

DEFINITION AND DIAGNOSTIC CRITERIA OF CLINICAL OBESITY

تعریف و معیارهای تشخیصی چاقی بالینی

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DEFINITION AND DIAGNOSTIC CRITERIA OF CLINICAL OBESITY

Executive summary

Current BMI-based measures of obesity can both underestimate and overestimate adiposity and provide inadequate information about health at the individual level, which undermines medically-sound approaches to health care and policy.

This Commission* sought to define clinical obesity as a condition of illness that, akin to the notion of chronic disease in other medical specialties, directly results from the effect of excess adiposity on the function of organs and tissues.

*The Lancet Diabetes & Endocrinology Commission

DEFINITION AND DIAGNOSTIC CRITERIA OF CLINICAL OBESITY

There is a clear distinction between **preclinical obesity** and **clinical obesity** for clinical and policy-related purposes. This Commission recommends that BMI should be used only as a surrogate measure of health risk at a population level, for epidemiological studies, or for screening purposes, rather than as an individual measure of health. Excess adiposity should be confirmed by either direct measurement of body fat, where available, or at least one anthropometric criterion (eg, waist circumference, waist-to-hip ratio, or waist-to-height ratio).

DEFINITION AND DIAGNOSTIC CRITERIA OF CLINICAL OBESITY

Introduction

Obesity was first recognised as a disease by WHO in 1948, and more recently also by several medical societies and countries. The current WHO International Classification of Disease labels obesity as “a chronic complex disease”, and gives it a specific code (5B81).

The idea of obesity as a standalone disease entity, however, remains controversial, both within and beyond the medical community. Those who support the recognition of obesity as a disease argue that even people with objective evidence of ill health face substantial barriers to access for healthcare services, in addition to widespread weight-related social stigma.

DEFINITION OF DISEASE AND ILLNESS IN MEDICINE

A distinct pathophysiology that can cause alterations of either a single organ or multiple organs (systemic diseases) , The ability to cause a specific illness, intended as an objective and subjective experience of ill health

What is an illness?

Illness implies a deviation from the normal functioning of organs and tissues or the whole individual, and is typically associated with specific clinical manifestations—physical and biochemical—that can be used as criteria for disease diagnosis.

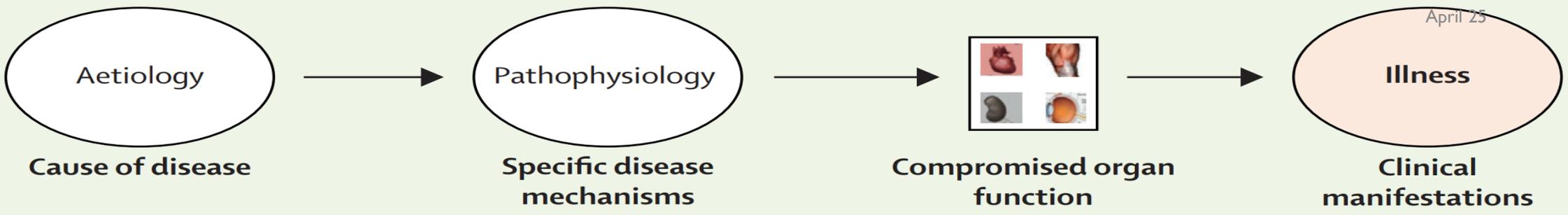


COMMISSIONERS' VIEWS ON OBESITY AS A DISEASE

The idea of obesity as a disease was a controversial subject also within this Commission.

a disease included evidence that excess adiposity is associated with the following: clear pathogenetic mechanisms (eg, inflammation, hormonal imbalances, alterations of appetite or satiety regulation, and insulin resistance); increased risk of mortality; persistence and recidivism despite treatment, consistent with a chronic, relapsing disease process; and the clear association of excess adiposity with complications or related diseases that impair health.

Elements of disease



Traditional framing of obesity

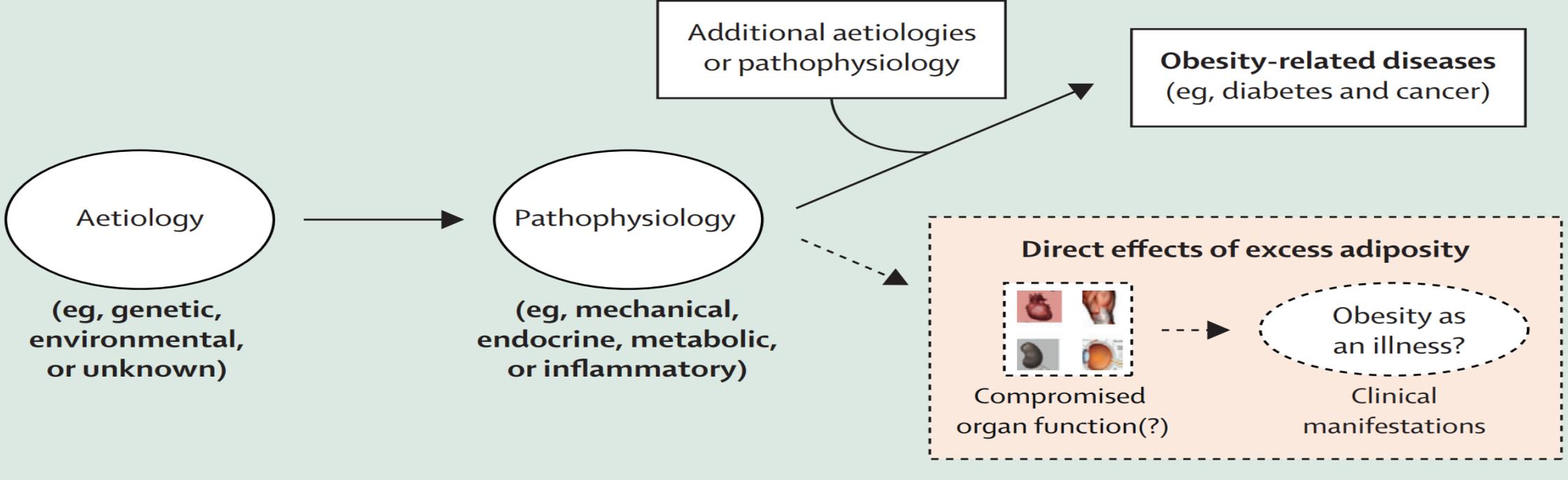


Figure 1: Illness is the missing piece in the traditional framing of obesity

REFRAMING OBESITY AND ITS CLINICAL CHARACTERISATION

- **Classification by cause :**

Depending on the cause of excess adiposity, this Commission distinguishes between **primary, secondary, and genetic** categories of obesity.

Genetic obesity refers to known genetic disorders that are characterised by hyperphagia, other abnormal eating behaviours, and early onset of excess adiposity, usually in the first years of life or during childhood. Genetic forms of obesity include, for example, Prader–Willi syndrome, congenital leptin deficiency, and melanocortin receptor 4 mutations.

REFRAMING OBESITY AND ITS CLINICAL CHARACTERISATION

Secondary forms of obesity are associated with various diseases and conditions (eg, Cushing's syndrome and hypothyroidism), and medications (eg, steroids, anti-depressants, and antipsychotics). In these cases, excess adiposity is associated with other typical signs and symptoms of the responsible disease or condition. Primary obesity results from unknown causes and is the most prevalent form.

- **Phenotypic classification**

Obesity can present with a primarily android phenotype (predominantly central or visceral fat deposition) or a gynoid phenotype (fat stored primarily around the hips and thighs). Central adiposity (android phenotype) and dysfunction of adipose tissue have been associated with greater risk of future metabolic disease and mortality.



LIMITATIONS OF OTHER ANTHROPOMETRIC MEASURES OF ADIPOSITY

- Anthropometric measures have been extensively studied as predictors of metabolic risk, but much less so as a sign of ongoing organ dysfunction caused by obesity. Thus, as for BMI, diagnostic methods exclusively based on anthropometric measures can underdiagnose or overdiagnose illness.
- Adding biochemical markers, such as plasma triglyceride levels, to the measurement of waist circumference—a phenotype described as hyper-triglyceridaemic waist—has been suggested as a useful method for identification of individuals with excess visceral adipose tissue and ectopic fat. Again.

Elevated body weight or BMI

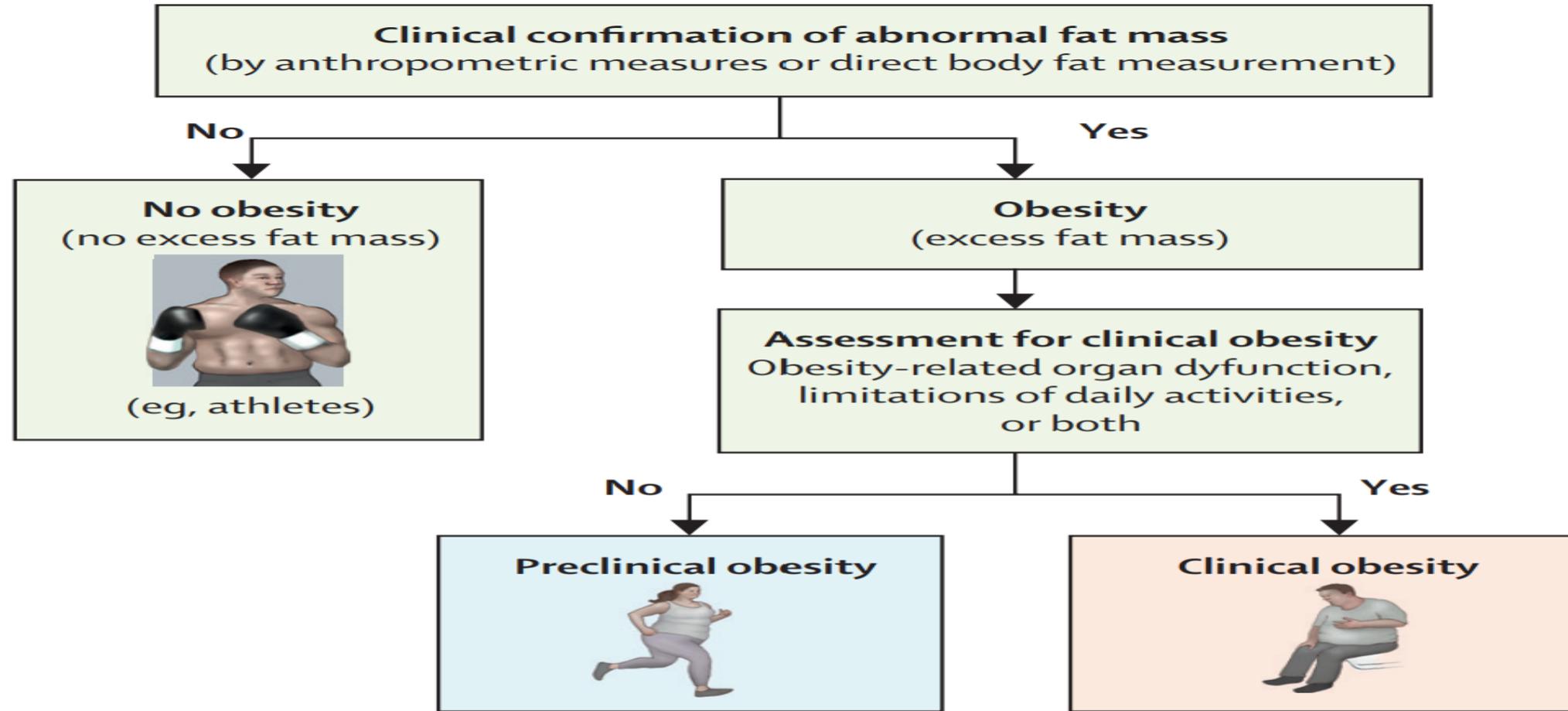


Figure 5: Diagnostic model of clinical obesity

The diagnostic model of clinical obesity includes an anthropometric component (to confirm excess adiposity or obesity status) and a clinical component to verify presence (clinical obesity) or absence (preclinical obesity) of clinical manifestations of organ dysfunction or limitations of an individual's ability to conduct daily activities.

	Preclinical obesity	Clinical obesity
Excess adiposity	 (BMI) +  (Waist circumference, etc)	 (BMI) +  (Waist circumference, etc)
Mechanisms and pathophysiology	Alterations of cells and tissue → Alterations of organ structure	Alterations of organ function → End-organ damage
Clinical manifestations	Minor or absent (substantially preserved organ function)	Signs and symptoms Limitations of daily activities Complications
Detection and diagnosis	Anthropometrics, medical history, review of organ systems, and further diagnostic assessment as needed   	   

Figure 4: Clinical and preclinical obesity

Mechanisms of pathophysiology, clinical manifestations, and methods of detection and diagnosis.

DELPHI RESULTS

All three rounds of Delphi were accomplished with 100% response rate (58 of 58 commissioners). A total of 82 statements (including definitions and diagnostic criteria) had consensus, of which 49 (60%) were unanimous consensus and 33 (40%) near-unanimous.

18 criteria for the diagnosis of clinical obesity in adults (range of consensus 90–100%; table 2), plus 13 criteria in children and adolescents.

	Organ, tissue, or body system	Diagnostic criterion (as agreed by commissioners)	Grade of agreement
Adults			
1	CNS	Signs of raised intracranial pressure such as vision loss and/or recurrent headaches	A, 93%
2	Upper airways	Apnoeas/hypopnoeas during sleep due to increased upper airways resistance	U, 100%
3	Respiratory	Hypoventilation and/or breathlessness and/or wheezing due to reduced lung and/or diaphragmatic compliance	U, 100%
4	Cardiovascular (ventricular)	Reduced Left Ventricular systolic function - Heart Failure with Reduced Ejection Fraction - HFrEF	A, 96%
5	Cardiovascular (atrial)	Chronic/recurrent atrial fibrillation	A, 98%
6	Cardiovascular (pulmonary)	Pulmonary artery hypertension	A, 96%
7	Cardiovascular	Chronic fatigue, lower limb edema due to impaired diastolic dysfunction– Heart Failure with Preserved Ejection Fraction - HFpEF	U, 100%
8	Cardiovascular (thrombosis)	Recurrent DVT and/or pulmonary thromboembolic disease	A, 90%
9	Cardiovascular (arterial)	Raised arterial blood pressure	U, 100%
10	Metabolism	The cluster of hyperglycaemia, high triglyceride levels, and low HDL cholesterol levels	U, 100%
11	Liver	NAFLD with hepatic fibrosis	U, 100%
12	Renal	Microalbuminuria with reduced eGFR	A, 96%
13	Urinary	Recurrent/chronic urinary incontinence	U, 100%
14	Reproductive (female)	Anovulation, oligo-menorrhoea and PCOS	U, 100%
15	Reproductive (male)	Male hypogonadism	A, 96%
16	Musculoskeletal	Chronic, severe knee or hip pain associated with joint stiffness and reduced range of joint motion	U, 100%
17	Lymphatic	Lower limbs lymphedema causing chronic pain and/or reduced range of motion	A, 98%
18	Limitations of day-to-day activities	Significant, age-adjusted limitations of mobility and/or other basic Activities of Daily Living (ADL=bathing, dressing, toileting, continence, eating)	U, 100%



Children and adolescents

1	CNS	Signs of raised intracranial pressure such as vision loss and/or recurrent headaches	U, 100% ^{April 25}
2	Upper airways	Apnoeas/hypopnoeas during sleep due to increased upper airways resistance	U, 100%
3	Respiratory	Hypoventilation and/or breathlessness and/or wheezing due to reduced lung and/or diaphragmatic compliance	A, 98%
4	Cardiovascular	Raised arterial blood pressure	U, 100%
5	Metabolism	The cluster of hyperglycaemia/glucose intolerance with abnormal lipid profile (high triglyceride levels or high LDL cholesterol or low HDL cholesterol)	U, 100%
6	Liver	Elevated LFTs due to metabolic (dysfunction)-associated fatty liver disease (MAFLD)	U, 100%
7	Renal	Microalbuminuria	U, 100%
8	Urinary	Recurrent/chronic urinary incontinence	U, 100%
9	Reproductive (female)	PCOS	A, 98%
10	Musculoskeletal (alignment)	Recurrent/chronic pain or tripping/falling due to pes planus or leg malalignment	A, 96%
11	Musculoskeletal (tibial)	Recurrent/chronic pain or limitation of mobility due to Tibia vara	U, 100%
12	Musculoskeletal (femoral)	Acute and/or recurrent/chronic pain or limitation of mobility or tripping/falling due to slipped femoral capital epiphysis	U, 100%
13	Limitations of day-to-day activities	Significant, age-adjusted limitations of mobility and/or other basic Activities of Daily Living (ADL=bathing, dressing, toileting, continence, eating)	U, 100%

Degree of consensus as agreed by commissioners via a delphi-like method and exact percentage shown for grade of agreement. Grade U=100% agreement (unanimous), grade A=90–99% agreement, grade B=78–89% agreement, grade C=67–77% agreement. DVT=deep vein thrombosis. LFTs=liver function tests. NAFLD=non-alcoholic fatty liver disease. PCOS=polycystic ovary syndrome.

Table 2: Consensus statements: diagnostic criteria for clinical obesity in adults, adolescents, and children



DIAGNOSTIC CRITERIA FOR CLINICAL OBESITY IN ADULTS

CNS: Vision loss, recurrent headaches, or both , (due to raised intracranial pressure)

Respiratory system : Hypoventilation, breathlessness, wheezing, or any combination of these (due to reduced lung compliance, diaphragmatic compliance, or both)

Metabolism: The cluster of hyperglycaemia, high , triglyceride levels, and low HDL , cholesterol

Liver : Metabolic dysfunction-associated , steatotic liver disease with fibrosis

Renal : Microalbuminuria with reduced eGFR



DIAGNOSTIC CRITERIA FOR CLINICAL OBESITY IN ADULTS

Reproductive : Anovulation, oligomenorrhea and polycystic ovary syndrome, male hypogonadism

Limitations of daily activities : Substantial, age-adjusted , limitations of daily living

Upper airways : Apnoea or hypopnoea during sleep (due to increased upper airways resistance)

Cardiovascular system : Heart failure with reduced ejection fraction (due to reduced left ventricular systolic function) , Chronic fatigue and lower limb oedema (due to impaired diastolic function—heart failure with preserved ejection fraction), Chronic or recurrent atrial fibrillation, Pulmonary artery hypertension, Recurrent deep-vein thrombosis or pulmonary embolism , Raised arterial blood pressure



DIAGNOSTIC CRITERIA FOR CLINICAL OBESITY IN ADULTS

Urinary system : Recurrent or chronic urinary , incontinence

Musculoskeletal system : Chronic, severe knee or hip pain , (associated with joint stiffness and reduced range of motion)

Lymphatic system : Lower limb lymphoedema (causing chronic pain, reduced range of motion, or both)



DIAGNOSTIC CRITERIA FOR CLINICAL OBESITY IN CHILDREN AND ADOLESCENTS

CNS: Vision loss, recurrent headaches, or both , (due to raised intracranial pressure)

Respiratory system : Hypoventilation, breathlessness, wheezing, or any combination of these (due to reduced lung compliance, diaphragmatic compliance, or both)

Metabolism: The cluster of hyperglycaemia, or glucose intolerance, with high triglyceride levels, high LDL cholesterol, or low HDL cholesterol

Liver : Elevated liver function tests due to , metabolic dysfunction-associated , steatotic liver disease



DIAGNOSTIC CRITERIA FOR CLINICAL OBESITY IN CHILDREN AND ADOLESCENTS

Renal : Microalbuminuria

Reproductive : Polycystic ovary syndrome

Limitations of daily activities : Substantial, age-adjusted limitations of mobility, daily living, other basic , activities of living, or both

Upper airways : Apnoea or hypopnoea during sleep (due to increased upper airways resistance)

Cardiovascular system : Raised arterial blood pressure



DIAGNOSTIC CRITERIA FOR CLINICAL OBESITY IN CHILDREN AND ADOLESCENTS

Urinary system : Recurrent or chronic urinary , incontinence

Musculoskeletal system ; Recurrent or chronic, severe knee pain, or tripping or falling (due to pes planus or leg malalignment) , Acute pain, recurrent or chronic pain, limitations of mobility, tripping or, falling, or any combination of these (due to slipped femoral capital epiphysis)

CLINICAL ASSESSMENT

People with confirmed excess adiposity should be assessed for clinical obesity. **This assessment should include:**

- A person's medical history
- A physical examination
- Standard laboratory tests, including full or complete blood count, glycaemia, lipid profile, and renal and liverfunction tests
- Additional diagnostic tests as appropriate if the patient's medical history or physical examination, or standard laboratory tests, or both suggest the possibility of one or more obesity-induced organ or tissue dysfunction

INTERVENTIONS FOR CLINICAL OBESITY (PRINCIPLES)

The choice of the intervention for clinical obesity (ie, lifestyle, pharmacological, psychological, or surgical) should be based on:

- Individual risk–benefit assessment
- Available clinical evidence that the intervention has reasonable chances to improve clinical manifestations and quality of life or reduce risk of disease progression and mortality.

MANAGEMENT OF PRECLINICAL OBESITY

Objective: Reduce the risk of developing clinical obesity and related conditions through evidence-based health advice and equitable healthcare access.

Approach: Tailored to individual risk–benefit profiles based on severity of adiposity, presence of risk factors, and coexisting conditions.

Interventions could include lifestyle changes, pharmacological treatments, psychological support, or, in rare cases, surgery.

MANAGEMENT OF PRECLINICAL OBESITY

Counselling: Individuals with low risk should focus on lifestyle advice and monitoring health indicators over time.

High-Risk Cases: May warrant medications or surgery to reduce risk quickly, especially to support other treatments like transplantation or cancer care. **Treatment Goals:** Emphasize risk reduction for preclinical obesity, with lower urgency and intensity than for clinical obesity. **Future Needs:** Development of scoring systems for better risk assessment and decision-making in treatment.

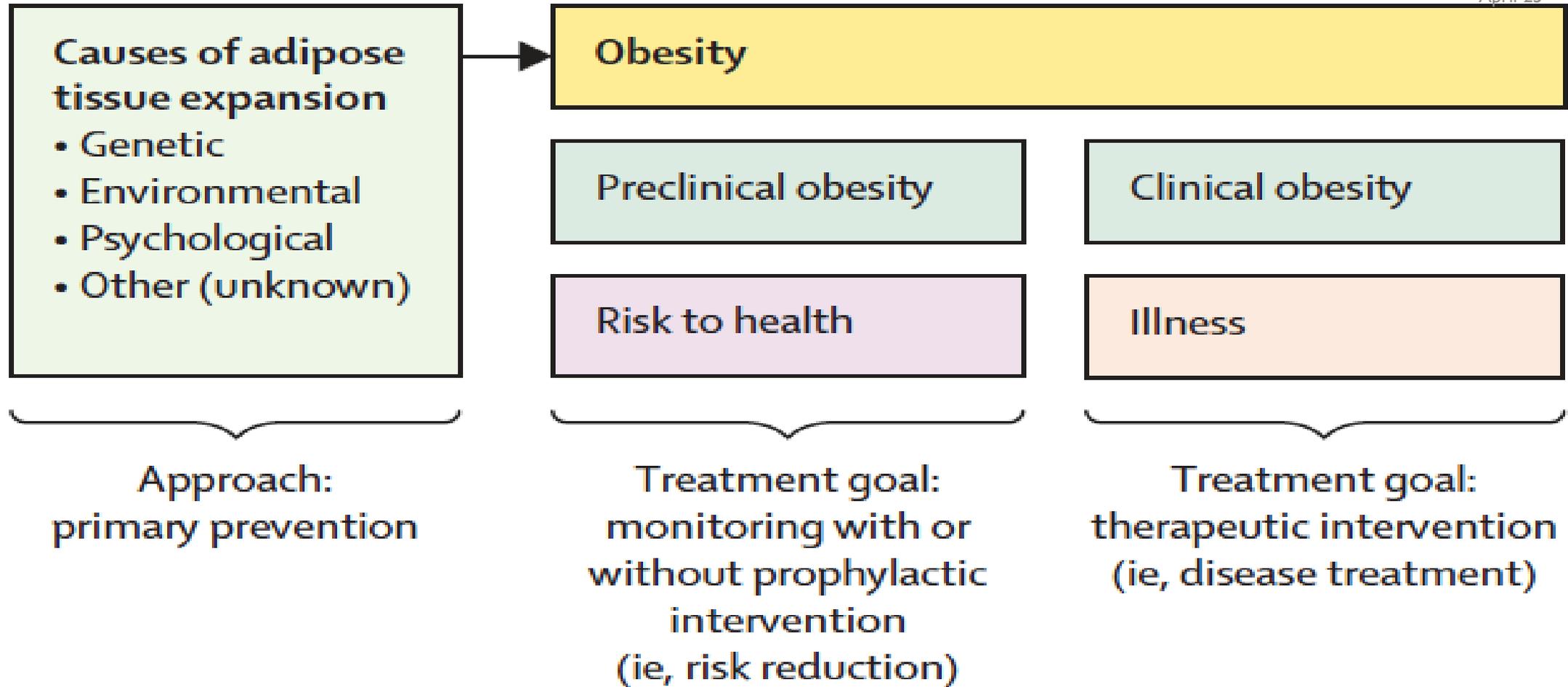


Figure 9: Goals of treatment in preclinical and clinical obesity

DEFINITION AND DIAGNOSIS OF CLINICAL OBESITY

- **What is clinical obesity?**
- **What characterises clinical obesity?**
- **Is clinical obesity the same as metabolically unhealthy obesity?**
- **How to diagnose clinical obesity?**
- **How should clinical obesity be managed?**

DEFINITION OF PRECLINICAL OBESITY

- **What is preclinical obesity?**
- **Is preclinical obesity a pre-disease state?**
- **Is preclinical obesity the same as overweight or pre-obesity? Is preclinical obesity the same as metabolically healthy obesity?**
- **What are the clinical implications of preclinical obesity?**
- **How should preclinical obesity be managed?**

CONCLUSION

The idea of obesity as a disease is at the centre of one of the most controversial and polarising debates in modern medicine, with broad and far-reaching implications for people affected and the society as a whole.

It is our hope that such reframing can inform public health policies, facilitate identification of appropriate targets for prevention versus treatment strategies, and contribute to overcoming misconceptions that reinforce weight-based bias and stigma.



References:

The Lancet Diabetes & Endocrinology Commission

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Thank you
for your attention

