

Commentary

B is for body fat: a practical implementation of the new clinical obesity definition into preventive cardiology clinic

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ABSTRACT

Obesity is a multifactorial disease with increasing prevalence in the United States and a major contributor to cardiovascular disease. The Lancet Commission recently issued a new definition of obesity to better capture individuals with excess body fat and metabolic consequences of adiposity. In this article, we provide a practical implementation of this new definition into a clinical practice using the ABC model of cardiovascular prevention, a framework to characterize major atherosclerotic risk factors. We recommend to emphasize B is for body fat, with routine measurement using body mass index and waist circumference, tailored history and physical exam to identify obesity-related diseases, lifestyle guidance, and pharmacological recommendations.

1. Manuscript

Obesity is a multifactorial illness with complex interactions from a variety of biological [1], socioeconomic [2], and environmental factors [3]. The obesity epidemic is worsening, with a prevalence rate expected to exceed 50 % of individuals in the United States by 2030 [4]. Obesity is a major contributor to cardiovascular disease (CVD) [5], leading to increased incidence of coronary artery disease, heart failure, and atrial fibrillation [6]. CVD is disproportionately the leading cause of death in individuals with obesity, accounting for over 60 % of deaths [7]. Thus, there is an urgent need to improve our diagnosis of obesity and refine therapeutic strategies to prevent the progression of cardiovascular disease.

To address this need, in January 2025 the Lancet Commission outlined a new definition of obesity, stratifying individuals by preclinical and clinical obesity [8]. Obesity is defined as excess body fat assessed by at least two different anthropometric measurements such as body mass index (BMI) and waist circumference, direct measurement via imaging, or if the BMI is extreme (i.e. ≥ 40 kg/m²) [8]. Clinical obesity is defined as organ dysfunction or significant functional limitation due to excess body fat whereas preclinical obesity has no corresponding deficits [8]. The Lancet Commission has provided guidance on how to define obesity to account for different regions or ethnicities and suggested a rubric for

how to diagnose clinical obesity [8]. However, there are ongoing needs to discuss clinical implementation of this obesity definition and how such change may inform risk assessment and treatment goals.

One approach to improve prevention of CVD is the ABC framework [9,10], which simplifies the health criteria described by the American Heart Association (AHA) to promote cardiovascular health [11]. In this framework, A refers to assessment of risk, anti-inflammation, anti-platelet/anticoagulation therapy; B corresponds to body weight and blood pressure; C for cigarette cessation and cholesterol; D for dream (sleep), diet, digital health, and diabetes; E for exercise; F for factors of the environment; and G for genetics [10].

In this article, we present a rationale for the new clinical obesity definition from the Lancet Commission, practical implementation for integrating this definition into a routine preventive cardiology clinic, and an outline of knowledge gaps and future research directions.

2. Rationale of the new obesity definition

The current definition of obesity by the WHO classifies obesity based on BMI, defining obesity as at least BMI of 30 kg/m², with further subdivisions into Class I (30–34.9 kg/m²), Class II (35–39.9 kg/m²), and Class III (≥ 40 kg/m²) or severe obesity [12]. While the WHO obesity definition is widely adopted in current research, clinical practice, and

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health policy, it has numerous limitations for clinical practice. BMI does not differentiate between fat and lean mass, account for visceral fat distribution, predict disability or functional limitation, or provide any information about obesity-related organ dysfunction [13], simultaneously underestimating and overestimating the consequences of adiposity in select populations [14].

The goal of the Lancet Commission was to define clinical obesity as a chronic illness, improve objective criteria for disease diagnosis beyond BMI, and aid in decision-making for future public health and therapeutic strategies [8]. With their new definition, obesity is instead defined as a condition characterized by excess adiposity or body fat, stratified by preclinical and clinical obesity [8]. Preclinical obesity is a condition of excess body fat associated with variable level of health risk but no ongoing illness. Excess body fat can be defined by at least two separate measurements of body size or direct fat measurement, such as with a DEXA scan [8]. In individuals with extreme body fat (i.e. BMI ≥ 40 kg/m²), excess body fat can be pragmatically assumed [15]. Additional body size measurements for excess body fat beyond BMI include a waist circumference at least 102 cm for men or 88 cm for women, waist-to-hip ratio of at least 0.9 for men or 0.85 for women, or waist-to-height ratio of at least 0.5 for all, with a supplemental document incorporating variation for different regions or ethnic groups [8].

Clinical obesity is defined as a chronic disease characterized by signs and symptoms of ongoing organ dysfunction and/or reduced ability to conduct activities of daily living due to excess body fat [8]. Examples of adiposity-related organ dysfunction for clinical obesity classification include heart failure, osteoarthritis, metabolic syndrome derangements, or physical immobility [6,8]. The Lancet Commission has provided a supplement of 12 different organ systems corresponding to obesity-related dysfunction with guidance for medical history, physical examination, and studies to aid in the diagnosis of clinical obesity [8].

3. Implementation into a preventive cardiology clinic

The Johns Hopkins Ciccarone Center for the Prevention of Cardiovascular Disease established an ABC model as a simplified framework to allow clinicians to focus on the most pertinent aspects of CVD prevention [10]. This ABC framework is regularly updated and revised to incorporate the latest practice changing updates in cardiology, recently incorporating components such as digital health, coronary calcium scoring, and genetics [10]. Now that the Lancet Commission provided guidance for a new definition and diagnostics of obesity, we can incorporate these elements into the ABC prevention framework.

We suggest amending the previous ABC mnemonic “B is for body weight” to “B is for body fat,” in line with the Lancet Commission focus on excess adiposity. Body weight alone is a poor surrogate for adiposity at an individual level. For example, individuals with normal weight can have excess body fat, which is more prominent in certain ethnicities (e.g. East Asians) [16] or elderly populations with increased bone or skeletal muscle loss [14]. Conversely, BMI can over diagnose obesity in high performance athletes with increased lean mass and minimal fat [17]. Body weight and BMI have a U-shaped curve of association with negative cardiovascular outcomes with factors such as smoking, occult malignancy, and frailty influencing the distribution [18]. On the other hand, visceral adipose tissue (VAT) and ectopic fat pads have a strong association with CVD risk, regardless of BMI [19]. While imaging is the gold standard to quantify VAT, anthropometric waist measurements (i.e. waist circumference, waist-to-hip ratio, waist-to-height ratio) better correlate with VAT and overall CVD risk [20], and are non-invasive measurements that are easier to assess in a preventive cardiology setting.

We recommend measuring BMI and waist circumference as initial screening tools for obesity, with medical staff training to accurately measure these metrics. Obesity should be characterized based on region-specific thresholds of BMI and waist circumference (Table 1) [8]. Waist circumference should be measured horizontally using a flexible,

Table 1
Body mass index and waist circumference criteria for obesity in different regions.

Region	Body mass index (kg/m ²)	Waist Circumference (cm)
Bangladesh	25	80 (F) / 90 (M)
China	28	85 (F) / 90 (M)
Hong Kong	25	80 (F) / 90 (M)
India	25	80 (F) / 90 (M)
Japan	25	90 (F) / 85 (M)
South Korea	25	85 (F) / 90 (M)
Malaysia	27.5	80 (F) / 90 (M)
Pacific Islanders	32	88 (F) / 102 (M)
Philippines	25	80 (F) / 90 (M)
Singapore	27.5	80 (F) / 90 (M)
Sri Lanka	25	80 (F) / 90 (M)
Taiwan	27	80 (F) / 90 (M)
Thailand	25	80 (F) / 90 (M)
Vietnam	25	80 (F) / 90 (M)
Others	30	88 (F) / 102 (M)

non-stretchable tape at the midpoint between the bottom of the rib cage and the top of the iliac crest when the patient is in the upright position with a relaxed abdomen after normal exhalation [21]. If there is significant discrepancy between BMI and waist circumference, waist-to-hip or waist-to-height ratios can serve as a tiebreaker to identify obesity [22]. We recommend BMI and waist circumference as first line measurements of adiposity as both these metrics are simple to calculate, well established in clinical guidelines, and less sensitive to measurement error [13,21,23]. We also recommend to evaluate fat mass in imaging when available, such as in a DEXA scan ordered for other reasons like osteoporosis screening [24].

Once obesity is accurately defined, we recommend screening for the clinical manifestations of excess adiposity with a comprehensive history and physical examination. Examples of diseases pertinent to a cardiovascular disease prevention clinic include obstructive sleep apnea, heart failure, pulmonary hypertension, atrial fibrillation, thromboembolism from deep vein thrombosis or pulmonary embolism, hypertension, metabolic syndrome, chronic kidney disease, metabolic associated steatosis liver disease, and functional limitation [6]. Subsequent diagnostic tests including chemistry tests, electrocardiograms, echocardiograms, coronary artery calcium scans, lipid panels, sleep studies, liver ultrasound elastography and others should be ordered based on clinical suspicion when indicated (Fig. 2).

In subsequent visits, we recommend measuring both BMI and waist circumference and assessing for progression of obesity-related diseases to evaluate for the improvement or worsening of clinical obesity [21]. Longitudinal assessment of BMI and waist circumference better prognosticate overall risk and indicate contributions of fat relative to muscle [20].

As the obesity epidemic worsens, there may be a growing need for specialized cardiometabolic clinics to enable multidisciplinary collaboration for early detection of obesity-related diseases and provide access to advanced therapies [25]. Formalized training in cardiometabolic medicine with emphasis on cardiovascular, endocrine, renal, and hepatic health can help clinicians manage complex cases and improve clinical care [25].

4. Treatment of clinical obesity

The management of clinical obesity includes healthy diet and physical activity, with medications and surgery referral in selected individuals (Fig. 1).

Dietary intake is an important modifiable factor that influences both clinical obesity and CVD risk [26]. Consumption of energy-dense, ultra-processed foods with increased saturated fats, added sugars, and refined carbohydrates is associated with both increased adiposity and CVD [26]. The AHA recommends the following components in a

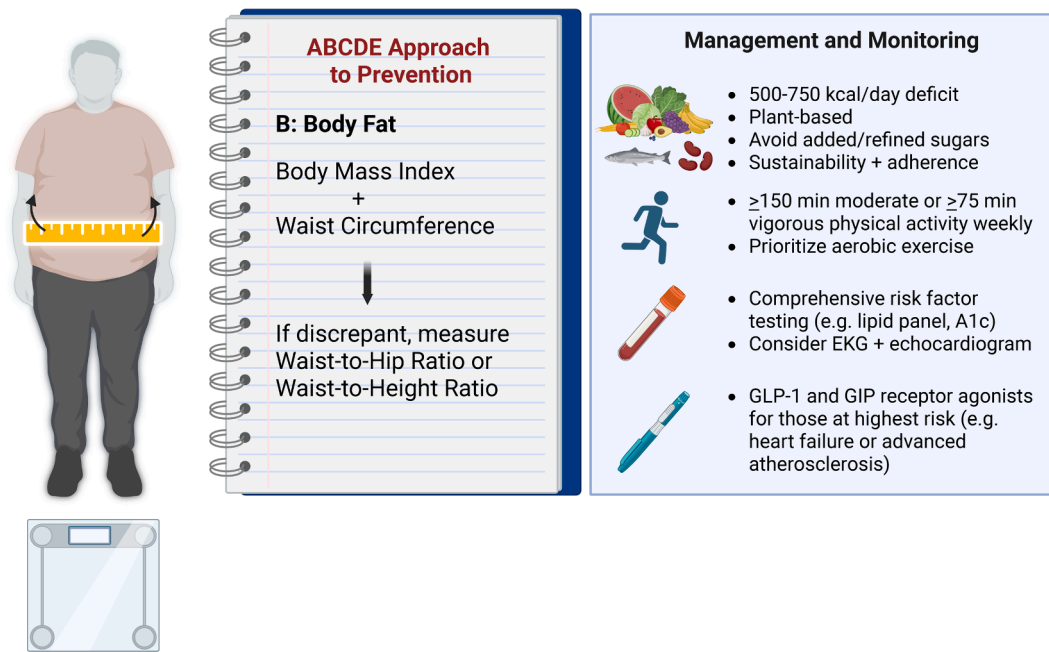


Fig. 1. Schematic of integrating the new clinical obesity definition into the ABC model of CVD prevention. B corresponds to body fat. Excess body fat can be assessed with a combination of anthropometric measurements such as body mass index and waist circumference, with other waist-to-hip ratio or waist-to-height ratio to supplement if values are discrepant. The management of clinical obesity includes diet, physical activity, assessment of risk factors and obesity-related diseases, and medications/surgery referral in select individuals.

heart-healthy diet to prevent CVD – a wide variety of fruits and vegetables, whole grains, plant-based and lean proteins like fish and poultry, unsaturated fats, minimal processed foods and added sugars, increased fiber, reduced sodium intake, and limited alcohol consumption [26]. A 500–750 kcal/day deficit is also recommended in individuals with obesity to promote weight loss [26]. Specific diets like a plant-based diet, Dietary Approaches to Stop Hypertension (DASH) or Mediterranean diet have been consistently associated with improvements in blood pressure, fasting blood glucose, and cholesterol, cumulatively decreasing the risk of CVD even if the magnitude of weight loss is modest [27,28].

Physical activity provides cardiovascular benefit and modest weight loss. Current guidelines recommend a minimum of 150 min per week of moderate-intensity aerobic exercise or 75 min per week of vigorous activity [29]. Additionally, a regular physical activity regimen (e.g. 10,000 steps per day) is associated with decreased all-cause mortality [30]. Although exercise and physical activity result does not result in much weight loss, the cardioprotective effects of sustained exercise are numerous, including improvement in endothelial function, insulin sensitivity, lipid metabolism, inflammation, and visceral adiposity [31]. Previous studies have shown that individuals with elevated BMI and normal cardiorespiratory fitness have significantly reduced CVD events [32,33].

In select individuals, pharmacotherapy is needed to treat clinical obesity and reduce CVD [34,35]. The Food and Drug Administration has approved several medications for the treatment of obesity and associated clinical conditions, which include orlistat, phentermine-topiramate (Topamax tablets), naltrexone-bupropion, as well as GLP-1 and GIP receptor agonists [34]. A few of these therapies, notably GLP-1 and GIP receptor agonists, have shown significant weight loss exceeding 10–15 % of body weight and promising benefits for CVD reduction as well as other obesity-driven diseases [34]. In the SELECT trial, semaglutide (GLP-1 agonist) led to a 10 % reduction in body weight and 1.5 % absolute risk reduction in further CVD over four years in individuals with a BMI ≥ 27 kg/m², existing CVD, and without diabetes [36]. In the SUMMIT trial, tirzepatide (GLP-1/GIP agonist) showed a 6.2 % absolute risk reduction in heart failure events in patients with a BMI ≥ 30 kg/m²

and heart failure with preserved ejection fraction [37]. Other studies have shown the beneficial impact of GLP-1 and GIP agonists for other metabolic diseases, including the regression of diabetes, improvement in sleep apnea, and improvement in metabolic associated steatotic liver disease [38–40]. Notably, there are additional pharmacotherapies for obesity in the clinical pipeline, including GIP agonists, glucagon agonists, amylin analogs, activin receptor inhibitors, calcitonin agonists, and peptide YY compounds [41]. Further studies are ongoing to investigate the application of these novel medications in preventing other obesity-related diseases.

Metabolic and bariatric surgery provides greater weight reduction and cardiovascular risk factor improvement than pharmaceutical drugs [34]. The 2022 ASMBS guidelines have expanded the indications for metabolic and bariatric surgery, now indicated for individuals with BMI ≥ 35 kg/m², BMI 30–34.9 kg/m² and a metabolic-associated disease (e.g., diabetes, obstructive sleep apnea), or BMI ≥ 27.5 kg/m² with a metabolic-associated disease in specific populations (e.g., Asians) [42]. Common procedures include Roux-en-Y gastric bypass, sleeve gastrectomy, and single anastomosis duodenoileostomy with sleeve gastrectomy (SADI-S), with patients typically achieving a 50–70 % loss of excess body weight within the first year [34]. Bariatric surgery confers improvement in several cardiovascular risk factors such as in blood pressure, lipid profiles, and glycemic control [42]. Large cohort studies and meta-analyses demonstrate significant reductions in major adverse cardiovascular events, stroke, heart failure, and all-cause mortality [43–46]. These findings place metabolic and bariatric surgery as the most effective means to reduce body weight and improve cardiovascular outcomes.

5. Knowledge gaps and future directions

There are several important knowledge gaps with the current Lancet Commission definition of obesity that would benefit from further investigation. One knowledge gap is that there is still uncertainty and debate about how to apply this new definition until studies about the epidemiology of preclinical and clinical obesity emerge [47]. The Lancet Commission recommends clinical monitoring and lifestyle counseling

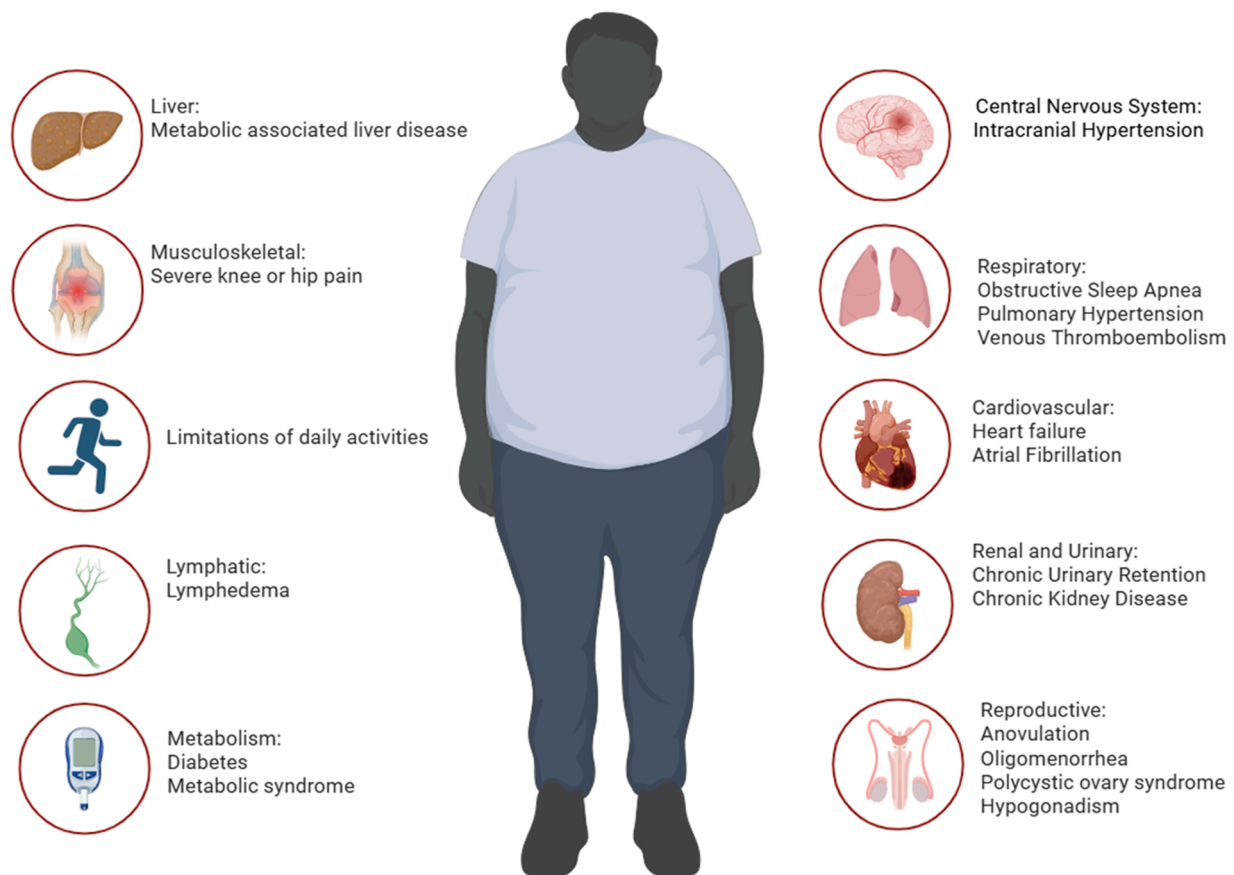


Fig. 2. Examples of qualifying conditions that constitute clinical obesity. Individuals with obesity and at least one of these qualifying diseases have clinical obesity whereas individuals with none of these diseases have preclinical obesity.

for individuals with preclinical obesity and evidence-based medical treatment based on risk and patient preference for those with clinical obesity [8]. **However, if preclinical obesity is uncommon, then distinguishing it from clinical obesity is unlikely to make a meaningful difference.** Another gap is that the natural history of preclinical obesity is unclear [47]. Whereas many cardiovascular conditions have established guidelines for surveillance and diagnostic studies for asymptomatic disease (e.g. serial echocardiograms for asymptomatic severe aortic stenosis), the guidance for preclinical obesity is not defined yet. However, recent guidelines and consensus statements from major cardiovascular societies have begun to incorporate recommendations involving weight loss therapy for CVD risk reduction [48]. For example, the 2024 ESC Clinical Consensus Statement on Obesity provides a Class 2a indication for semaglutide to reduce CVD risk among individuals with BMI ≥ 27 kg/m² with chronic coronary syndrome without diabetes [48]. Additionally, the Obesity Association and the American Diabetes Association is currently developing the Standards of Care and Obesity –2025 to provide evidence based recommendations for screening, diagnosis, and management of obesity and related complications [49]. Some key questions include: the transition length from preclinical to clinical obesity, genetics and prognostic biomarkers for disease progression, how to improve healthcare literacy, and public health implications to prevent the development of clinical obesity [8].

One significant challenge for obesity assessment is that current clinical trials and insurance coverage are dependent on BMI criteria, which is problematic for individuals with increased adiposity at normal BMI levels [13]. We recommend greater advocacy for waist circumference in routine clinical practice through society guidelines, regulatory frameworks, pharmaceutical agencies, and clinical education, as numerous studies have shown waist circumference is a strong

independent predictor of visceral adiposity and cardiovascular outcomes [20,21]. As obesity is increasing in prevalence but still grossly undertreated, we believe that greater recognition and characterization of obesity will improve overall care. Incorporation of obesity frameworks beyond BMI, such as with the Lancet Definition, may help achieve this goal, especially given accurate risk factor diagnosis is paramount in preventive cardiology.

6. Conclusion

The purpose of our review was to integrate the new Lancet Commission definition of obesity into a simplified ABC framework to improve the focus on body fat/adiposity rather than body weight, provide practical guidance for therapy, monitor adiposity longitudinally, and prevent future CVD.

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Sohail Zahid: Writing – review & editing, Writing – original draft, Conceptualization. **Allison W. Peng:** Writing – review & editing, Writing – original draft, Conceptualization. **Alexander C. Razavi:** Writing – review & editing, Writing – original draft, Conceptualization. **Zhiqi Yao:** Writing – review & editing, Writing – original draft, Conceptualization. **Roger S. Blumenthal:** Writing – review & editing, Supervision, Conceptualization. **Michael J. Blaha:** Writing – review & editing, Supervision, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests

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