# Malnutrisi dan Sindroma Metabolik

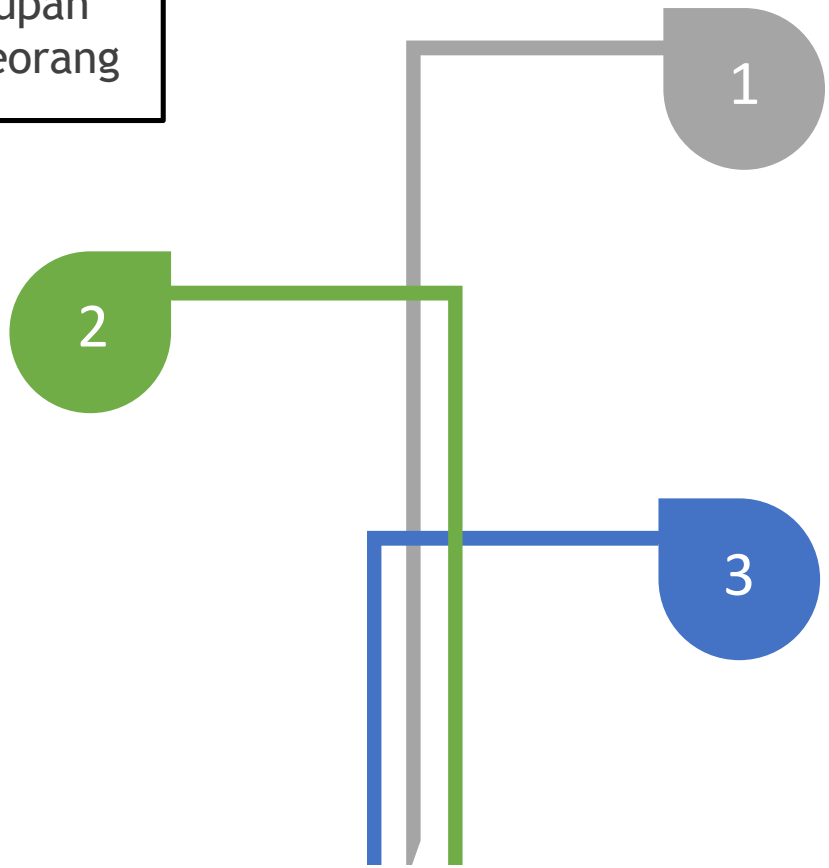
Arta Farmawati

# Fakta WHO 2020

- Malnutrisi ➔ kekurangan gizi (*wasting, stunting, underweight*), kekurangan vitamin atau mineral, kelebihan berat badan, obesitas, dan penyakit tidak menular yang berhubungan dengan diet.
- 1,9 miliar orang dewasa kelebihan berat badan atau obesitas, sementara 462 juta mengalami kekurangan berat badan.
- 47 juta anak di bawah usia 5 tahun menderita wasting, 14,3 juta sangat kurus dan 144 juta mengalami stunting, sedangkan 38,3 juta mengalami kelebihan berat badan atau obesitas.
- Sekitar 45% kematian pada anak di bawah usia 5 tahun terkait dengan kekurangan gizi, terutama di negara berpenghasilan rendah dan menengah. Pada saat yang sama, di negara-negara yang sama ini, tingkat kelebihan berat badan dan obesitas pada masa kanak-kanak meningkat.

# Tiga kelompok besar malnutrisi

Malnutrisi mengacu pada kekurangan, kelebihan, atau ketidakseimbangan dalam asupan energi dan / atau nutrisi seseorang




Kekurangan gizi, yang meliputi wasting (*low weight-for-height*), stunting (low weight-for-age) dan *underweight* (*low weight-for-age*)

Malnutrisi terkait mikronutrien, yang meliputi defisiensi mikronutrien (kekurangan vitamin dan mineral penting) atau kelebihan mikronutrien

Kelebihan berat badan, obesitas, dan penyakit tidak menular terkait diet (seperti penyakit jantung, stroke, diabetes, dan beberapa jenis kanker)

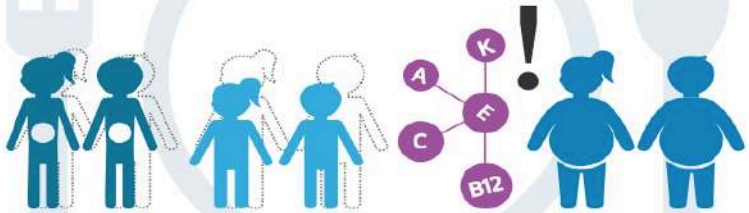
World Health Organization

# THE DOUBLE BURDEN OF MALNUTRITION

## WHAT

THE DOUBLE BURDEN OF MALNUTRITION IS CHARACTERISED BY THE COEXISTENCE OF:

1




Undernutrition (wasting, stunting & micronutrient deficiencies) along with overweight and obesity

2

and diet-related noncommunicable diseases

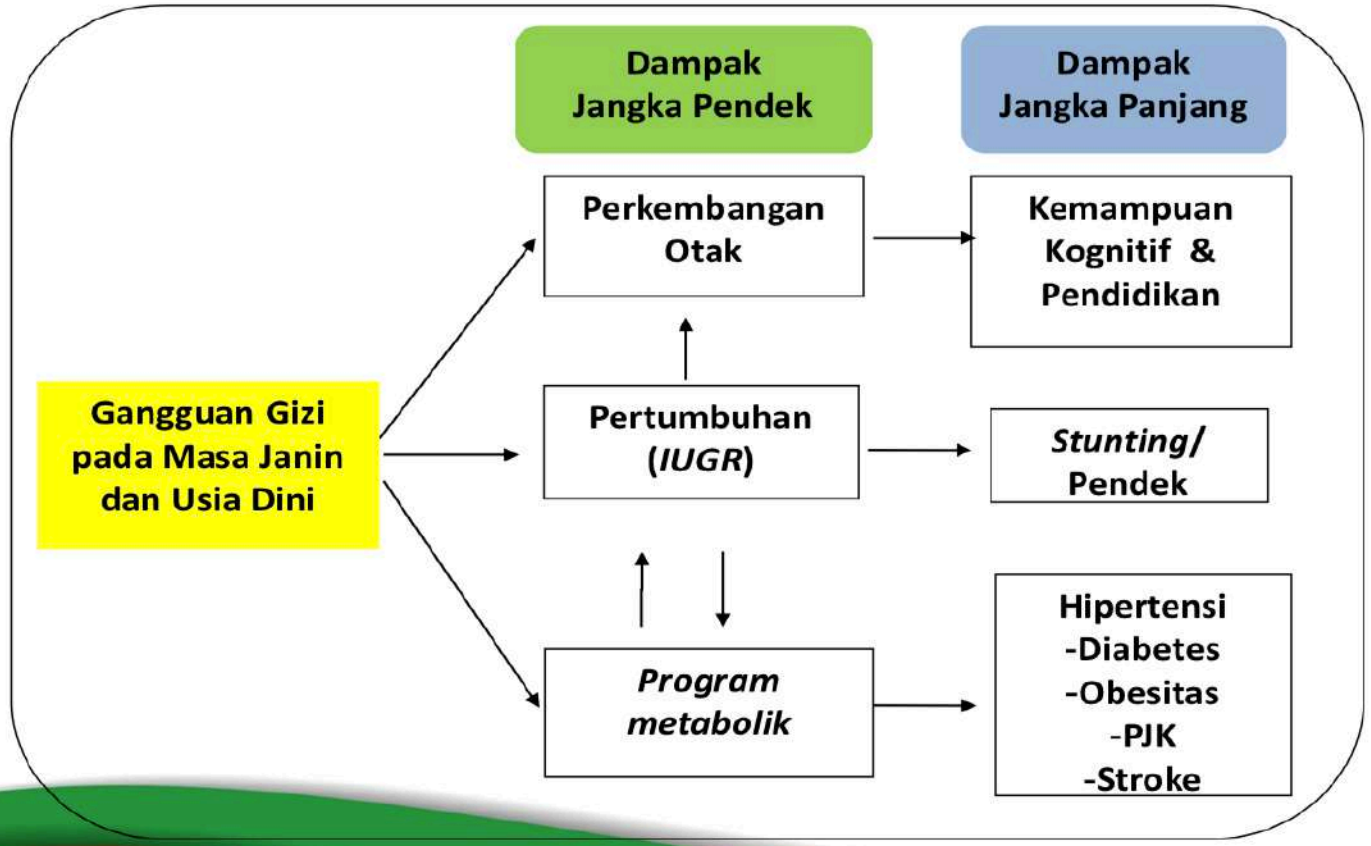
3

within individuals, households and populations

4

throughout life

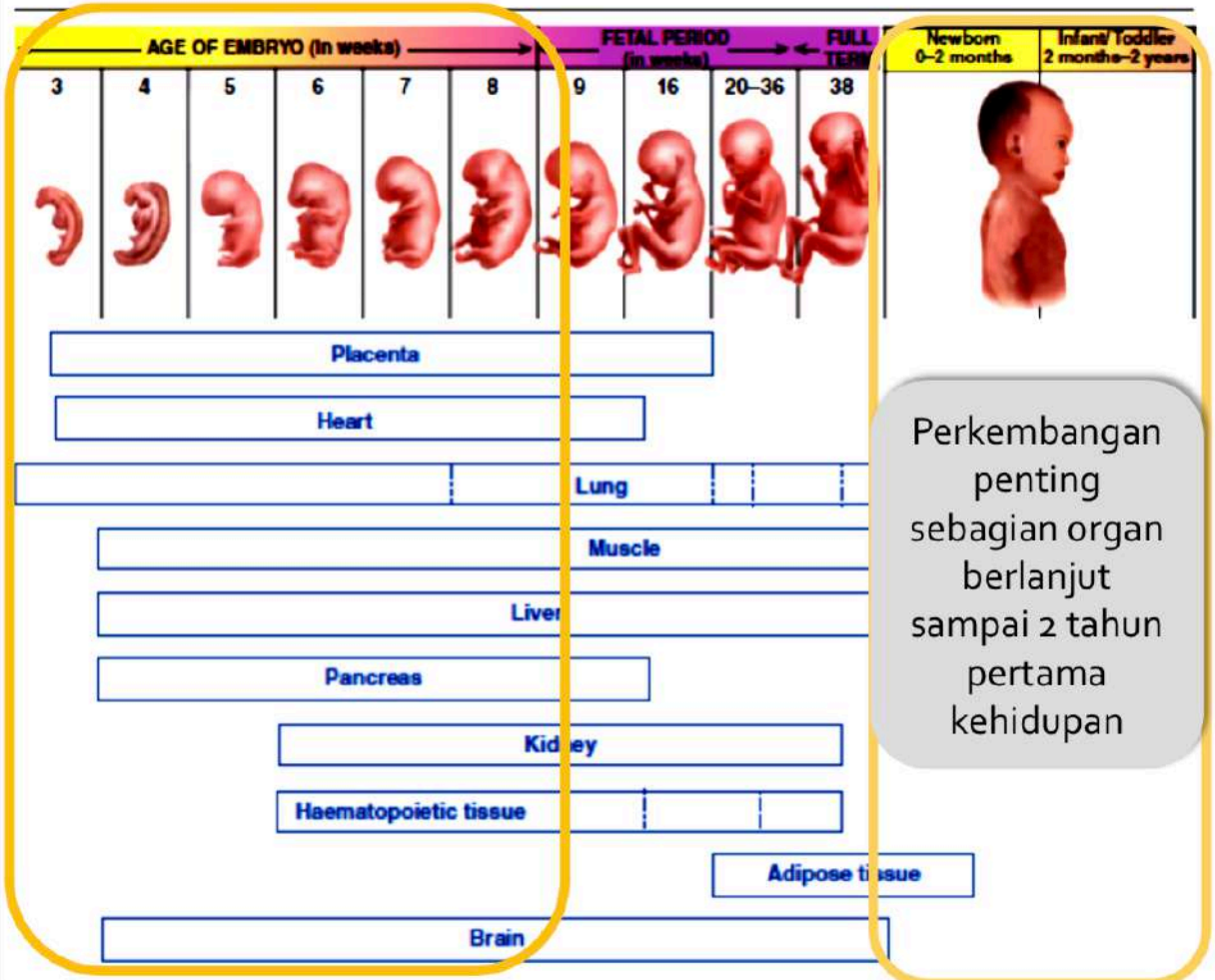
# Apakah yang terjadi bila terjadi gangguan gizi pada periode kehamilan dan usia dini?




*Sumber : Modifikasi dari Rajagopalan, S, Nutrition and challenges in the next decade, Food and Bulletin vol 24 no.3, 2003*

**Jendela Kritis Pertumbuhan dan Perkembangan Janin**

**8 minggu pertama sejak pembuahan terjadi pembentukan semua cikal bakal organ tubuh**




Perkembangan penting sebagian organ berlanjut sampai 2 tahun pertama kehidupan

**Figure 4.2** Critical windows in embryo and fetal development. As can be seen, all bar one of the organs discussed in this chapter begin their development during the critical first 8 weeks of gestation.
**Note:** Horizontal bars indicate time periods of development of different organs. The vertical dotted lines indicate distinct stages of organ development.
Medical illustrations: James Dowdalls. Graph production, Jane Teis Graphic Services. From the UCLA Institute of the Environment and Sustainability, Southern California Environmental Report Card, 'Air Pollution Impact on Infants and Children', Beate Ritz, MD, Ph.D. and Michelle Wilhelm, Ph.D.

# Akar Trans-generasi Penyakit Kronis

*Barker, Public Health 2012*

## *100 Tahun Alur Gizi*

**NENEK:**

Mewariskan gen dalam pembuatan sel telur/ovum cucunya

**IBU:**

- Melepaskan sel telur (mewariskan gen);
- Menyediakan zat gizi/makanan;
- Mempengaruhi perkembangan plasenta;
- Melahirkan bayi;
- Memberi makan bayi;
- Menstimulasi bayi;
- Memberi makan anak.

**BAPAK:**

- Mewariskan gen

**PLACENTA:**

- Mentransportasikan zat gizi;
- Memproduksi hormon;
- Mengeluarkan buangan

**JANIN:**

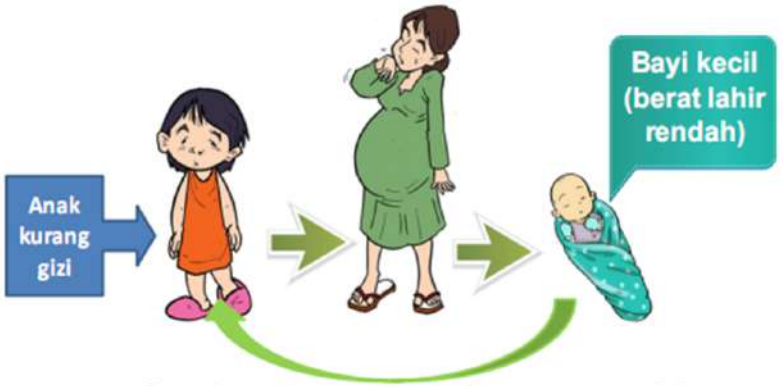
- Mengambil zat gizi melalui plasenta;
- Terbentuk organ;
- Bertumbuh dan berkembang.

**BAYI/ANAK**

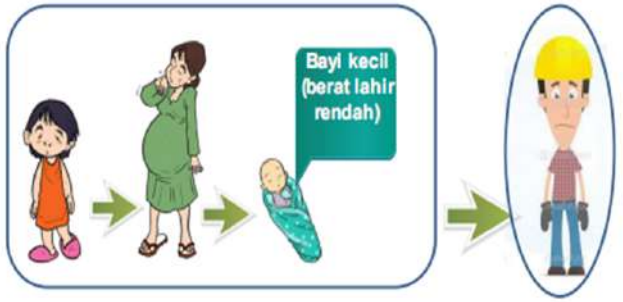
- Makan makanan;
- Bertumbuh dan berkembang.

**Kerentanan thd Penyakit Kronik, Kanker dan infeksi**

***Perkembangan 1000 hari***

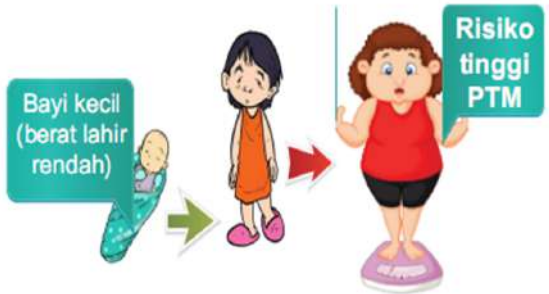



- Cenderung menjadi dewasa pendek
- Cenderung melahirkan bayi kecil




- Risiko mempunyai prestasi pendidikan yg rendah → tingkat pekerjaan rendah → gaji rendah → Status ekonomi rendah


- Risiko rendahnya kecerdasan → prestasi pendidikan yg rendah → status ekonomi rendah




Bayi dg BBLR memiliki risiko tinggi menderita PTM, seperti Penyakit Jantung, Hipertensi, Diabetes dan Obesitas

## *Mengukur obesitas*

### Klasifikasi IMT menurut WHO untuk etnis Asia

| Klasifikasi | IMT ($kg/m^2$) | Risiko penyakit | |
|---|---|---|---|
| | | Lingkar pinggang | |
| | | < 90 cm (pria)<br>< 80 cm (wanita) | ≥ 90 cm (pria)<br>≥ 80 cm (wanita) |
| Underweight | <18.5 | Rendah (tetapi risiko untuk penyakit lain meningkat) | Batas normal |
| Normal range | 18.5-22.9 | Batas normal | Meningkat |
| Overweight | ≥23 | | |
| *At risk* | 23.0-24.9 | Meningkat | Sedang |
| *Obese I* | 25-29.9 | Sedang | Berat |
| *Obese II* | ≥ 30.0 | Berat | Sangat berat |

**World Health Organization,** 1998

TIME TO ACT

International Diabetes Federation

# Nutrition indicators for monitoring and impact assessment

| Intervention | Most relevant nutritional indicators |
|---|---|
| Improved availability of food (dietary energy) at the household level, *in areas where dietary energy intake is initially constrained* | BMI (adults)<br>Weight-for-height Z-score (2-5 year olds)<br>Weight-for-age Z-score (2-5 year olds)<br>Height-for-age Z-score (long-term evaluations only; 2-5year olds) |
| Improved availability of food at the individual level, plus improvements in other basic needs, especially health | Height-for-age Z-score (under 5s)<br>Weight-for-age Z-score (under 5s)<br>Weight-for-height Z-score (under 5s) |
| Increased intake of animal products | Anemia (Hemoglobin)<br>Serum Vitamin A (retinol) |
| Increased intake of fruits and leaves | Serum Vitamin A (retinol) |



PPT Mohammad Abdul Mannan, Phd (FAO)

# Malnutrisi Protein Energi(PEM)

- Disebabkan konsumsi protein atau kalori atau keduanya yang tidak adekuat

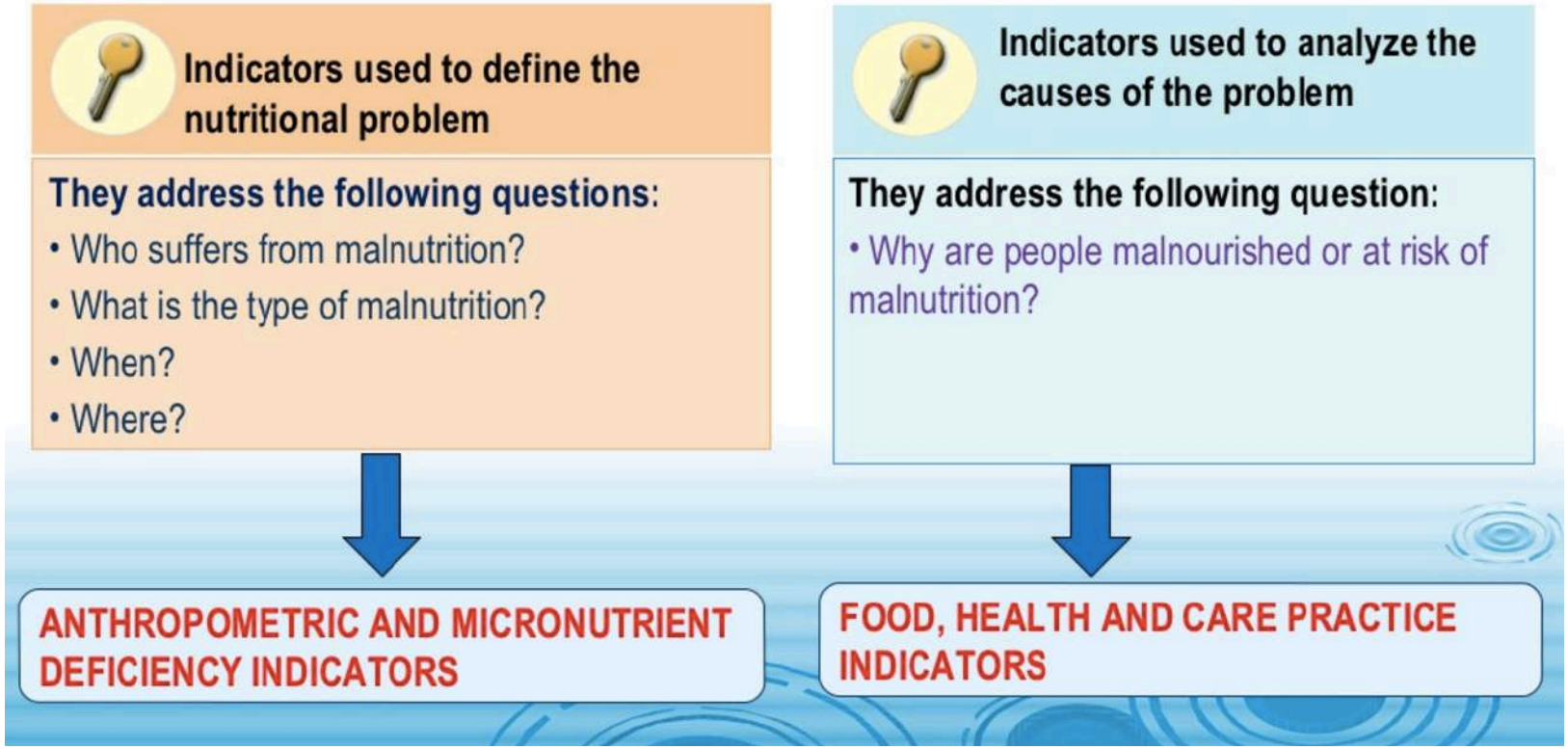
- Marasmus: inadekuat konsumsi protein dan kalori
- Gambaran klinis:
  - retardasi, severe wasting, kehilangan lemak tubuh, kepala besar tidak proporsional, retardasi mental, kulit kering dan dehidrasi, lesi mata (defisiensi vitamin A)
- Biokimiawi:
  - Albumin darah rendah, vitamin A darah rendah

- Kwasiorkor: inadekuat protein
- Gambaran klinis:
  - Edema, distensi abdomen, nafsu makan turun, dermatitis, rambut abnormal, apatis dan anemia, diarem rentan infeksi
- Biokimiawi:
  - Hipoproteinemia, perlemakan hepar

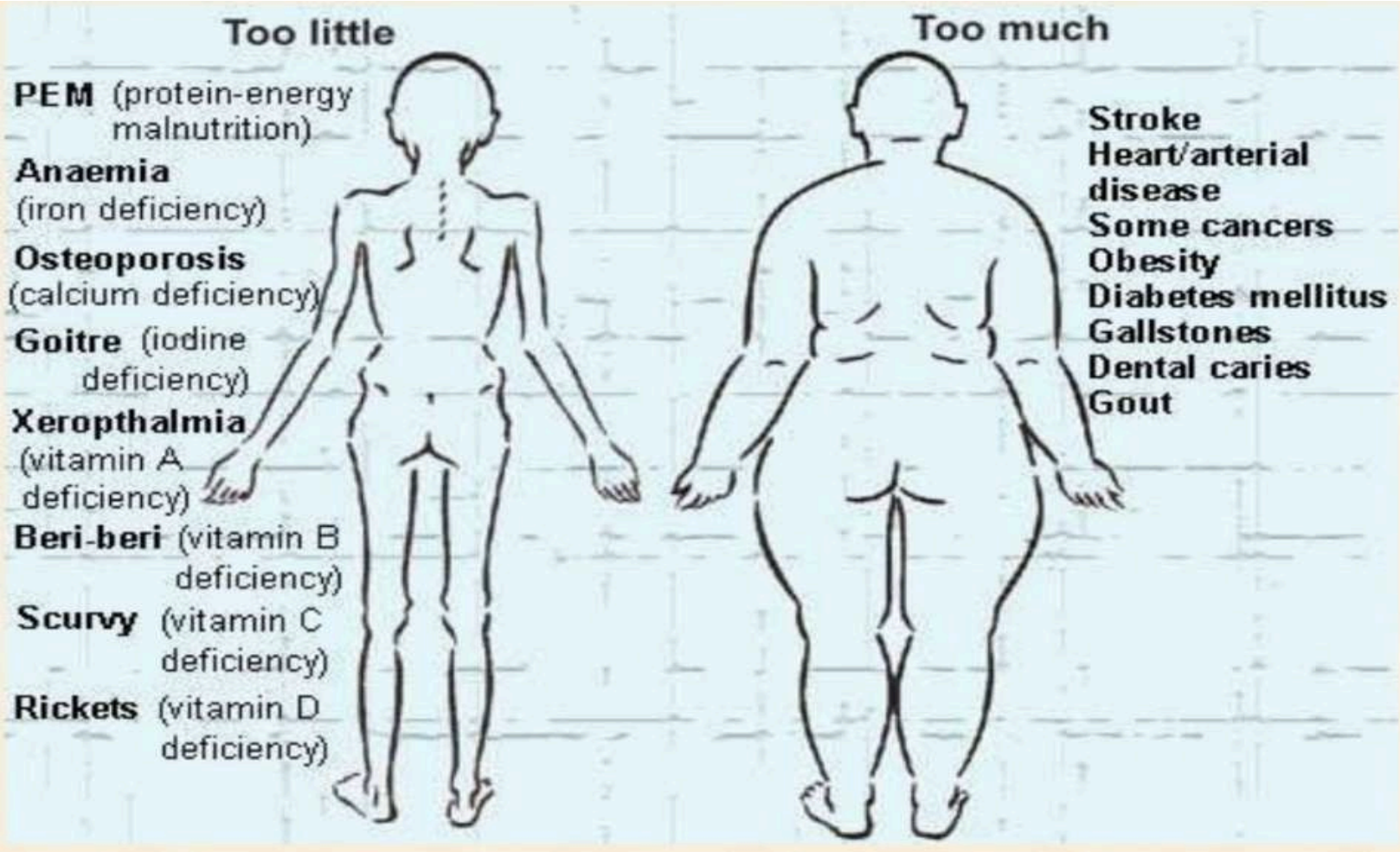
| FEATURES | MARASMUS | KWASHIORKOR |
|---|---|---|
| CLINICAL | *ALWAYS PRESENT* | |
| Muscle wasting | Obvious | Sometimes hidden by oedema and fat |
| Fat wasting | Severe loss of subcutaneous fat | Fat often retained but not firm |
| Oedema | None | Present in lower legs, and usually in face and lower arms |
| Weight for height | Very low | Low but may be masked by oedema |
| Mental changes | Sometimes quiet and apathetic | Irritable, moaning, apathetic |
| CLINICAL | *SOMETIMES PRESENT* | |
| Appetite | Usually good | Poor |
| Diarrhoea | Often (current and past) | Often (current and past) |
| Skin changes | Usually none | Diffuse pigmentation, sometimes 'flaky paint dermatosis' |
| Hair changes | Seldom | Sparse, silky, easily pulled out |
| Hepatic enlargement | None | Sometimes, due to accumulation of fat |
| *BIOCHEMICAL* | | |
| Serum albumin | Normal or slightly decreased | Low (<3 g/100 ml blood) |
| Urinary urea per g creatinine | Normal or decreased | Low |
| Hydroxyproline/creatinine ratio | Low | Low |
| Plasma/amino acid ratio | Normal | Elevated |

# Nutrition and health indicators

Different **indicators** are used for assessment and analysis purposes.



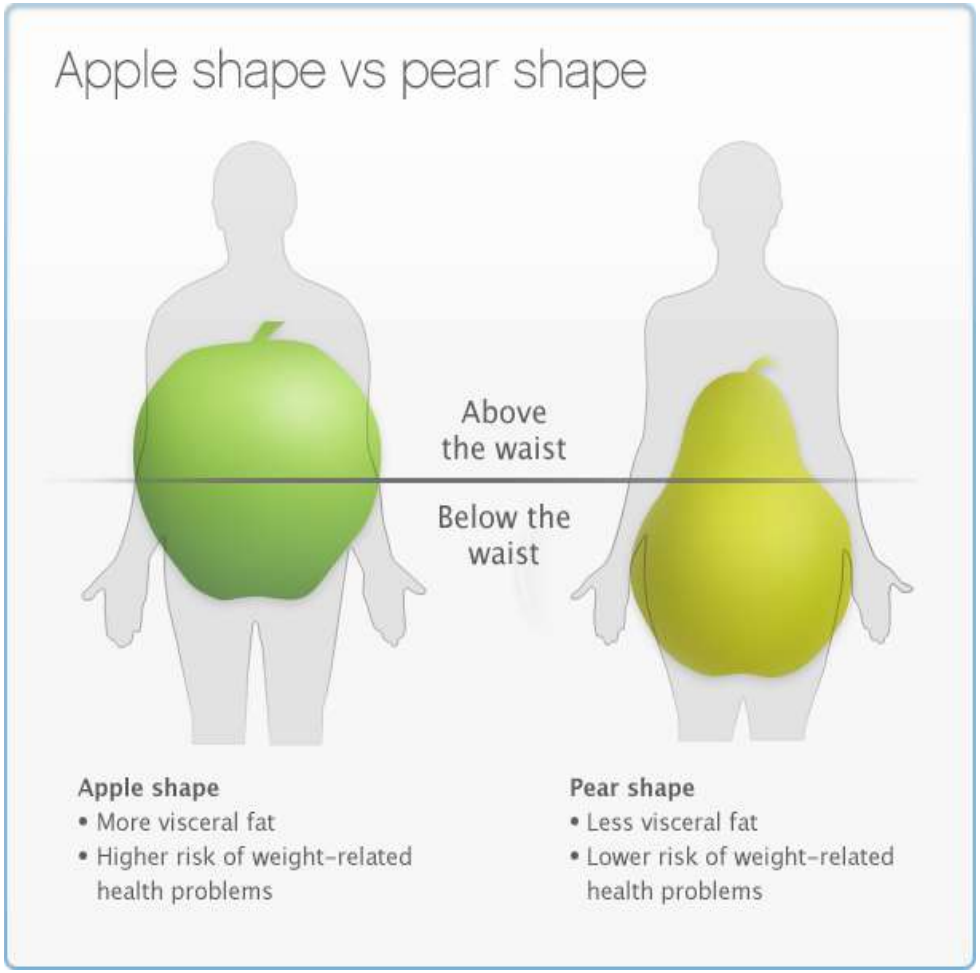
Examples of illnesses caused by improper nutrient consumption




Source: Dr. Soha Rashed

# Obesitas

- Peningkatan berat badan yang melebihi batas kebutuhan skeletal dan fisik sebagai akibat dari akumulasi lemak berlebihan dalam tubuh
- Berdasarkan distribusi jaringan lemak, dibedakan menjadi:
  - *Apple shape body* (distribusi jaringan lemak lebih banyak dibagian dada dan pinggang)
  - *Pear shape body/gynecoid* (distribusi jaringan lemak lebih banyak dibagian pinggul dan paha)



# Etiologi

- Gangguan emosi
- Konsumsi berlebih
- Gangguan fungsi endokrin
- Gangguan pusat pengatur kenyang dan selera makan di hipotalamus
- Herediter
- Faktor eksternal
- Aktifitas fisik kurang

**Secondary causes of obesity**

- Constitutional
- Hypothyroidism
- Cushing's syndrome
- Hypothalamic damage (extreme hyperphagia)
- Genetic, e.g. Prader-Willi
- Growth hormone deficiency
- Drugs, e.g. antidepressants

**Specific causes**

***Genetic***
E.g. Prader-Willi syndrome, Laurence-Moon (Biedl-Bardet) syndrome.

***Single gene defects***
E.g. mutations of leptin (provides feedback from adipocytes to hypothalamus about body fat stores) or its hypothalamic receptor (very rare).

***Hypothalamic lesions***
Lesions which damage the ventromedial nucleus (the 'satiety' area) may lead to obesity.

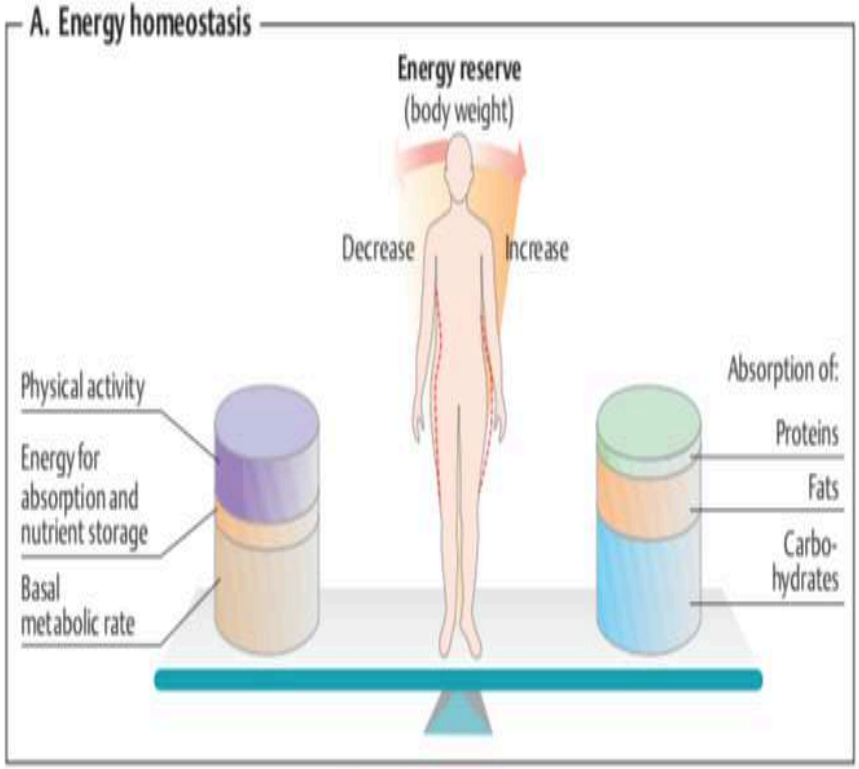
***Lesions include***
- Trauma.
- Tumours—craniopharyngiomas and astrocytomas.
- Inflammation—such as TB and meningitis.
- Infiltration—histiocytosis and sarcoidosis.

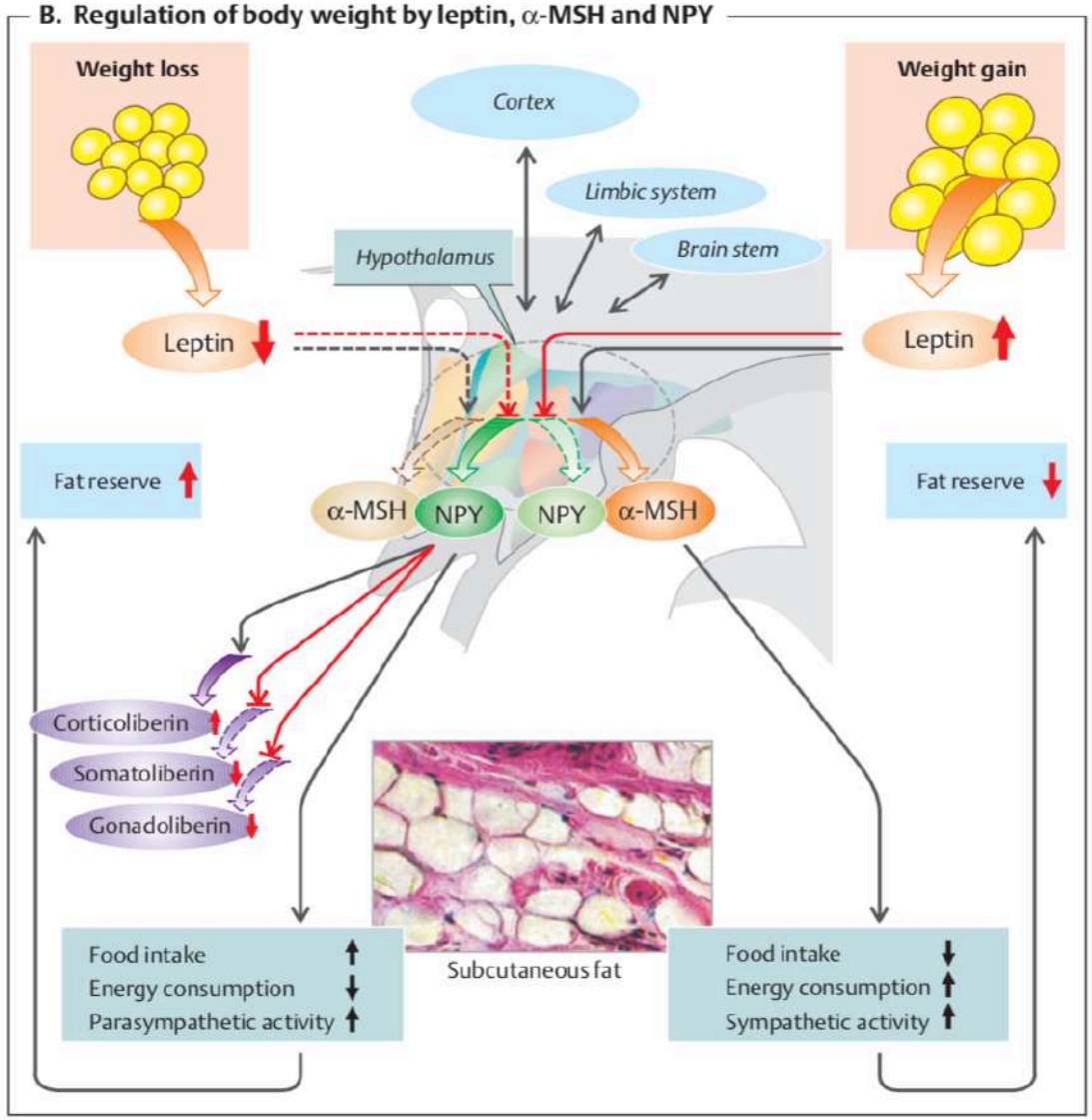
***Cushing's syndrome***
With 'buffalo' hump and central obesity.

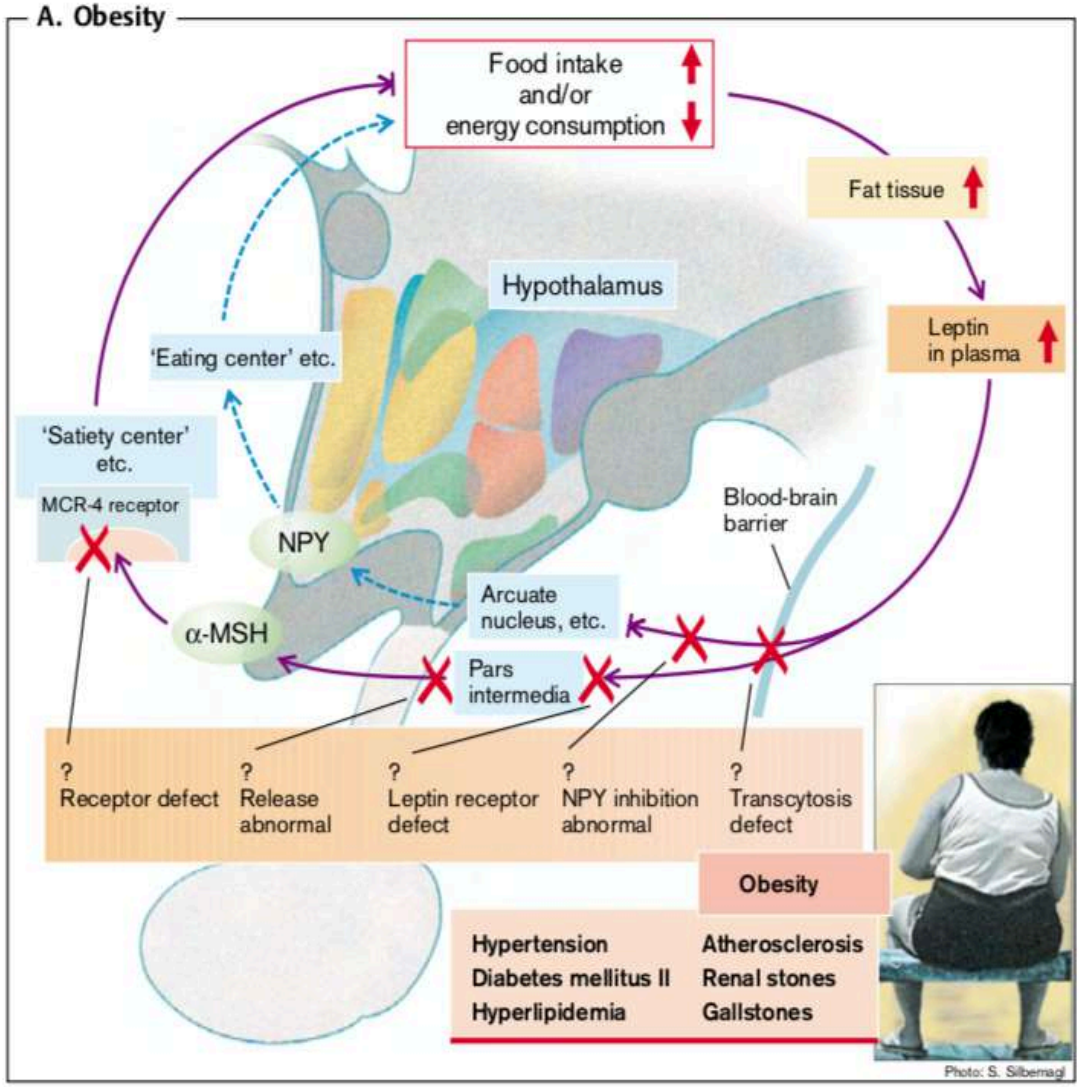
***Hypothyroidism***
Disputed unless severe myxoedema, but hyperthyroidism is associated with unphysiological weight loss.

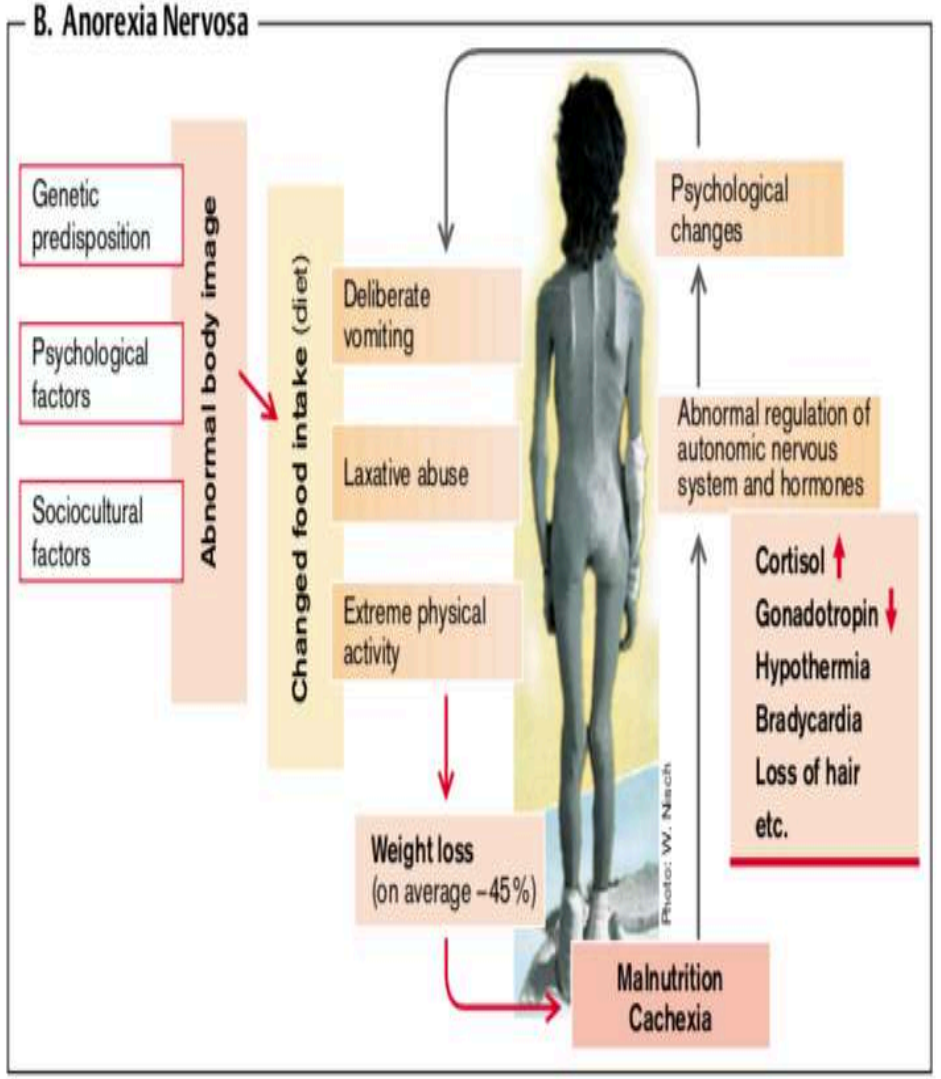
***Insulinoma***
Often associated with moderate weight gain; rare.

***Marked ↓ motor inactivity***
E.g. severe mental retardation or physical disability.


A. Energy homeostasis
Energy reserve
(body weight)
Decrease
Increase
Physical activity
Energy for absorption and nutrient storage
Basal metabolic rate
Absorption of:
Proteins
Fats
Carbo-hydrates


B. Regulation of body weight by leptin, α-MSH and NPY
Weight loss
Weight gain
Cortex
Limbic system
Hypothalamus
Brain stem
Leptin
Leptin
Fat reserve
Fat reserve
α-MSH
NPY
NPY
α-MSH
Corticoliberin
Somatoliberin
Gonadoliberin
Subcutaneous fat
Food intake
Energy consumption
Parasympathetic activity
Food intake
Energy consumption
Sympathetic activity



# Regulasi Berat Badan (Essential Biochemiostry for Medicine)

**Table 3.1** Candidate satiety factors

| Factor | Description | Comments |
|---|---|---|
| Leptin | A cytokine, produced predominantly by fat cells (adipocytes); plasma levels of leptin rise and fall in parallel to body fat content (> fat > leptin). | A major site of leptin receptors is in the hypothalamus (the arcuate nucleus). The janus kinase (JAK)–signal transducer and activator of transcription (STAT) pathway play a critical role in the signalling of a wide array of cytokines and growth factors, leading to various cellular functions, including proliferation, growth, haematopoiesis and immune response. It also has an effect on ATP-sensitive potassium channels in glucose-responsive neurones, which affect the neuronal firing rate. Leptin has major effects on reproductive behaviour (sexual maturation is delayed by lack of food). Starving women, female athletes and anorexics with low fat stores experience secondary amenorrhea. Leptin signalling defects lead to gross obesity, but these are very rare in humans. |
| Peptide tyrosine tyrosine (PYY) | A gut hormone present in endocrine cells in the lower intestine that can be released by the presence of luminal free fatty acids. | Shown to inhibit gut motility and gastrointestinal and pancreatic secretions. It inhibits 'appetite-stimulating' NPY/AgRP-producing neurons (see below), thus signalling food intake and damping hunger. Many of these gut peptides are incretin hormones, which also stimulate insulin release. |
| Resistin | A peptide hormone produced by adipocytes (and probably by other tissues). | Polymorphism of the resistin gene is associated with obesity. Resistin has an anti-insulin action, and is itself suppressed by insulin and the pro-inflammatory cytokines. Output is increased by thyroid hormone T4 but the physiological function is not yet understood. |
| Adiponectin | A mixture of anti-inflammatory peptide hormones secreted by adipocytes, which also regulate energy homeostasis and the metabolism of glucose and lipids. | By increasing glucose catabolism, adiponectin achieves a reduction of glucose levels in vivo. Adiponectin increases insulin sensitivity in target tissues, but also stimulates fatty acid oxidation and blocks the differentiation of new adipocytes in bone marrow. |

**Table 3.1** (*continued*)

| Factor | Description | Comments |
|---|---|---|
| Pro-inflammatory cytokines | TNF-$\alpha$, IL-6 and IL-1 act on the hypothalamus to reduce appetite and raise body temperature in response to infection and other illnesses. First identified as products of the immune system (macrophages), it is now known that many other tissues (including adipocytes) can secrete these compounds. | There is a major negative feedback loop involving the hypothalamus, corticotrophin-releasing hormone, corticotropin (=ACTH) secreted by the pituitary and corticosteroids from the adrenal cortex, which dampen pro-inflammatory cytokine production. This loop normally acts to stabilise immune system activity, but it also has spillover effects on appetite and weight regulation. |
| Amylin | Pancreatic $\beta$-cells co-release a second polypeptide hormone called amylin at the same time as they release insulin. | Amylin produces a feeling of satiation, and may assist in the regulation of food intake. A modified amylin, Pramlintide, is being investigated as a hypoglycaemic agent in early type 2 diabetes. It potently reduces glucagon secretion and therefore postprandial hyperglycaemia. |
| Apolipoprotein A-IV | A glycoprotein synthesised by enterocytes in response to long-chain dietary fat. | Apolipoprotein A-IV may regulate PYY (see above). It is thought to regulate food intake, possibly by stimulating CCK production. It may be effective in its own right because it is also present in the brain. |
| Endocannabinoids, anandamide (orexigenic) and oleoylethanolamide (anorexigenic) | May be important for gastrointestinal function and the regulation of food intake. | Rimonabant is an inverse agonist for CB1 cannabinoid receptors, which is approved in Europe for weight-loss therapy, but has significant side effects on the central nervous system. |

TIME FOR A

# COFFEE BREAK

# Definisi Metabolik Sindrom

Sindrom metabolik adalah kelompok gejala obesitas abdominal, dislipidemia, hiperglikemia, dan hipertensi.

**International Diabetes Federation, 2005:**

Central obesity (defined as waist circumference ≥ 94 cm for Europoid men and ≥ 80 cm for Europoid women)

Plus any two of the following:

- Raised triglycerides > 1.7 mmol/L, or specific treatement for this lipid abnormality
- Reduced HDL cholesterol: < 1.03 mmol/L in males, and 1.29 mmol/L in females, or specific treatement for this lipid abnormality
- Raised blood pressure: systolic blood pressure ≥ 130mmHg or diastolic blood pressure ≥ 85 mmHg
- Raised fasting plasma glucose ≥ 5.6 mmol/L, or previously diagnosed diabetes mellitus

**World Health Organisation, 1998:**

- Diabetes or impaired fasting glycaemia or impaired glucose tolerance or insulin resistance (hyperinsulinaemic, euglycaemic clamp-glucose uptake in lowest 25%)
- Plus any two of the following:
- Obesity: BMI > 30 or waist-to-hip ratio > 0.9 (male) or > 0.85 (female)
- Dyslipidaemia: triglycerides ≥ 1.7 mmol/L or HDL cholesterol < 0.9 (male) or < 1.0 (female) mmol/L
- Hypertension: blood pressure > 140/90 mm Hg
- Microalbuminuria: albumin excretion > 20 µg/min

**National Cholesterol Education Program's Adult Treatment Panel III (NCEP: ATP III), 2001:**

Any 3 of the following:

- Central obesity: waist circumference > 102 cm (male), > 88 cm (female)
- Hypertriglyceridaemia: triglycerides ≥ 1.7 mmol/L
- Low HDL cholesterol: < 1.0 mmol/L (male), < 1.3 mmol/L (female)
- Hypertension: blood pressure ≥ 135/85 mm Hg or medication
- Fasting plasma glucose ≥ 6.1 mmol/L

McCracken *et al.*, 2018

## *Sindrom metabolik:*
## Definisi NCEP ATP III*

Diagnosis sindrom metabolik harus memenuhi **tiga atau lebih** dari lima faktor risiko berikut:

| Faktor risiko | Kadar definisi |
|---|---|
| Obesitas abdominal<br>Pria<br>Wanita | Lingkar pinggang<br>>102 cm (>40 in)<br>>88 cm (>35 in) |
| Trigliserida | ≥150 mg/dL (1.7 mmol/L) |
| HDL cholesterol<br>Pria<br>Wanita | <br><40 mg/dL (1.04 mmol/L)<br><50 mg/dL (1.29 mmol/L) |
| Tekanan darah | ≥130/ ≥85 mmHg |
| Glukosa puasa | ≥100 mg/dL (5.6 mmol/L) |

*2001, updated 2005

TIME TO ACT

International Diabetes Federation

## *Sindrom metabolik:*
## IDF consensus definition (2005)

| Obesitas sentral | | |
|---|---|---|
| Lingkar pinggang | - Tergantung etnik*<br>- untuk Eropa: | <br>Pria ≥ 94 cm<br>Wanita ≥ 80 cm |
| **Ditambah dua dari berikut:** | | |
| Kenaikan Trigliserida | ≥150mg/dL (1.7mmol/L)<br>*Atau sedang dalam pengobatan* | |
| Kolesterol HDL rendah | <40mg/dL (1.03 mmol/L) pada pria<br><50mg/dL (1.29 mmol/L) pada wanita<br>*Atau sedang dalam pengobatan* | |
| Kenaikan tekanan darah | Systolic : ≥130 mmHg *atau*<br>Diastolic: ≥85 mmHg *atau*<br>*Atau sedang dalam pengobatan* | |
| Kadar gula darah puasa terganggu | Gula darah puasa ≥100 mg/dL (5.6 mmol/L)<br>Atau sudah didiagnosis diabetes tipe II | |

TIME TO ACT

International Diabetes Federation

**Table 1** Historical and recent definitions of MetS

| Clinical and biochemical features | WHO 1998[2] | EGIR 1999[3] | NCEP ATP III 2001[4] | AACE 2003[5] | IDF 2005[6] | AHA/NHLBI 2005[7] | Consensus (AHA/NHLBI + IDF) 2009[8] |
|---|---|---|---|---|---|---|---|
| Insulin resistance | Impaired glucose tolerance, impaired fasting glucose, T2DM, or lowered insulin sensitivity **plus any two of the following** | Plasma insulin concentration > 75th percentile of nondiabetic patients, **plus any two of the following** | **Any three of the following** | Impaired glucose tolerance or impaired fasting glucose, **plus any of the following** | | **Any three of the following** | **Any three of the following** |
| Obesity | Abdominal obesity (waist-to-hip ratio >0.9 in men or >0.85 in women, or BMI > 30 kg/m$^2$) | WC ≥94 cm in men; ≥80 cm in women | WC >102 cm in men, >88 cm in women | BMI ≥25 kg/m$^2$ | BMI >30 kg/m$^2$ or WC with ethnicity-specific values,[a] **plus any two of the following** | WC ≥102 cm in men, ≥ 88 cm in women | Raised WC (population- and country-specific definitions) |
| Plasma glucose concentration[b] | Impaired glucose tolerance, impaired fasting glucose, or T2DM | FPG ≥110 mg/dL | FPG ≥110 mg/dL | Impaired fasting glucose, or Impaired glucose tolerance | FPG ≥100 mg/dL | FPG ≥100 mg/dL | FPG ≥100 mg/dL or on diabetes treatment |
| Hypertension | BP ≥140/90 mm Hg | BP ≥140/90 mm Hg, or on antihypertensive medication | BP ≥130/85 mm Hg | BP ≥130/85 mm Hg | BP ≥130/85 mm Hg, or on antihypertensive medication | BP ≥130/85 mm Hg, or on antihypertensive medication | BP ≥130/85 mm Hg, or on antihypertensive treatment |
| Triglycerides (TG)[c] | TG ≥150 mg/dL | TG ≥150 mg/dL or on treatment | TG ≥150 mg/dL | TG ≥150 mg/dL | TG ≥150 mg/dL or on treatment | TG ≥150 mg/dL or on treatment | TG ≥150 mg/dL or on treatment |
| HDL-cholesterol (HDLC)[d] | HDLC <40 mg/dL in men and <50 mg/dL in women | HDLC <39 mg/dL in men or women, or on treatment | HDLC <40 mg/dL in men and <50 mg/dL in women | HDLC <40 mg/dL in men and <50 mg/dL in women | HDLC <40 mg/dL in men and <50 mg/dL in women, or on treatment | HDLC <40 mg/dL in men and <50 mg/dL in women, or on treatment | HDLC <40 mg/dL in men and <50 mg/dL in women, or on treatment |
| Other | Urinary albumin excretion ≥20 μg/min, or ACR ≥30 mg/g | | | | | | |

*AACE*, American Association of Clinical Endocrinologists; *ACR*, albumin-creatinine ratio; *AHA*, American Heart Association; *BMI*, body mass index; *BP*, blood pressure; *EGIR*, European Group for Study of Insulin Resistance; *FPG*, fasting plasma glucose concentration; *HDLC*, high-density lipoprotein cholesterol; *IDF*, International Diabetes Federation; *NCEP ATP III*, National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III); *NHLBI*, National Heart, Lung, and Blood Institute; *T2DM*, type 2 diabetes mellitus; *TG*, triglycerides; *WC*, waist circumference; *WHO*, World Health Organization.

[a] Waist circumference: for Europids, >94 cm in men and >80 cm in women; for South Asians, Chinese, and Japanese, >90 cm in men and >80 cm in women; for ethnic South and Central Americans, use South Asian data; for sub-Saharan Africans and Eastern Mediterranean and Middle East (Arab) populations, use European data.

[b] To convert glucose concentration from milligrams per deciliter to millimoles per liter, multiply by 0.0555.

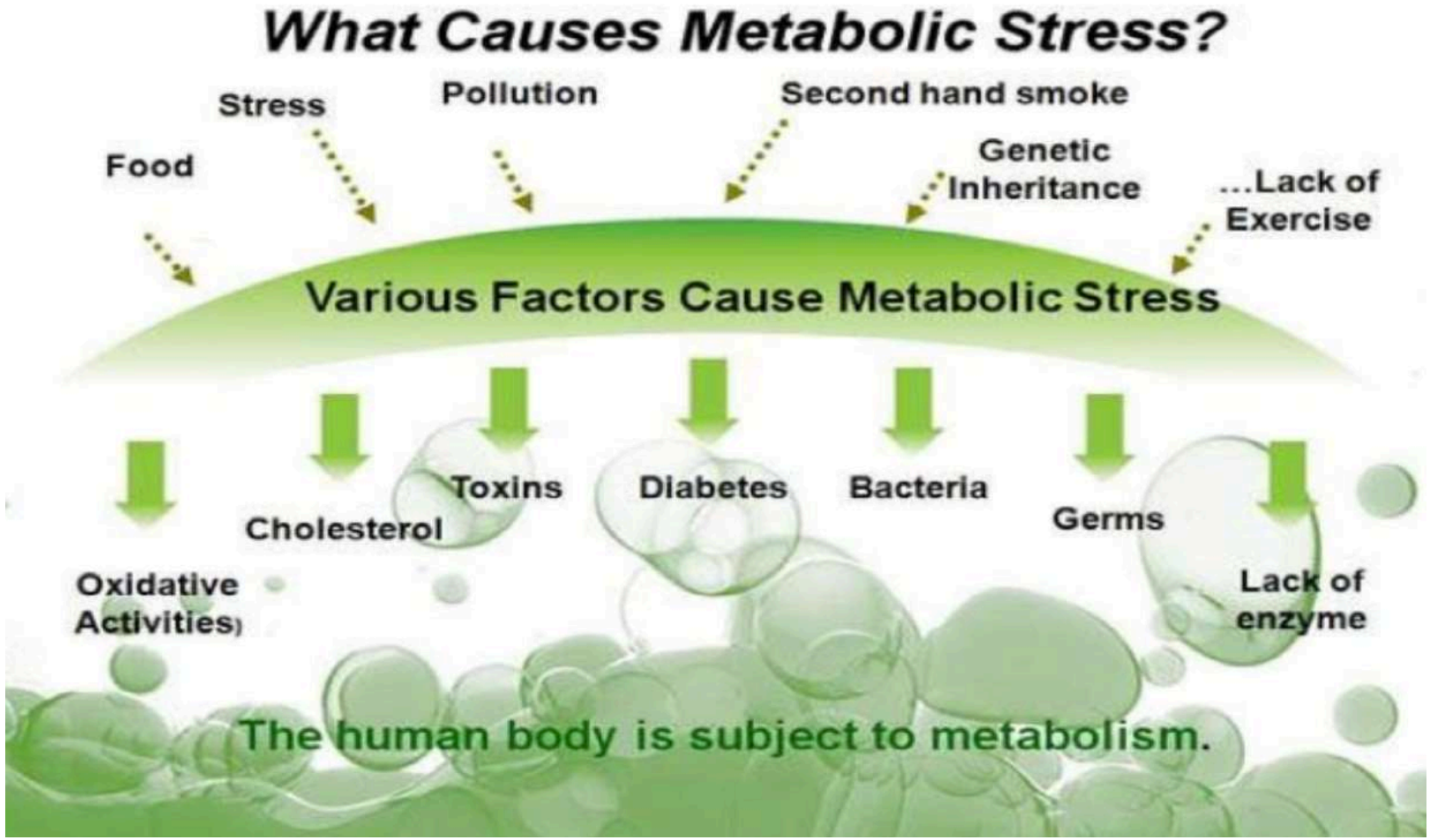
[c] To convert triglyceride concentration from milligrams per deciliter to millimoles per liter, multiply by 0.0113.

[d] To convert HDL-cholesterol concentration from milligrams per deciliter to millimoles per liter, multiply by 0.02586.

McCracken *et al*., 2018

# Faktor Risiko Sindrom Metabolik

- Riwayat keluarga
- Merokok
- Usia lanjut
- Obesitas
- Sosioekonomi rendah
- Etnik Mexican American
- Status postmenopause
- Aktivitas fisik kurang
- Konsumsi minuman manis dan bersoda
- Konsumsi alkohol berlebihan
- Pola diet barat
- Kebugaran kardiorespirasi rendah
- Menonton televisi berlebihan
- Penggunaan obat antiretroviral pada infeksi HIV
- Penggunaan obat antipsikotik atipikal (misalnya clozapine)
- Riwayat lahir kecil masa kehamilan
- Riwayat DM pada ibu selama kehamilan


What Causes Metabolic Stress?
Stress
Pollution
Second hand smoke
Food
Genetic Inheritance
...Lack of Exercise
Various Factors Cause Metabolic Stress
Toxins
Diabetes
Bacteria
Cholesterol
Germs
Oxidative Activities)
Lack of enzyme
The human body is subject to metabolism.

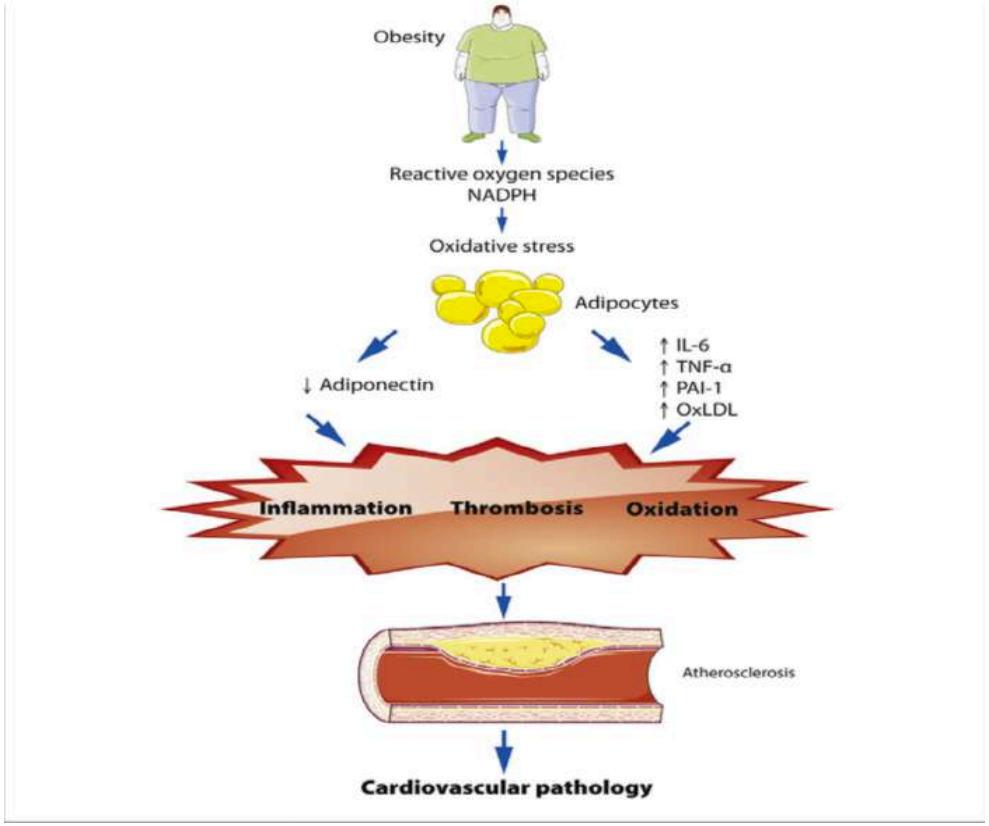
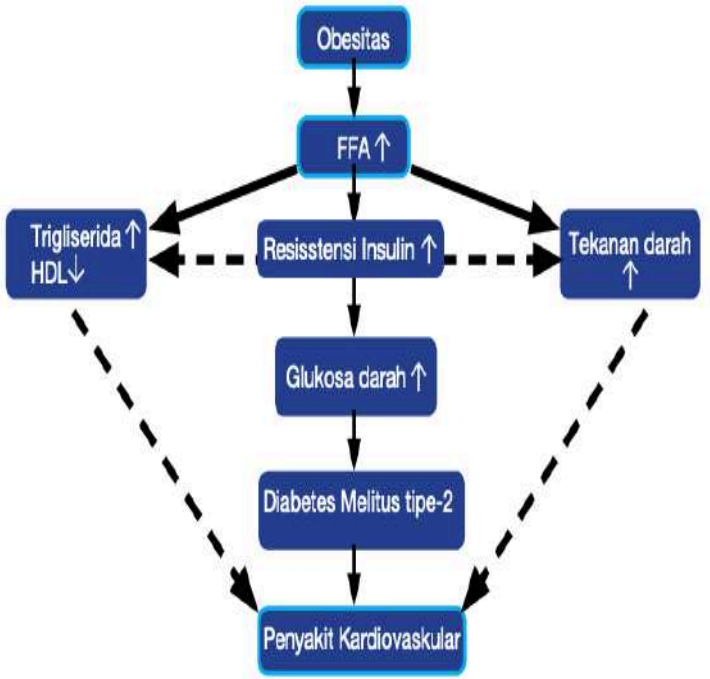


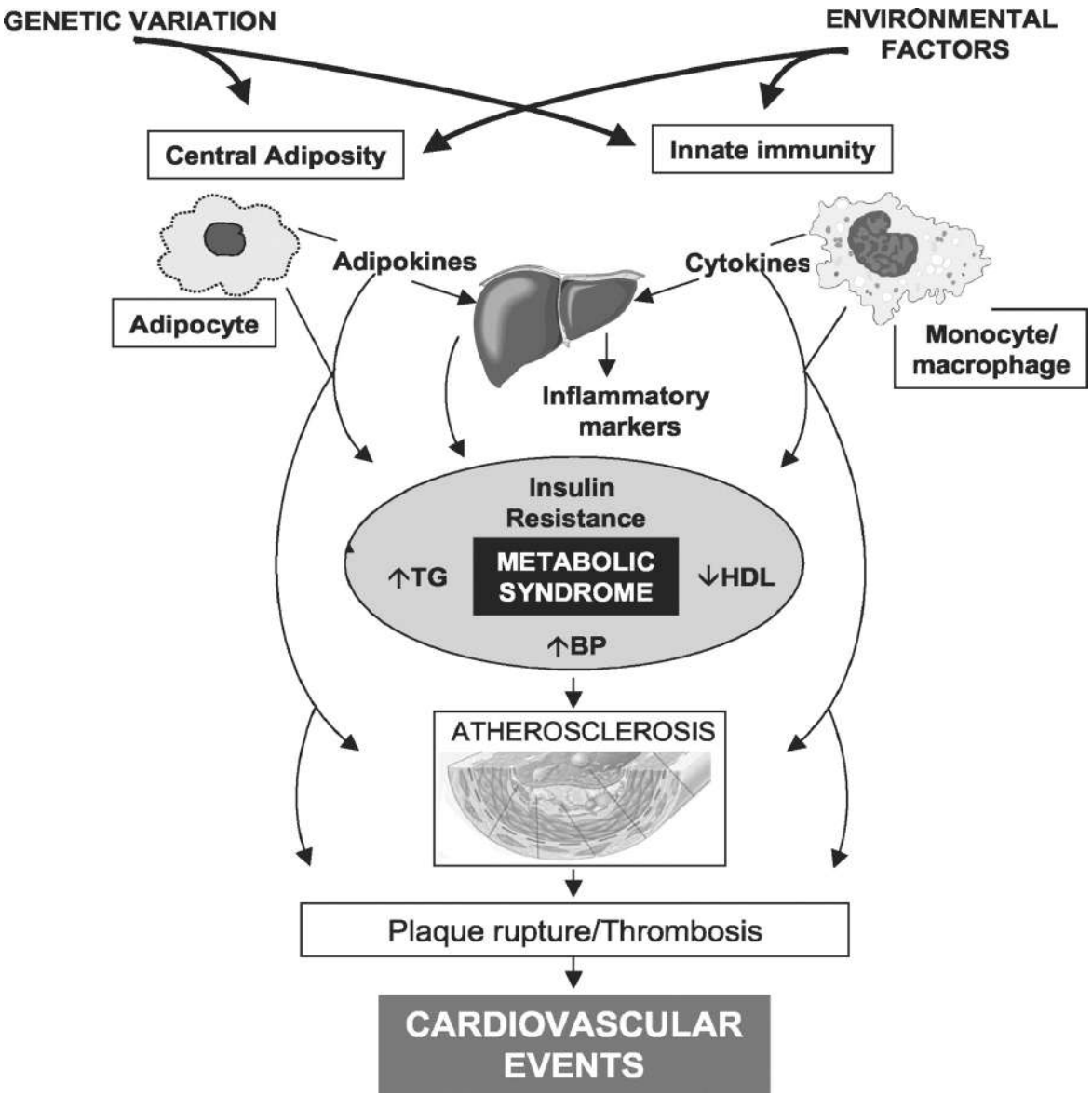

**Fig. 2** **NADPH** Nicotinamide adenine dinucleotide phosphate; **IL-6** Interleukin-6; **TNF-α** Tumour necrosis factor alpha; **PAI-1** Plasminogen activator inhibitor-1; **OxLDL** Oxidized low density lipoprotein. Schematic diagram of the role of inflammatory and oxidative mediators in the pathogenesis of atherosclerosis in the metabolic syndrome.

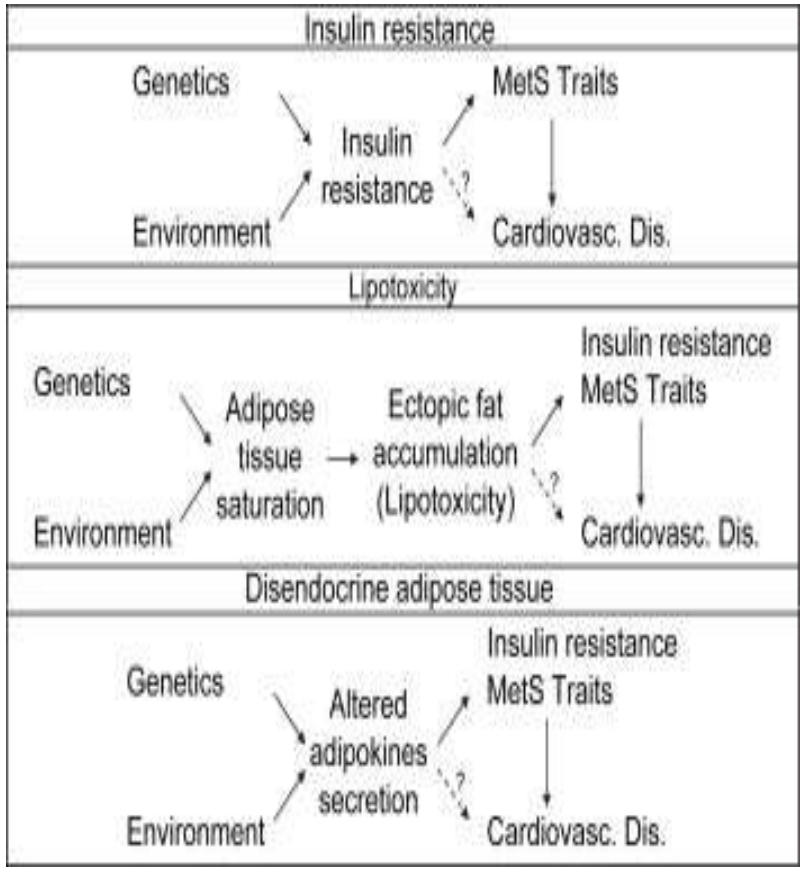
McCracken *et al*., 2018




**Gambar 1.** Peranan obesitas pada sindrom metabolik [16]

Konsensus IDAI


GENETIC VARIATION
ENVIRONMENTAL FACTORS
Central Adiposity
Innate immunity
Adipocyte
Adipokines
Cytokines
Monocyte/ macrophage
Inflammatory markers
Insulin Resistance
↑TG
METABOLIC SYNDROME
↓HDL
↑BP
ATHEROSCLEROSIS
Plaque rupture/Thrombosis
CARDIOVASCULAR EVENTS

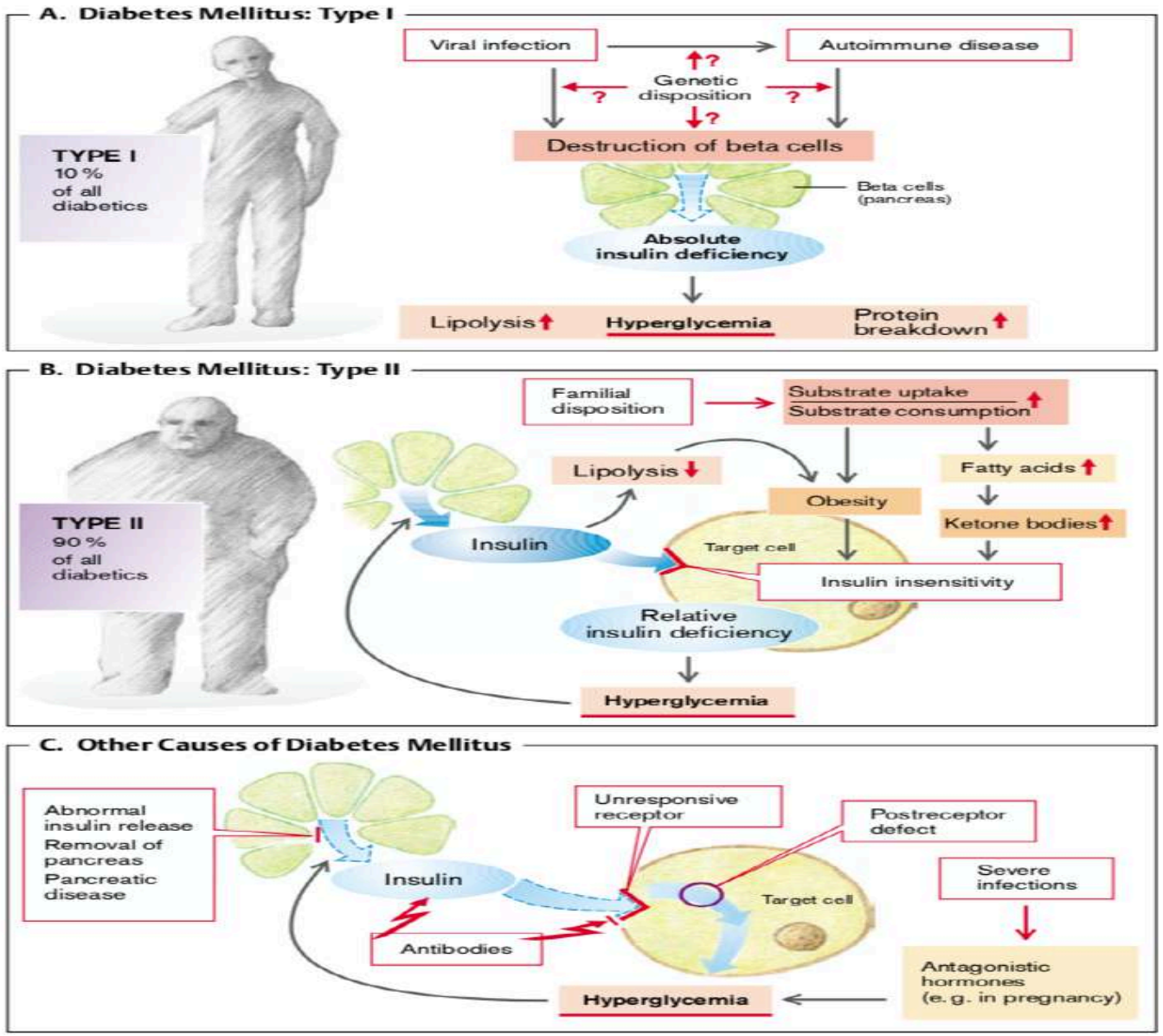

Insulin resistance
Genetics
Environment
Insulin resistance
MetS Traits
Cardiovasc. Dis.
Lipotoxicity
Genetics
Environment
Adipose tissue saturation
Ectopic fat accumulation (Lipotoxicity)
Insulin resistance MetS Traits
Cardiovasc. Dis.
Disendocrine adipose tissue
Genetics
Environment
Altered adipokines secretion
Insulin resistance MetS Traits
Cardiovasc. Dis.

**Tabel 1**. Kriteria sindrom metabolik yang digunakan pada anak[2]

| Studi/ Kelompok | Parameter Diagnostik | Obesitas | Tekanan Darah | Trigliserida | Kolesterol HDL | Intoleransi Glukosa |
|---|---|---|---|---|---|---|
| IDF, 2007 | Obesitas sentral dan 2 faktor risiko lainnya* | ≥10-16 tahun: Lingkar pinggang ≥$P_{90}$ atau nilai batasan dewasa jika lebih rendah | ≥10-16 tahun:Tekanan darah sistolik ≥130 mmHg atau Tekanan darah diastolik ≥85 mmHg | ≥10-16 tahun: ≥150 mg/dL | ≥10-16 tahun: <40 mg/dL | Glukosa darah puasa ≥100 mg/dL atau terdiagnosis diabetes melitus tipe-2 |
| | | 16+ tahun: Kriteria dewasa | 16+ tahun: Tekanan darah sistolik ≥130 mmHg atau Tekanan darah diastolik ≥85 mmHg atau mendapat terapi hipertensi | 16+tahun: ≥150 mg/dL atau terapi spesifik untuk kadar trigliserida yang tinggi | 16+ tahun: <40 mg/dL pada laki-laki dan <50 mg/dL pada perempuan atau mendapat terapi spesifik untuk kolesterol HDL | |
| de Ferranti dkk., 2004 | ≥3 faktor risiko | Lingkar pinggang ≥$P_{75}$ (spesifik terhadap usia dan jenis kelamin, ATP III) | ≥$P_{90}$ (spesifik terhadap usia, jenis kelamin, dan tinggi badan, NHBPEP) | ≥97 mg/dL (*Lipid Research Clinics*) | <50 mg/dL(*Lipid Research Clinics*) | Glukosa darah puasa ≥110 mg/dL |
| Cruz dkk., 2004 | ≥3 faktor risiko | Lingkar pinggang ≥$P_{90}$ (spesifik terhadap usia, jenis kelamin,dan ras NHANES III) | ≥$P_{90}$ (spesifik terhadap usia, jenis kelamin, dan tinggi badan, NHBPEP) | ≥$P_{90}$ (spesifik terhadap usia dan jenis kelamin, NHANES III) | ≤$P_{10}$ (spesifik terhadap usia dan jenis kelamin, NHANES III) | Toleransi glukosa terganggu: (ADA) |
| Goodman dkk., 2004 | ≥3 faktor risiko | Lingkar pinggang ≥102 cm, laki-laki; Lingkar pinggang ≥88 cm, perempuan (ATP III) | Tekanan darah ≥130/85 mmHg (ATP III) | ≥150 mg/dL (ATP III) | ≤40 mg/dL, laki-laki; ≤50 mg/dL perempuan (ATP III) | Glukosa darah puasa ≥110 mg/dL atau toleransi glukosa terganggu (ADA) |
| Weiss dkk., 2004 | ≥3 faktorrisiko | BMI >$P_{97}$ (kurva CDC) atau BMI z *score* ≥2 (spesifik terhadap usia dan jenis kelamin) | ≥$P_{95}$ (spesifik terhadap usia, jenis kelamin, dan tinggi badan, NHBPEP) | >$P_{95}$ (spesifik terhadap usia, jenis kelamin, dan ras, NGHS) | <$P_5$ (spesifik terhadap usia, jenis kelamin, dan ras, NGHS) | Toleransi glukosa terganggu (ADA) |
| Cook dkk., 2003; Ford dkk., 2005 | ≥3 faktor risiko | Lingkar pinggang ≥$P_{90}$ (spesifik terhadap usia dan jenis kelamin, ATP NHANES III) | ≥$P_{90}$ (spesifik terhadap usia, jenis kelamin, dan tinggi badan, NHBPEP) | ≥110 mg/dL (spesifik terhadap usia, NCEP) | ≤40 mg/dL (semua usia/jenis kelamin, NCEP) | Glukosa darah puasa ≥110 mg/dL atau toleransi glukosa terganggu (ADA) |

*ADA: American Diabetes Association, ATP: Adult Treatment Panel III of the NCEP, NGHS: National Growth and Health Survey, NHBPEP: National High Blood Pressure Education Program, CDC: Center for Disease Control and Prevention; BMI: body mass index*

**IDF menyarankan bahwa sindrom metabolik tidak dapat didiagnosis di bawah usia 10 tahun, tetapi penurunan berat badan harus didukung pada anak usia 6 sampai <10 tahun dengan bukti obesitas sentral.*


A. Diabetes Mellitus: Type I
Viral infection
Autoimmune disease
Genetic disposition
TYPE I
10 %
of all
diabetics
Destruction of beta cells
Beta cells (pancreas)
Absolute insulin deficiency
Lipolysis
Hyperglycemia
Protein breakdown
B. Diabetes Mellitus: Type II
Familial disposition
Substrate uptake
Substrate consumption
Lipolysis
Fatty acids
Obesity
Ketone bodies
TYPE II
90 %
of all
diabetics
Insulin
Target cell
Insulin insensitivity
Relative insulin deficiency
Hyperglycemia
C. Other Causes of Diabetes Mellitus
Abnormal insulin release
Removal of pancreas
Pancreatic disease
Unresponsive receptor
Postreceptor defect
Insulin
Target cell
Severe infections
Antibodies
Antagonistic hormones (e.g. in pregnancy)
Hyperglycemia

Coronary heart disease
Hypertension
Atherosclerosis
Congestive heart failure
Cardiovascular Diseases
Obesity
Diabetes
Weight Management
Overweight
Osteoarthritis
Osteoporosis
Musculoskeletal Disorders
Back Pain
Physical Inactivity & Sedentary Lifestyle
Bone fractures & connective tissue
Breast
Colo
Cancer
Lung
Prostate
Psychological Disorders
Mood
Anxiety
Depression
Emphysema
Pulmonary Diseases
Chronic Bronchitis
Asthma

Be Healthy!

Eat well, Live well.


*Being healthy and fit isn't a fad or a trend. Instead, it's a lifestyle*

TERIMA KASIH