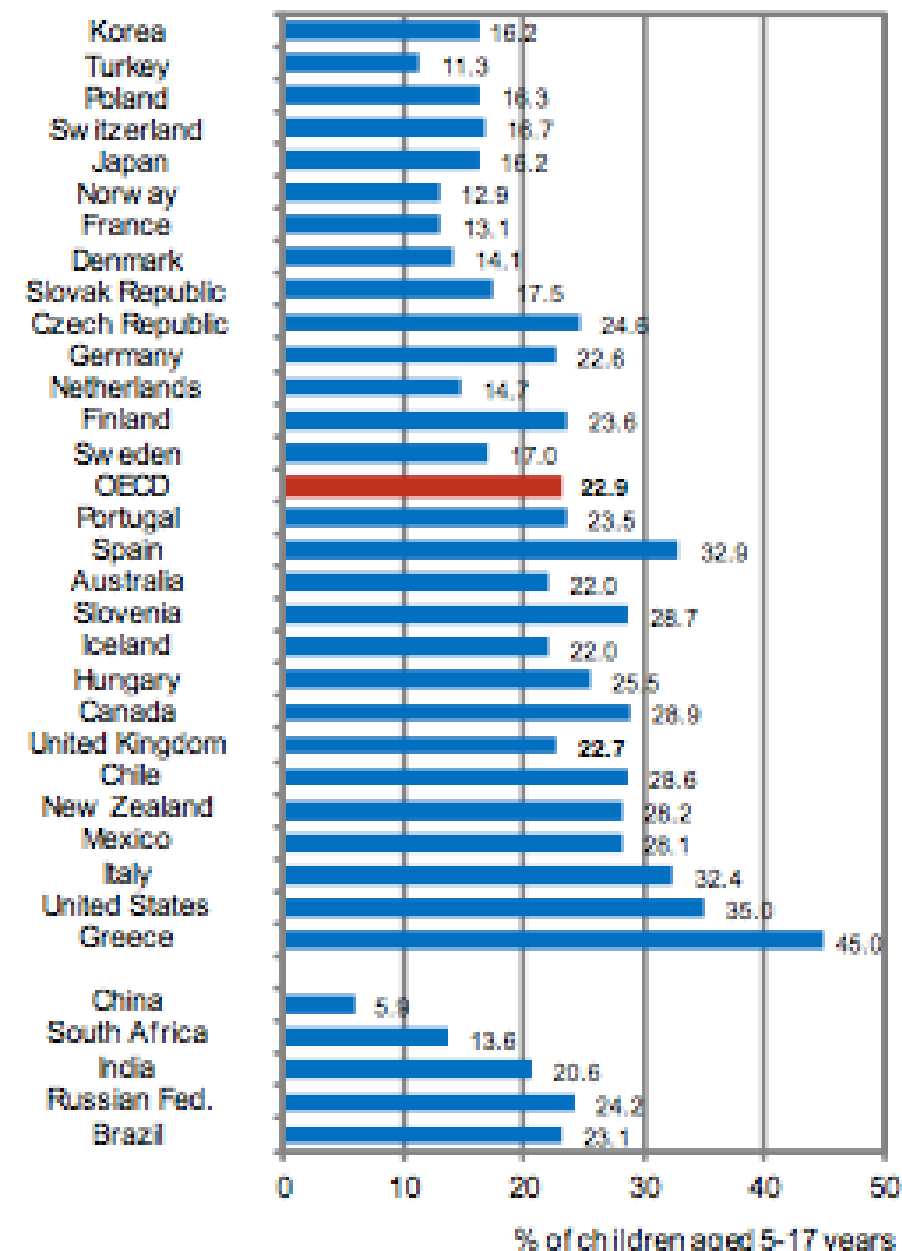
Childhood obesity trends in Hungary

2001

2012

Children aged 5-17 years who are overweight (including obese), latest available estimates

Girls **Boys**

Prevalence of overweight and obesity in schoolchildren aged 10-16 years, as defined by body mass index, 2001-2

BMI = 25.0–29.9
BMI = 30.0

% Prevalence Overweight Children

Source: The International Association for the Study of Obesity

www.vexen.co.uk

13%

25%

% of children aged 5-17 years

% of children aged 5-17 years

Source: International Association for the Study of Obesity (2011).

Statlink: http://dx.doi.org/10.1787/88893252399

3.1Epidemiology

1977   **3x**   **2000**

1977 Weight-for-age percentiles
Girls: birth to 36 months

Revised Weight-for-age percentiles:
Girls, birth to 36 months
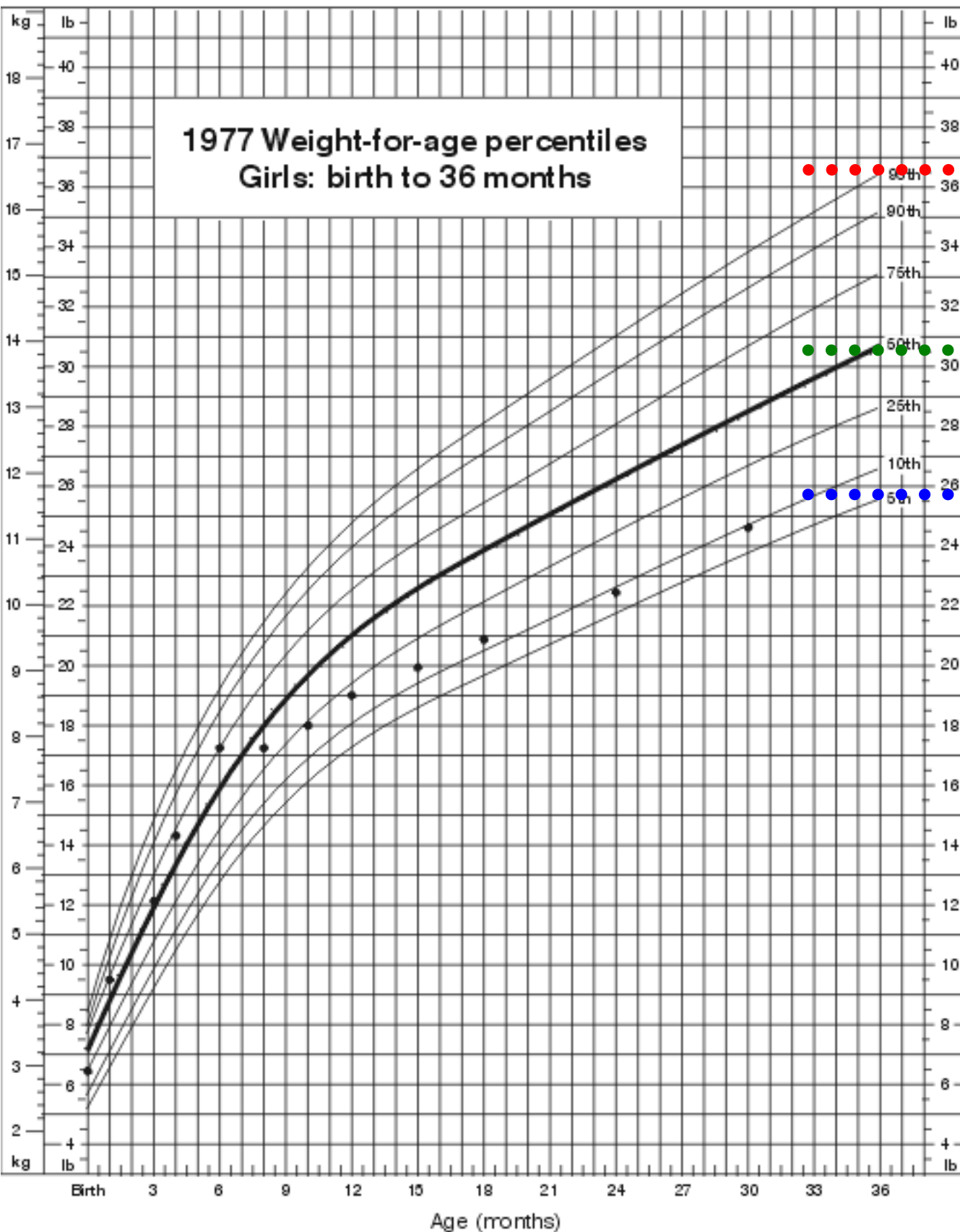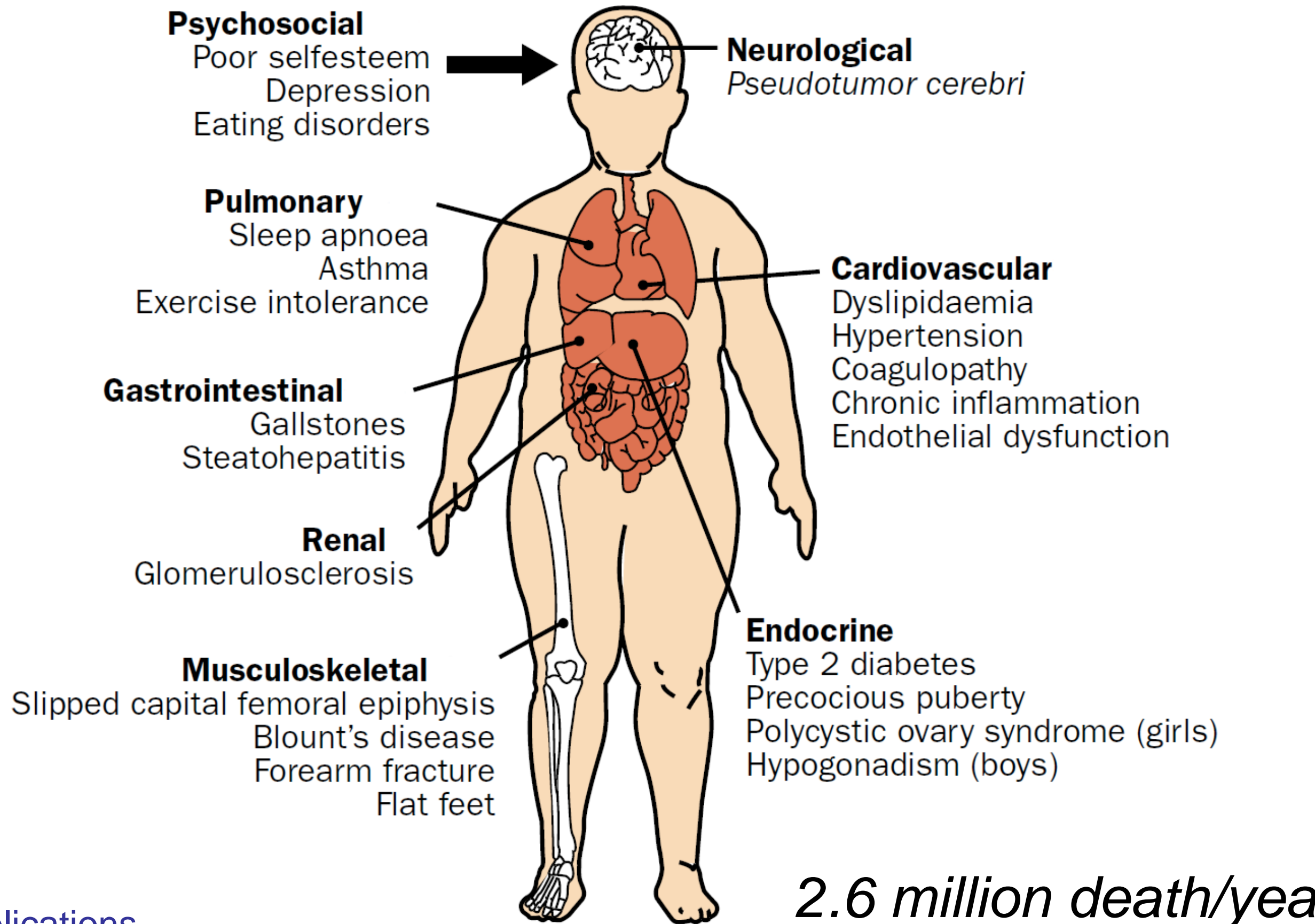
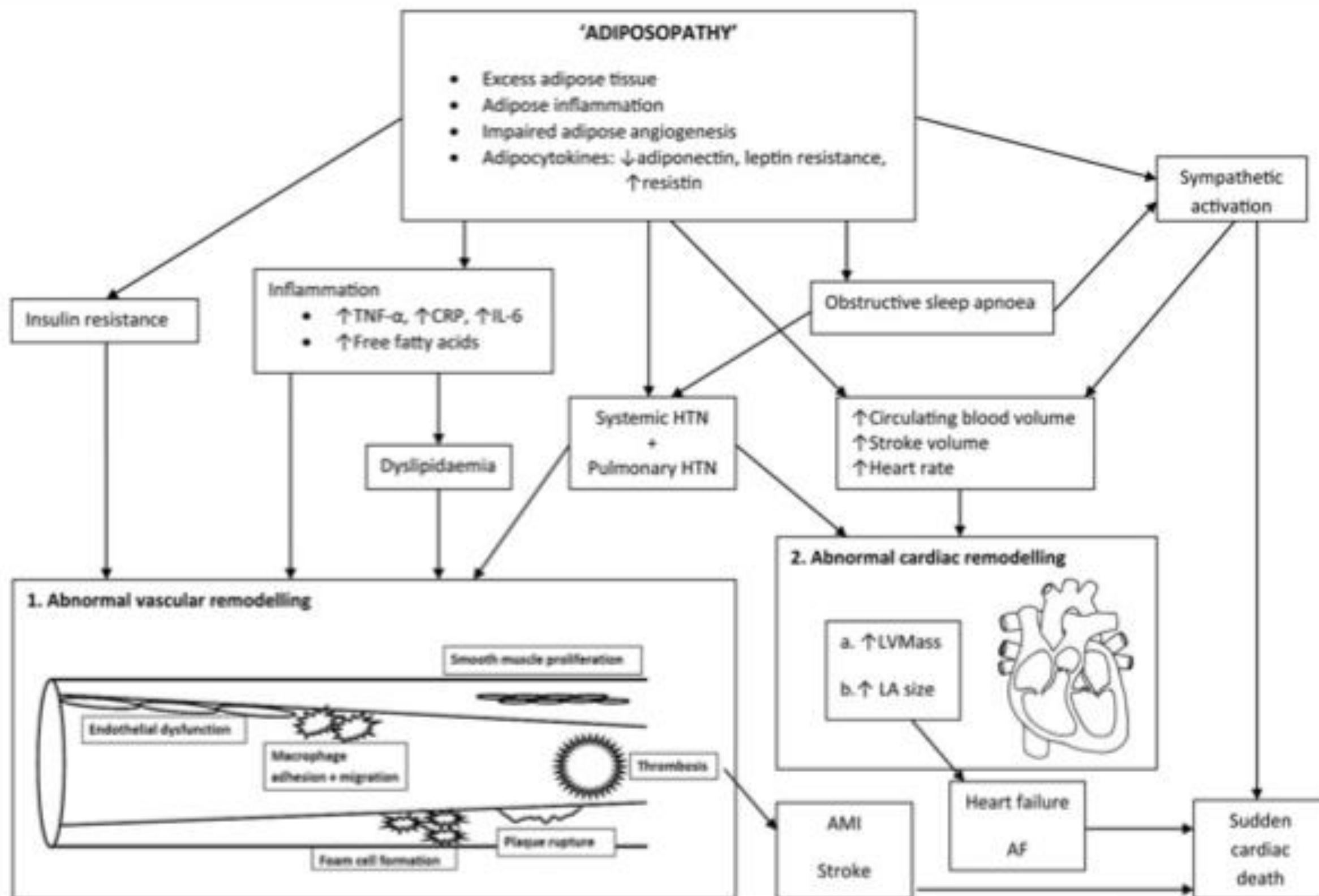# Do obese children become obese adults? – most likely

- 26-41% of overweight preschoolers

- 42-63% of overweight school children become obese adults

- Obese kids have a double chance to grow up to be obese adults than normal kids

COMPLICATIONS OF CHILDHOOD OBESITY

Psychosocial
Poor selfesteem
Depression
Eating disorders

Neurological
Pseudotumor cerebri

Pulmonary
Sleep apnoea
Asthma
Exercise intolerance

Cardiovascular
Dyslipidaemia
Hypertension
Coagulopathy
Chronic inflammation
Endothelial dysfunction

Gastrointestinal
Gallstones
Steatohepatitis

Renal
Glomerulosclerosis

Endocrine
Type 2 diabetes
Precocious puberty
Polycystic ovary syndrome (girls)
Hypogonadism (boys)

Musculoskeletal
Slipped capital femoral epiphysis
Blount's disease
Forearm fracture
Flat feet

2.6 million death/yea

4. Complications

'ADIPOSOPATHY'

- Excess adipose tissue
- Adipose inflammation
- Impaired adipose angiogenesis
- Adipocytokines: ↓adiponectin, leptin resistance, ↑resistin

Sympathetic activation

Insulin resistance

Inflammation
- ↑TNF-α, ↑CRP, ↑IL-6
- ↑Free fatty acids

Obstructive sleep apnoea

Systemic HTN + Pulmonary HTN

↑Circulating blood volume
↑Stroke volume
↑Heart rate

Dyslipidaemia

1. Abnormal vascular remodelling

Smooth muscle proliferation

Endothelial dysfunction

Macrophage adhesion + migration

Thrombosis

Foam cell formation

Plaque rupture

2. Abnormal cardiac remodelling

a. ↑LVMass

b. ↑ LA size

Heart failure

AF

AMI

Stroke

Sudden cardiac death

4. Complications

4. Complications

Size (and time) matter:
Continuous overweight since childhood increases the risk of complications in adulthood

Associations between patterns of overweight in childhood, adolescence and adulthood and cardiovascular outcomes in three British birth cohorts, from logistic regression analyses.

Overweight pattern	Weight status[*]			n	Adult BMI Mean (SD)	Hypertension OR (95% CI)	Type 2 diabetes OR (95% CI)	CHD OR (95% CI)
	Childhood	Adolescence	Adulthood					
Never overweight	0	0	0	8,587	24.4 (2.79)	1	1	1
Childhood only	1	0	0	374	25.3 (2.53)	0.87 (0.54 to 1.40)	0.99 (0.35 to 2.80)	0.44 (0.20 to 1.89)
Adolescence only	0	1	0	397	26.5 (2.38)	0.97 (0.61 to 1.55)	0.88 (0.31 to 2.50)	1.63 (0.37 to 7.19)
Adulthood only	0	0	1	1,144	33.3 (4.64)	2.28 (1.76 to 2.95)	5.47 (3.39 to 8.82)	3.83 (1.98 to 7.42)
Childhood+adolescence	1	1	0	161	26.4 (2.20)	1.01 (0.46 to 2.21)	1.24 (0.29 to 5.25)	3.43 (0.60 to 19.64)
Childhood+adulthood	1	0	1	130	34.0 (4.78)	2.91 (1.54 to 5.49)	4.70 (1.89 to 11.67)	1.10 (0.14 to 8.48)
Adolescence+adulthood	0	1	1	388	35.0 (4.61)	3.01 (2.11 to 4.29)	6.61 (3.61 to 12.09)	3.74 (1.35 to 10.35)
Persistent overweight[†]	1	1	1	266	36.4 (4.82)	2.56 (1.40 to 4.68)	12.60 (6.61 to 23.98)	6.62 (1.94 to 22.65)

Park MH, Sovio U, Viner RM, Hardy RJ, Kinra S. Overweight in childhood, adolescence and adulthood and cardiovascular risk in later life: pooled analysis of three british birth cohorts. PLoS One. 2013 Jul 24;8(7):e70684.

4. Complications

TABLE 2. For males measured in 1963–1975, relative risks of death (cause-specific and overall mortality) with 95% confidence intervals, adjusted for age and birth year, by body mass index in adolescence, Norway

| Cause of death | Body mass index category ((weight in kg)/(height in meters)^2) in adolescence* | | | | | | | | | | | p value† |
| | <25th (942,467 person-years) | | | 25th–74th (2,539,557 person-years)‡ | | 75th–84th (307,375 person-years) | | | ≥85th (207,816 person-years) | | | |
	No. of deaths	RR§	95% CI§	No. of deaths	RR	No. of deaths	RR	95% CI	No. of deaths	RR	95% CI	
Malignant neoplasms	265	0.9	0.8, 1.0	836	1.0	101	1.0	0.8, 1.2	77	1.2	0.9, 1.5	0.2
Colon	21	1.0	0.6, 1.6	59	1.0	7	1.0	0.4, 2.2	10	2.1	1.1, 4.1	0.1
Larynx and trachea/bronchus/lung	57	1.1	0.8, 1.5	144	1.0	16	0.9	0.6, 1.5	13	1.2	0.7, 2.0	0.5
Lymphatic/hematopoietic tissue	57	1.1	0.8, 1.5	139	1.0	15	0.9	0.5, 1.5	13	1.2	0.7, 2.1	0.3
Endocrine, nutritional, and metabolic diseases	38	1.4	1.0, 2.1	68	1.0	19	2.4	1.4, 4.0	13	2.5	1.4, 4.5	0.5
Diabetes mellitus	32	1.7	1.1, 2.6	49	1.0	12	2.1	1.1, 4.0	7	1.9	0.8, 4.1	0.1
Mental and behavioral disorders	67	1.2	0.9, 1.6	197	1.0	17	0.7	0.4, 1.2	12	0.7	0.4, 1.3	0.05
Diseases of the circulatory system	240	1.0	0.8, 1.1	660	1.0	139	1.8	1.5, 2.1	129	2.5	2.1, 3.0	<0.001
Ischemic heart diseases	149	1.0	0.8, 1.2	411	1.0	89	1.8	1.5, 2.3	93	2.9	2.3, 3.6	<0.001
Other heart diseases	24	0.8	0.5, 1.2	62	1.0	14	1.4	0.8, 2.5	12	1.9	1.0, 3.4	0.2
Cerebrovascular diseases	52	1.2	0.9, 1.7	115	1.0	17	1.2	0.7, 2.0	18	1.9	1.2, 3.2	1.0
Diseases of the respiratory system	30	1.5	1.0, 2.4	52	1.0	7	1.1	0.5, 2.5	11	2.7	1.4, 5.2	0.1
Chronic lower respiratory diseases	13	1.4	0.7, 2.8	25	1.0	4	1.3	0.5, 3.8	8	4.1	1.8, 9.0	0.6
Digestive system diseases	64	1.2	0.9, 1.6	144	1.0	16	0.9	0.5, 1.5	16	1.4	0.8, 2.3	0.1
Chronic liver disease	46	1.3	0.9, 1.8	99	1.0	13	1.1	0.6, 1.9	10	1.3	0.7, 2.4	0.3
Symptoms, signs, abnormal findings, ill-defined causes	41	0.7	0.5, 1.1	147	1.0	24	1.4	0.9, 2.1	20	1.7	1.1, 2.7	0.02
Sudden death	16	0.7	0.4, 1.2	62	1.0	6	1.1	0.5, 2.3	11	2.2	1.2, 4.3	0.003
External causes of injury and poisoning	574	1.0	0.9, 1.1	1,526	1.0	172	0.9	0.8, 1.1	137	1.1	0.9, 1.3	0.9
Suicide and intentional self-harm	186	1.1	0.9, 1.3	454	1.0	62	1.1	0.9, 1.5	37	1.0	0.7, 1.4	0.7
Other	107	1.0	0.8, 1.3	265	1.0	33	1.0	0.7, 1.4	29	1.3	0.9, 1.9	0.6
Total	1,466	1.0	0.9, 1.1	3,915	1.0	526	1.1	1.0, 1.2	444	1.4	1.3, 1.6	0.006

* Categorized according to percentiles in a US reference population (18).

† p value for test of linear trend using body mass index in eight categories (<3rd, 3rd–4th, 5th–9th, 10th–24th, 25th–74th, 75th–84th, 85th–94th, ≥95th) as a continuous variable.

‡ Reference category.

§ RR, relative risk; CI, confidence interval.

Body Mass Index in Adolescence in Relation to Cause-specific Mortality: A Follow-up of 230,000 Norwegian Adolescents

10% weight loss:

- -10 Hgmm BP
- Fasting glucose -50% in T2DM
- -10% cholesterol

- -20% all cause mortality
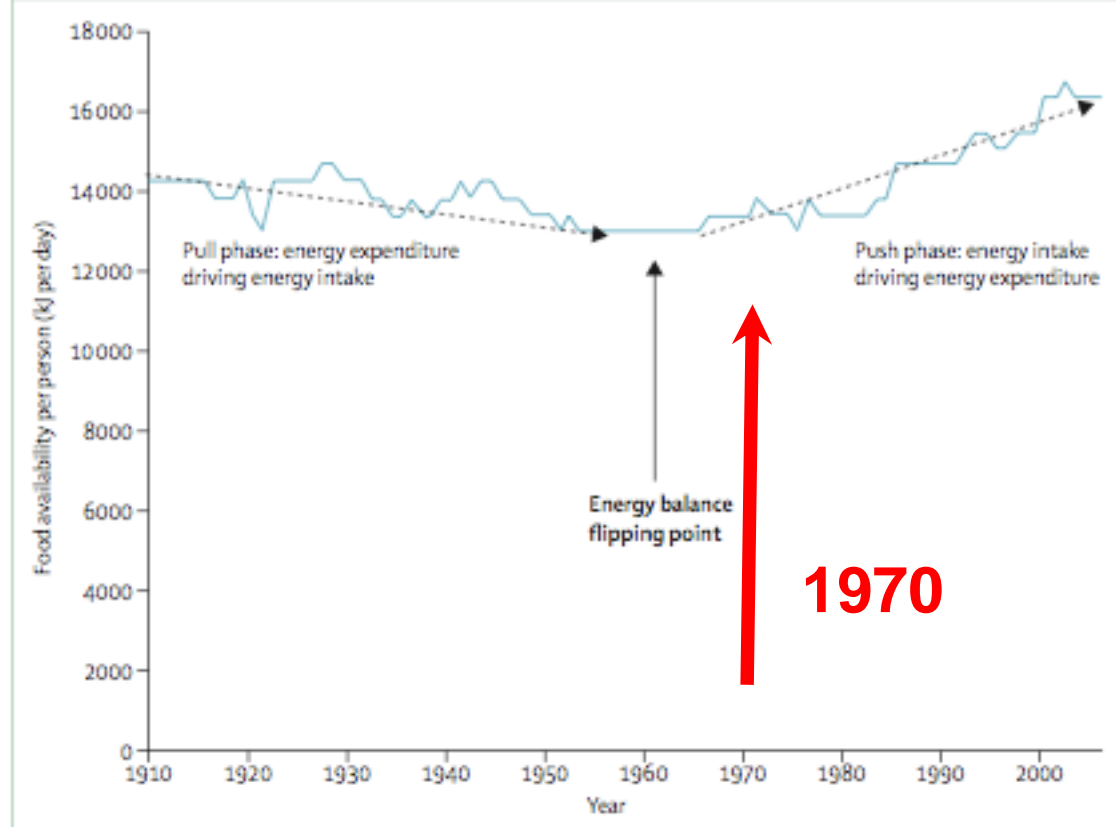- -30% diabetes related mortality
- -40% obesity related mortality

Figure 3: Food availability for the USA, 1910–2006
There are two distinct phases: a decrease in food energy supply (postulated to be pulled down by reduced energy expenditure requirements for daily living), followed by an increase in food energy supply (postulated to be pushed up by increasing food access). An energy balance flipping point is proposed, marking the change in how the US population generally achieved energy balance.
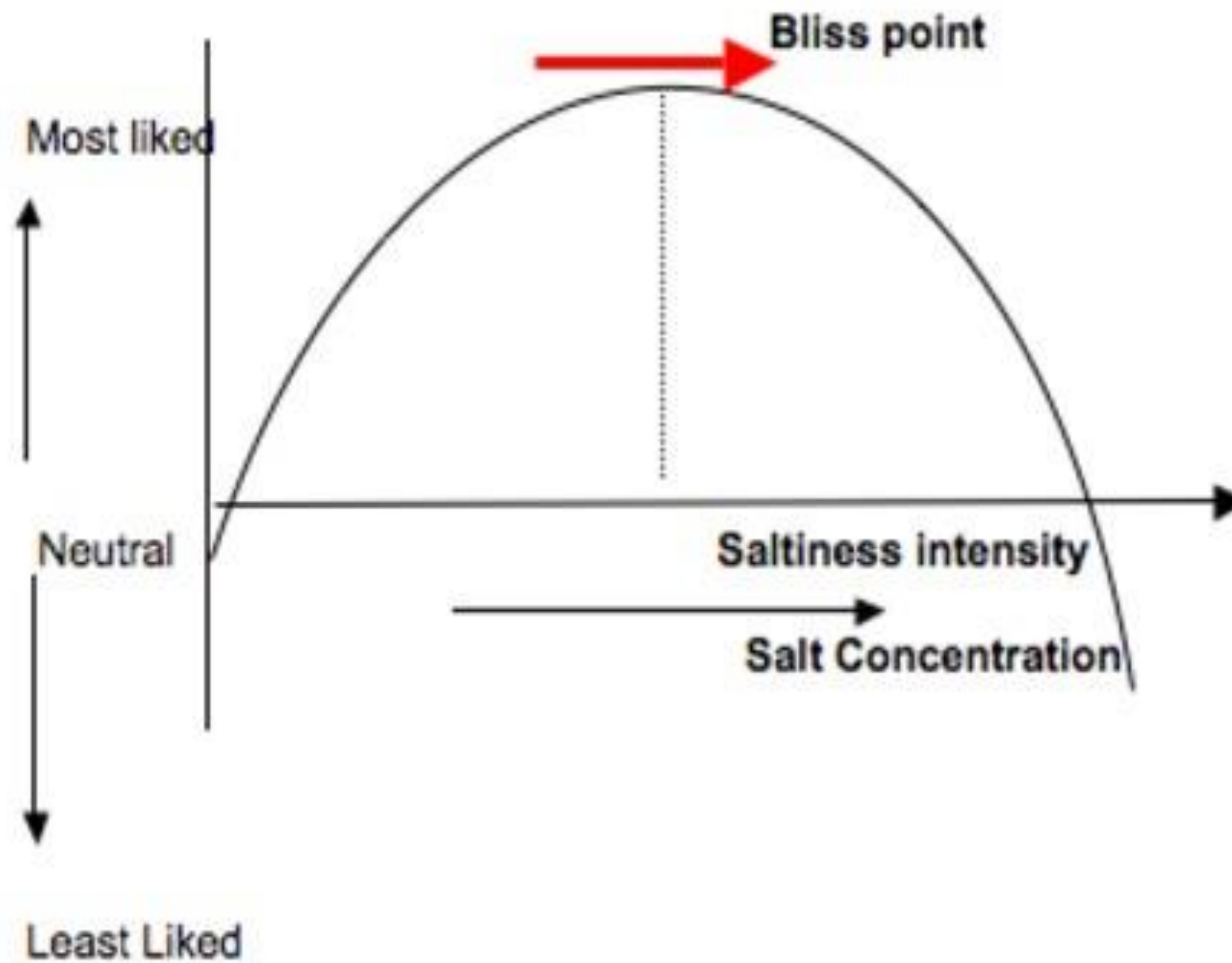
Lancet 2011; 378: 804–14

Critical Reviews in Food Science and Nutrition, 47:561–582 (2007)
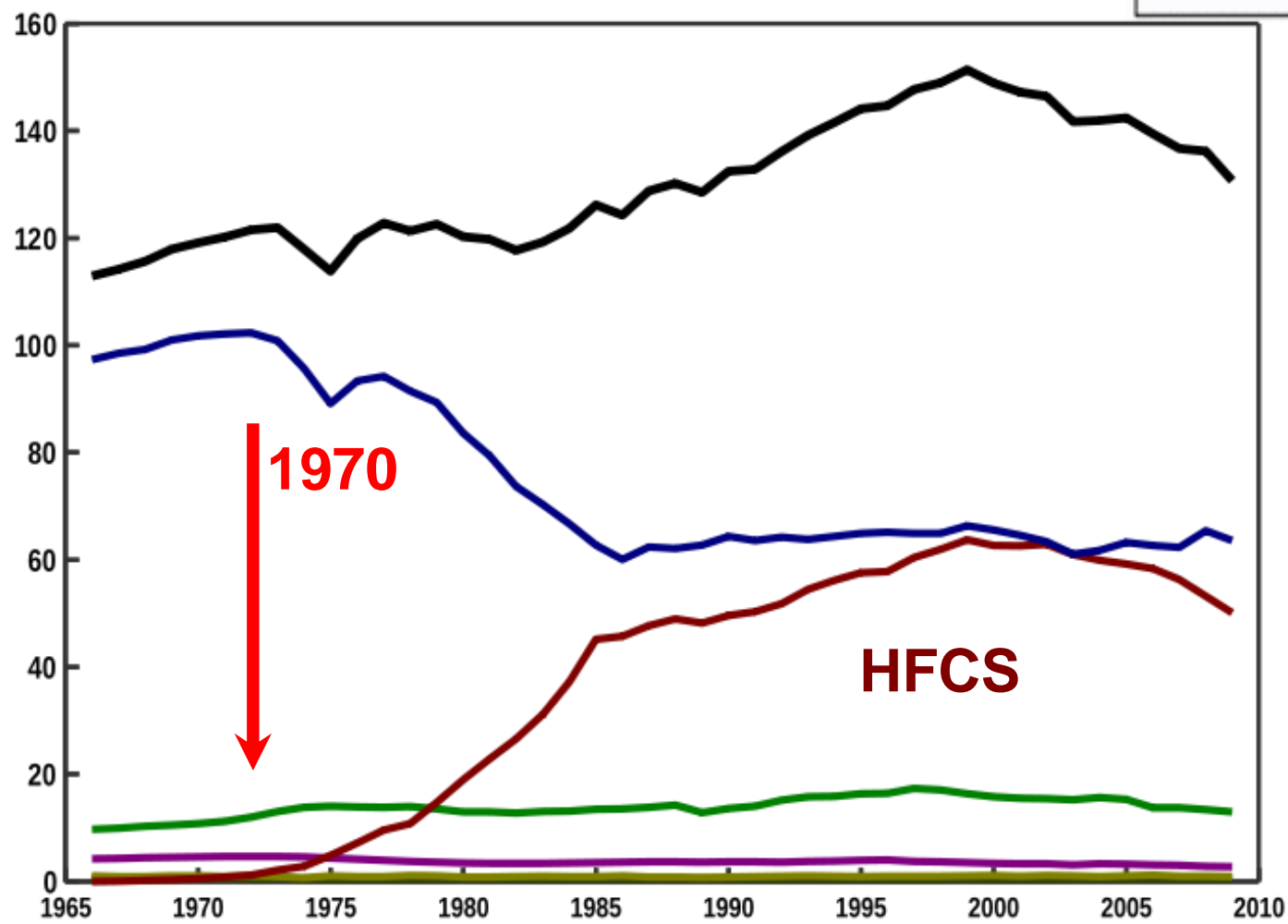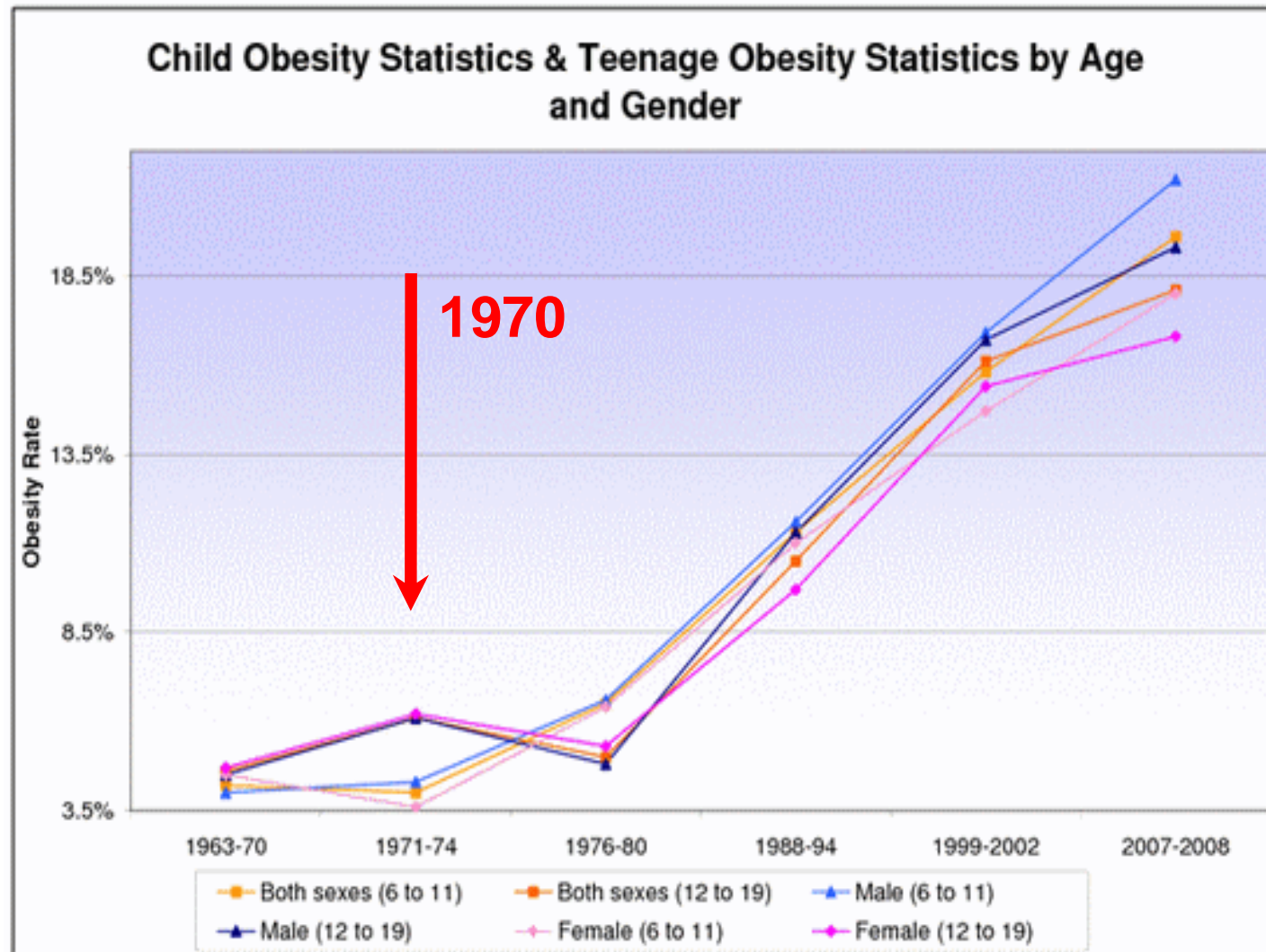
Figure 1. Bliss point graph. Relationship between salt (sodium chloride) concentration, saltiness intensity, and optimal level of liking demonstrated on the bliss point model.

- same brain areas are activated
- similar sensitization, compulsion and relapse pattern
- food environment helps addictive-type behaviour(ads, availability, larger serving sizes)
- animals: cross-sensitization between sucrose and amphetamine
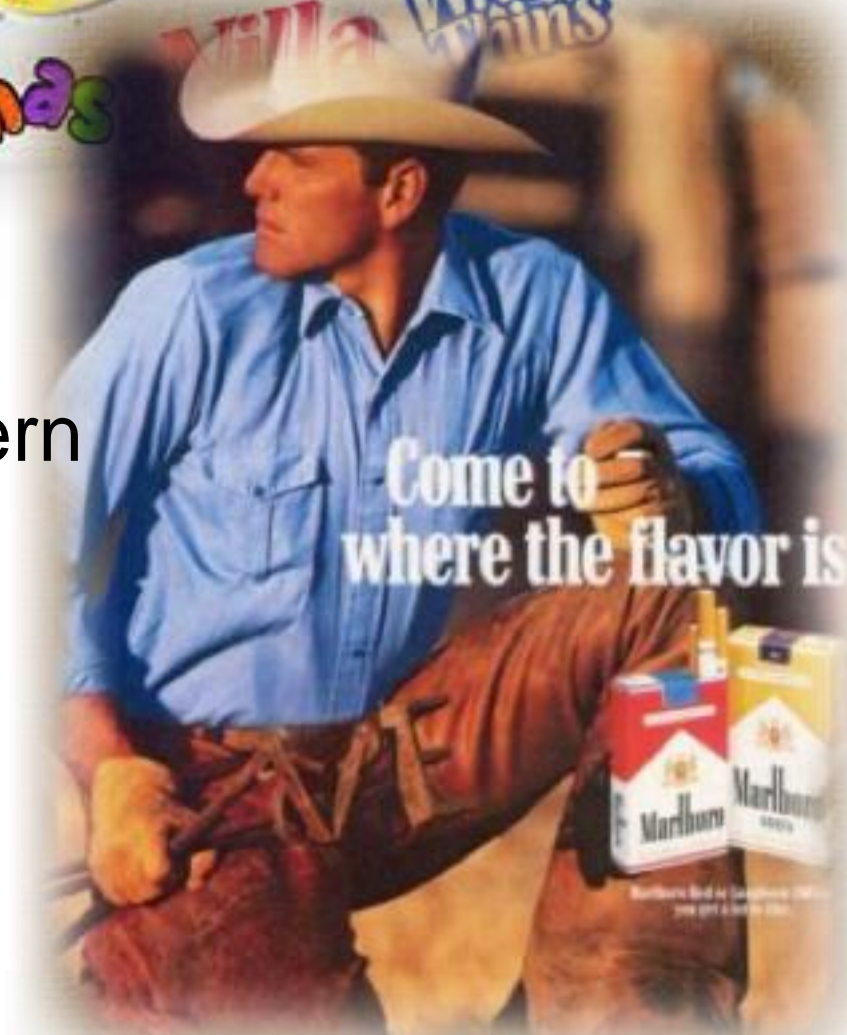
What's the cause of "idiopathic" obesity?

Energy disbalance: larger energy intake than expenditure

"**Obesitogenic environment**": obesity is a normal response of healthy people to an unhealthy environment

Therapy of obesity in childhood:

- Lifestyle modification:
  - Increase physical activity
  - Decrease calorie intake

- Medications
- Bariatric surgery

What to do?

Environmental/populati on change:

WORLD HEALTH ORGANIZATION

GLOBAL STRATEGY
ON DIET, PHYSICAL
ACTIVITY AND HEALTH

In May 2004, the 57th World Health Assembly (WHA) endorsed the World Health Organization (WHO) Global Strategy on Diet, Physical Activity and Health. The Strategy was developed through a wide-ranging series of consultations with all concerned stakeholders in response to a request from Member States at World Health Assembly 2002 (Resolution WHA55.23).

America's Move to Raise a Healthier Generation of Kids

Individual:
Correction of energy balance

BUT: children depend on their families and community!

Involve family!!!!!
Frequent visits!!!!!

A BALANCED

DIET IS

CHOCOLATE

IN BOTH HANDS

- minimum 1000 kcal/day!

- daily -500 kcal / weekly -0,5 kg

- cca. 60-20-20%

Traffic Light Foods: How To Talk To Kids About Real Food

Red Light Foods = Foods To Avoid

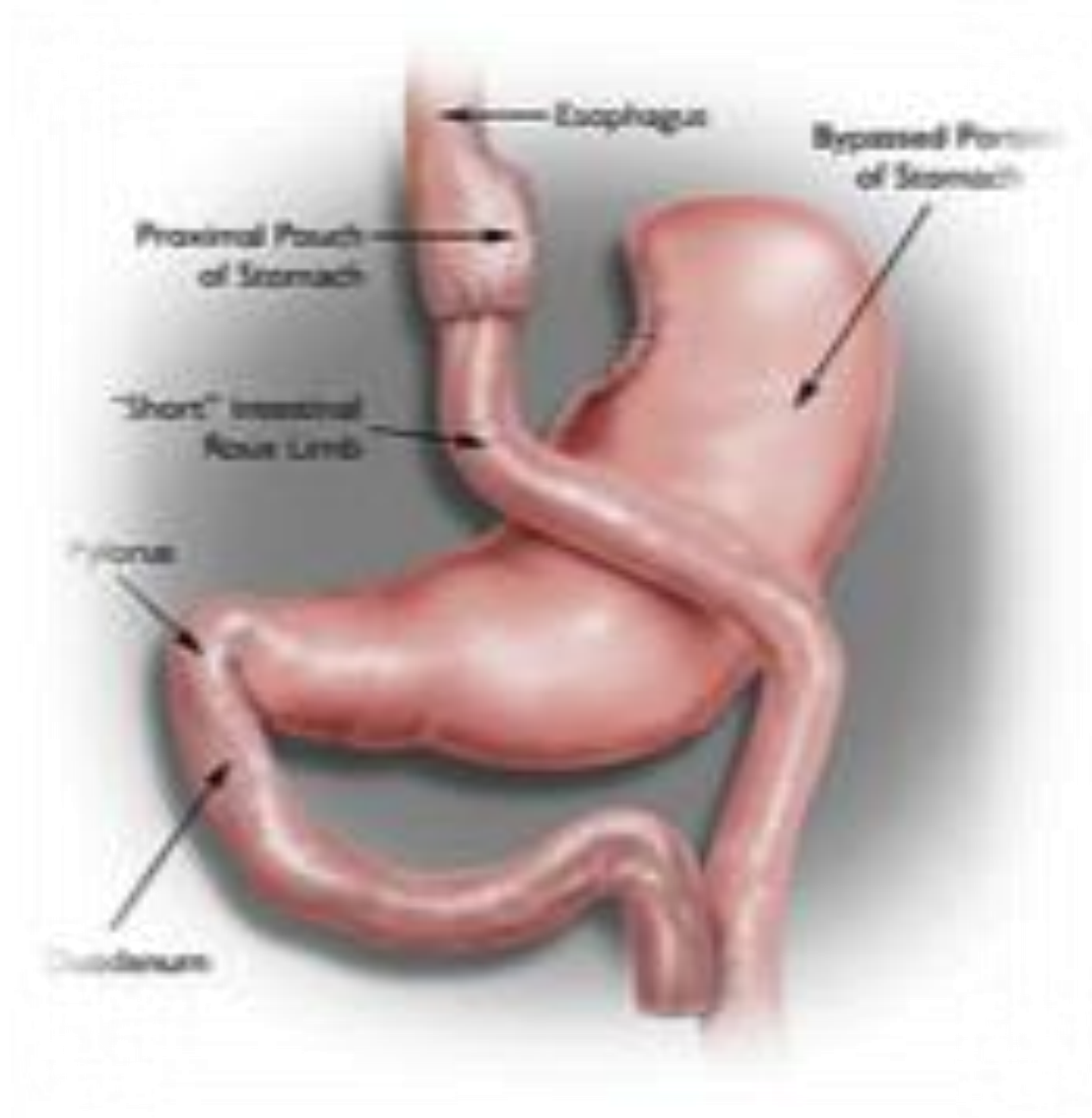Yellow Light Foods = Sometimes Foods

Green Light Foods = Go Foods

5. Therapy

Weight loss pills

Eg. Orlistat, caffeine

Bariatric surgery

Extreme obesity (BMI>35 kg/m2) + serious complication
Not routine!

Gold standard: Roux-en-Y bypass

Complications: screen and treat!

- Hypercholesterolemia
  - statins >10 years
- T2DM, IGT
  - metformin >10 years
- Hypertension
  - ACEi, ARB
- Androgen excess

Bottomline

- Genetic and endocrine obesity is rare, other symptoms!

- Idiopathic obesity is very frequent and increasing

- Work-up: rule out underlying disease and complications

- Lifestyle modification: Weight-loss programs: family and frequent visits

- Childhood obesity is strongly associated with childhood poverty