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Clinical Practice Guideline Communication

Defining disease or delaying care? A conceptual and clinical appraisal of the Lancet obesity framework

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Abstract

The *Lancet Diabetes & Endocrinology Commission* undertook the complex task of addressing limitations in existing definitions and classification of obesity. Its consensus framework moves beyond body mass index (BMI) toward direct or surrogate measures of excess adiposity and distinguishes "clinical obesity" (excess adiposity plus objective organ/system dysfunction or functional limitation attributable to adiposity) from "preclinical obesity" (excess adiposity without such evidence). The Endocrine Society (ES) recognizes the substantial effort and expertise underlying this work and its intent to improve diagnostic precision and therefore provides an independent appraisal of the framework's conceptual coherence, empirical support, operational feasibility, and implications for coverage, equity, and clinical implementation. The shift away from BMI-only screening is supported by evidence that central adiposity and fat distribution better predict cardiometabolic risk than BMI alone. Validation studies (All of Us; UK Biobank) demonstrate elevated risk among individuals classified with "preclinical" obesity and even higher risk among those with "clinical" obesity, underscoring the importance of safeguards against undertreatment in the preclinical state. At the same time, lack of standardized anthropometric measurement protocols, increased resource utilization, limited distinction between subcutaneous and visceral fat depots, and insufficient data regarding the long-term implications of obesity-related disease absence prompted the ES to pause before fully endorsing the Commission's consensus. Accordingly, we outline an evaluation framework addressing available evidence, feasibility, coverage and equity considerations, and clinical impact. We advocate harmonization with established staging systems (EOSS, EASO), explicit measurement protocols, age-, sex-, and ancestry-specific thresholds, integration of mental health and patient-reported outcomes, and policies that prevent unintended care restrictions. The Commission's reframing represents a meaningful conceptual advance; broader adoption will require practical and equitable implementation.

Keywords obesity diagnosis, clinical obesity, preclinical obesity, central adiposity, risk stratification, organ dysfunction, anthropometric measures, equity, Lancet Commission on Obesity

Obesity is a major global health challenge and a leading cause of morbidity and premature mortality (1-3). It affects cardiometabolic, respiratory, musculoskeletal, hepatic, reproductive, and neurocognitive systems, driving substantial disease burden and health care expenditure worldwide (4, 5). The World Health Organization (WHO) defines obesity by body mass index (BMI) of 30 kg/m^2 or greater, categorized as Class I (30-34.9), Class II (35-39.9), and Class III (40 or greater). This classification is simple and reproducible, which supports its use in clinical practice, epidemiological research, and public policy. Yet BMI does not distinguish adipose from lean tissue, capture fat distribution, or directly reflect organ dysfunction.

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These limitations overestimate risk in muscular individuals and underestimate it in persons with excess visceral or ectopic fat (6, 7). As with many biomarkers, BMI requires complementary measures for accurate risk assessment.

To address these issues, *The Lancet Diabetes & Endocrinology Commission* was established to conceptualize clinical obesity as a chronic illness, develop more accurate diagnostic standards extending beyond BMI, and guide evidence-based approaches to treatment and public health policy (4). The Commission introduced criteria linking excess adiposity causally to measurable tissue and organ dysfunction to define *clinical obesity*. It also created a *preclinical obesity* category for individuals with excess adiposity but no detectable metabolic or organ dysfunction. Although the influence of fat distribution and adipose-tissue function on disease risk is well established (8-11), the framework's novelty lies in formally codifying a disease state based on demonstrable pathophysiologic consequences rather than anthropometric thresholds alone (4).

This *Guideline Communication* is published under the auspices of the Endocrine Society to provide an independent appraisal of the *Lancet Commission* framework. The Commission should be commended for highlighting the diagnostic and policy challenges facing the field and for undertaking the complex task of building consensus among diverse stakeholders. Given the Endocrine Society's decision not to co-sponsor the Lancet Commission's report, the purpose of this *Guideline Communication* is to articulate a constructive appraisal of the framework's conceptual and operational implications and suggest refinements that may enhance its feasibility, equity, and clinical coherence. Definitions in this area are likely to evolve as our understanding of adipose biology, still nascent in many respects, continues to mature.

Definition of preclinical and clinical obesity

As outlined by *The Lancet Diabetes & Endocrinology Commission*, diagnosing clinical obesity requires meeting 2 criteria. First, an *anthropometric criterion*: excess body fat must be demonstrated via direct measurement or by at least one additional anthropometric index alongside BMI—waist circumference (WC), waist-to-hip ratio, or waist-to-height ratio (WHtR)—using validated techniques and cutoffs tailored to age, sex, and ethnicity. For BMI >40 kg/m^2, excess adiposity is presumed. For BMI near but below conventional thresholds, direct fat assessment or concordant abnormalities in 2 anthropometric indicators may establish excess adiposity irrespective of BMI. Second, a *clinical criterion*: evidence of (1) dysfunction in one or more organ systems due to excess adiposity or (2) age-adjusted impairment in mobility or basic activities of daily living (4).

Preclinical obesity denotes excess adiposity identified by anthropometric measures in the absence of detectable organ dysfunction, yet with increased risk of progression to clinical obesity and to conditions such as type 2 diabetes (T2DM), cardiovascular disease (CVD), and certain cancers (12-16). The Commission frames this state as heightened future risk rather than present illness and notes that no single measure currently has sufficient accuracy to function as a stand-alone clinical test.

Empirical applications of the Lancet Commission framework

Analyses from nationally representative and large cohort datasets illustrate how the Commission's criteria reshape obesity prevalence estimates and risk stratification. In NHANES among US adults aged 20 to 59, obesity prevalence defined by BMI alone (39.7%) nearly matched prevalence after confirming excess adiposity with WC or body fat measures (39.1%); nearly all individuals classified as having obesity by BMI alone (98.4%) also had confirmed excess adiposity by additional measures (17).

In All of Us, applying the Commission framework increased obesity prevalence from 42.9% to 68.6%, driven largely by anthropometric-only obesity (25.9%) (12). In UK Biobank, the framework classified 33.1% of participants as having obesity vs 24.4% by BMI alone, with another analysis estimating prevalence at 67.8% (14, 15). Nearly all individuals identified as having obesity by BMI were captured within the Commission's BMI-plus-anthropometric category (12). Together, these findings suggest that BMI performs well as an indicator of excess adiposity in US adults and that additional anthropometric testing provides limited value for individuals already exceeding the BMI $\geq$ 30 kg/m^2 threshold.

Across cohorts, the Commission framework provided sharper risk stratification than BMI alone. In All of Us, adjusted hazard ratios (HRs) for incident diabetes, CVD, and mortality were 3.21, 1.70, and 1.21, respectively, under the Commission criteria, compared with 2.60, 1.39, and 1.10 using BMI alone. *Clinical obesity* carried the highest risk (HRs: T2DM 6.11; CVD 5.88; mortality 2.71), whereas *preclinical obesity* showed intermediate diabetes risk and modest CVD risk without excess mortality. Importantly, organ dysfunction in the absence of obesity conferred substantial hazard (HRs: 2.50 for T2DM; 4.68 for CVD; 2.82 for mortality), underscoring the prognostic weight of dysfunction itself (12).

Among individuals with clinical obesity, the most frequent manifestations were hypertension (64.6%), physical limitation (28.8%), and obstructive sleep apnea (22.8%) (12, 17). The anthropometric-only phenotype tended to be older, more often male, and within US data—of higher socioeconomic status and more frequently White (12, 17). UK Biobank showed clearer mortality risk separation using the Lancet framework, particularly for CV mortality (13). For cancer mortality, both frameworks show smaller, broadly similar risk elevations (HRs <2) (14).

The misclassification problem in obesity assessment

Clinical assessment of obesity still begins with BMI, yet this metric captures only a limited dimension of adiposity. Although BMI correlates with Dual X-ray Absorptiometry (DXA) measured fat mass, it cannot distinguish lean from fat tissue or account for fat distribution, which drives much of the cardiometabolic risk. Direct fat measures are more accurate but impractical at scale, leading guidelines to rely on anthropometric combinations with numerical cut points that vary across age, sex, and ancestry and introduce inconsistency. Using fixed thresholds for a continuous physiologic trait is likely to generate some misclassification.

Central adiposity better reflects visceral and ectopic fat burden and therefore provides more specific risk information (18-20).

WC and WHtR capture this biology more effectively than BMI, yet they also rely on fixed cut points (9). Historically, US WC thresholds were anchored to a BMI of 30 in White adults rather than outcome data. Newer analyses show that BMI-stratified WC thresholds improve risk identification (9). These differences support sex- and ancestry-specific standards, while highlighting that race functions as a proxy for environmental and biological heterogeneity (21, 22). Data gaps remain substantial in many populations, particularly children, making it difficult to apply population-level standards consistently (23, 24). Nevertheless, evidence from the All of Us Research Program shows how these thresholds produce substantial reclassification, creating a large "anthropometric-only" obesity group driven entirely by the chosen cut point (12).

Indeed, "anthropometric-only" obesity reclassification in older adults reintroduces some of the same limitations seen with BMI: it equates body size with excess adiposity despite age-related height loss, fat redistribution, and most importantly declines in lean mass. These anthropometric criteria systematically misclassify heavier, higher–lean-mass individuals as "obese" and miss adults with "normal" measurements with low lean mass who face the highest mortality risk (25), reproducing the same misclassification error seen with BMI alone.

The European Association for the Study of Obesity (EASO) reclassified adults with BMI 25 to 30 kg/m^2, WHtR above 0.5, and a comorbidity as having obesity (10), paralleling aspects of the Commission's model. In NHANES, this shifted 18.8% of adults from overweight to obesity, yet their mortality risk was indistinguishable from normal-weight adults with comorbid illness (26). This pattern, also observed in the All of Us study, suggests that the excess risk reflects underlying disease burden (organ dysfunction) rather than additional adiposity beyond what BMI and existing comorbidities already capture (12). Because current guidelines already consider WC and comorbidities in guiding therapeutic management of obesity, the incremental value of anthropometric measures is unclear. Likewise, in 2 large UK cohorts, substituting BMI with waist-based measures did not substantially improve the prediction of multiple long-term conditions or mortality when BMI was ≥30 kg/m^2 (27).

These findings are consistent with prior evidence showing cardiometabolic risk increasing continuously with adiposity and lacking a natural inflection point. Adding BMI or WC to pooled CVD risk equations marginally improves discrimination, and site-to-site measurement variation of several centimeters further undermines classification (28-30). A more coherent approach would use direct or composite adiposity measures when feasible, apply population-specific thresholds only when empirically justified, and interpret anthropometric values primarily as continuous indicators, not fixed diagnostic categories.

Practical limits of causal attribution in the diagnosis of clinical obesity

According to the *Lancet Commission*, "the definition of clinical obesity implies the combination of an obesity phenotype with objective and specific evidence of ongoing illness due to obesity. Importantly, all diagnostic criteria of clinical obesity assume exclusion of obvious other causes of organ dysfunction or signs and symptoms." This framing elevates the clinical threshold from excess adiposity plus risk to demonstrable illness "due" to adiposity, thereby embedding causal attribution into the diagnostic construct (4). Although designed to balance sensitivity for illness detection with specificity for adiposity-driven dysfunction, this standard may have limited relevance for guiding treatment decisions in routine practice.

Evidence shows that individuals with obesity benefit from timely weight-management interventions regardless of demonstrated etiologic pathways. Regulatory labeling permits pharmacotherapy at defined BMI thresholds with comorbidities, and clinical guidelines support surgical and nonsurgical treatment without requiring proof that obesity caused the associated condition. Weight reduction lowers blood pressure, decreases atrial fibrillation burden, and improves functional capacity in obesity-heart failure with preserved ejection fraction (HFpEF)—benefits that also apply in older adults with anthropometric obesity and prevalent hypertension. Requiring causal adjudication in such settings delays treatment, introduces inequities through differential access to diagnostics, and does not meaningfully change eligibility.

Insisting on exclusive causation in multifactorial diseases further misaligns clinical priorities. Conditions such as atrial fibrillation, HFpEF, polycystic ovary syndrome (PCOS), and hypertension arise from interacting anatomic, neurohumoral, inflammatory, and genetic influences. Adiposity is often a meaningful contributor but rarely the sole cause, and demanding exclusive attribution provides little clinical value. Trials consistently show that weight reduction improves outcomes regardless of etiologic purity, likely through shared mechanisms—hemodynamic unloading, reduced ectopic fat, and lowered inflammation. These mechanisms justify treatment without a singular causal narrative.

The demand for causal specificity becomes even less practical when examined through the lens of the Commission's own recommended assessments (4). For reduced lung or diaphragmatic compliance, clinicians are advised to perform spirometry or arterial blood gas testing. For suspected lymphedema, lymphoscintigraphy may be required (4). These investigations are resource-intensive, often unavailable in primary care, and susceptible to overuse when applied broadly. Expecting primary care physicians to resolve causal chains across cardiopulmonary, lymphatic, metabolic, and musculoskeletal domains risks delaying treatment, inflating downstream costs, and creating barriers for populations already facing constrained access to specialty evaluation (31, 32).

Taken together, these considerations suggest: clinicians should perform targeted exclusions that change management, initiate obesity treatment when guideline criteria are met, and allow clinical response to refine causal judgments retrospectively. Policy frameworks should tie coverage to expected benefit and safety, not proofs of exclusive causation. A pragmatic approach that prioritizes expected benefit over strict causal adjudication will likely speed care, reduce unnecessary diagnostic barriers, and promote equitable access to effective obesity treatment.

Conceptual challenges in defining preclinical obesity as a diagnostic category

Consider a 42-year-old woman with marked central adiposity, normal cardiometabolic tests, preserved exercise tolerance,

and no functional limitations—yet newly labeled with "pre-clinical obesity." This category is described as not a pre-disease but a possible early stage with "unknown" progression risk, defined by excess adiposity plus the absence of dysfunction across multiple organ systems (4, 33). This makes the label (1) diagnostically indeterminate—it becomes whatever remains after failing to detect dysfunction today—and (2) screening-dependent, because the more extensively one looks (eg, OSA testing, hepatic fat, diastolic dysfunction, early osteoarthritis, spirometry), the more individuals transition from "pre-clinical" to "clinical." A diagnostic category whose membership shifts with the intensity of the work-up raises questions about clinically coherent diagnostic category; accordingly, the Commission's definition raises conceptual tensions.

Medicine recognizes disease before symptoms when a state shows dysfunction plus foreseeable harm that early intervention can mitigate. Philosophical accounts similarly link dysfunction and harm (eg, Boorse; Wakefield's "harmful dysfunction") (34). Stage 1 hypertension (130-139/80-89 mm Hg), for example, carries a 38% higher CV event risk vs normotension (HR 1.38) (35). Likewise, in All of Us, preclinical obesity—excess adiposity without overt dysfunction—confers a 40% higher hazard for CV events (HR 1.40) vs individuals without obesity and without dysfunction (12). By the Commission's own logic, this satisfies an "ability to cause illness" threshold (4). Likewise, The Seventh Report of the Joint National Committee (JNC 7) introduced the term "pre-hypertension," which was later replaced in the 2017 ACC/AHA guidelines by "stage 1 hypertension." This shift reflected growing evidence of graded CV risk at lower blood pressure levels and highlighted how terminology evolves as evidence accumulates and as professional consensus changes precisely to avoid implying clinical inaction (36).

By contrast, the Commission's preclinical category is defined in the absence of demonstrable dysfunction, reflects uncertainty about progression risk, and raises questions about its alignment with harm-based thresholds applied elsewhere. Individuals with preclinical obesity already face elevated cardiometabolic and cancer risks, yet analogies to colon polyps, monoclonal gammopathy, or prediabetes may not be directly comparable (33); those conditions have validated lesions or biomarkers, quantifiable progression risks, and established surveillance strategies. At the same time, increased adiposity alone does not guarantee progression to clinically significant disease. Risk is probabilistic rather than deterministic, reinforcing the importance of individualized risk assessment rather than categorical assumptions. The Commission argues that diagnosis is separable from risk stratification and will not delay care (33). In practice, however, coverage, care pathways, and quality metrics track diagnoses. A label certifying "no substantial dysfunction" will be interpreted as low acuity, reducing eligibility for disease-directed therapies even when risk is high—as recent coverage patterns for obesity pharmacotherapy illustrate (32, 37). Because treatment benefits, harms, and costs vary, appropriate use depends on quantified risk, not a binary threshold based on dysfunction or limitations. Thus, policy should link access and treatment intensity to expected benefit. As currently defined, it may have limited explanatory or therapeutic value and could complicate communication about risk and management priorities. A more coherent alternative may be explicit risk-and-harm staging grounded in pathophysiology and calibrated event risk.

Reconsidering type 2 diabetes in the definition of clinical obesity

A clinician who sees a patient with obesity and new-onset T2DM instinctively recognizes an adiposity-driven disease process that merits intensive intervention. The Commission, however, excludes T2DM from its list of obesity-induced organ dysfunctions, despite defining clinical obesity as an obesity phenotype accompanied by "objective and specific evidence of ongoing illness due to obesity," after excluding obvious alternative causes (4). This approach is meant to privilege causal specificity over risk association, but when applied to T2DM it creates interpretive challenges in clinical application.

First, the Commission's own taxonomy already uses diseases as evidence of adiposity-induced dysfunction (4). It distinguishes incidental comorbidities from obesity-related diseases that share plausible causal pathways—explicitly citing T2DM as an archetype. Yet it simultaneously lists OSA, atrial fibrillation, HFpEF, pulmonary hypertension, and osteoarthritis as diagnostic manifestations of clinical obesity. These conditions, like T2DM, are heterogeneous, can occur without obesity, and have multifactorial causation. If "one should not diagnose a disease with another disease" were consistently applied, these endpoints would also be excluded. The differential treatment of T2DM raises questions about how the framework's criteria are applied across conditions.

Second, T2DM meets the Commission's selection criteria more strongly than several included endpoints. Candidate manifestations should be frequent in obesity, mechanistically linked to adiposity, and of substantial health impact. Roughly 90% of adults with T2DM are overweight or obese (38), exceeding many accepted criteria. Mendelian randomization using abdominal adiposity instruments supports a causal effect of adiposity on T2DM risk (18-20, 39). Its pathophysiology is tightly coupled to visceral and ectopic fat, insulin resistance, and beta cell dysfunction, and its vascular complications impose greater morbidity, mortality, and cost than several listed organ dysfunctions. Excluding it raises questions about alignment with the framework's stated rationale. Third, the decision to prefer a metabolic cluster over the diagnosis of diabetes creates clinically counterintuitive diagnostic boundaries. The Commission's metabolic criterion requires hyperglycemia together with elevated triglycerides and low HDL cholesterol (4). Under this rule, a patient with an HbA1c of 6.1% and dyslipidemia meets the definition of clinical obesity, whereas a patient with an HbA1c of 6.8% and therefore diagnosed T2DM, but normal lipids may not. When dysglycemia has emerged in parallel with weight gain and improves with weight loss, it creates a paradoxical situation where a milder glycemic abnormality carries more diagnostic weight than overt diabetes solely because of lipid patterns. Such a result may be challenging to reconcile within a framework that seeks to identify organ dysfunction that is truly driven by adiposity.

Fourth, specificity is applied more stringently to T2DM than to other included conditions, despite stronger evidence of reversibility. The Commission notes that diabetes includes non–adiposity-driven forms such as autoimmune, monogenic, and pancreatogenic subtypes. Yet obstructive sleep apnea, atrial fibrillation, and heart failure with preserved ejection fraction are

also etiologically diverse, and for these, the framework accepts reversibility with weight loss, mechanistic plausibility, and epidemiologic strength as sufficient. Intensive weight loss programs in primary care achieve diabetes remission in nearly half of participants at 1 year, with durability at 2 years, and metabolic surgery provides superior long-term glycemic control and medication independence (40, 41). Few other listed manifestations demonstrate comparably robust within-patient reversibility.

Finally, excluding T2DM may create policy and implementation challenges, particularly in coverage decisions. For decades, diabetes has been a principal qualifier for obesity pharmacotherapy and metabolic surgery in guidelines and coverage decisions, and professional societies have warned that a definitional split between classification and treatment may confuse payers and delay access. A pragmatic alternative is to recognize an adiposity-driven subtype of T2DM, defined by confirmed excess adiposity, exclusion of alternative diabetes types, evidence of adiposity-linked mechanisms, and, where feasible, observed reversibility with weight loss or metabolic intervention. Such a criterion would preserve specificity, align classification with clinical evidence, and better reflect how obesity and diabetes intersect in practice.

Excess adiposity, mental health, and cancer risk

The Commission emphasizes stigma reduction and person-first language but gives limited prominence to mental health and health-related quality of life within its diagnostic criteria, despite strong evidence linking excess adiposity to depression, anxiety, disordered eating, and reduced quality of life (42-45). Staging systems such as the Edmonton Obesity Staging System explicitly include these domains because of their prognostic and therapeutic significance (46).

Importantly, cancer is not an organ-dysfunction criterion in the Commission framework; it is treated as a downstream consequence, so observed obesity-cancer associations are not circular by definition. In UK Biobank, cancer risk was elevated in preclinical obesity and increased further in clinical obesity, especially for metabolically driven cancers (hepatocellular, endometrial, colorectal, pancreatic), with inverse associations for nonfatal prostate cancer. Together, preclinical and clinical obesity accounted for a substantial share of obesity-related cancers, suggesting that carcinogenesis may begin before overt dysfunction appears (14). Growing evidence indicates that excess adiposity may contribute to both mental health disorders and selected cancers via shared metabolic, inflammatory, and neuroendocrine pathways. This underscores the need for research that clarifies these mechanisms and determines how early detection and intervention can mitigate risk.

Anthropometric accuracy and why it matters

Because WC and WHtR are central to the Commission framework, their measurement accuracy directly determines diagnosis and treatment access. Yet methods are neither harmonized nor consistently validated across populations (47, 48). There is no consensus on the optimal protocol for WC measurement (9): WHO recommends measurement at the midpoint between the lower rib and iliac crest; US National Institutes of Health specifies the iliac crest superior border. Protocol choice has little impact on population-level associations but affects absolute values (49). When guidelines rely on fixed cut points, protocol differences can reclassify individuals without a change in underlying biology.

Site choice produces modest differences in men but striking differences in women and across ethnic groups (50, 51). Measurement mode adds further error (52). Self-measured WC typically underestimates by 1 to 3 cm (9). Such nontrivial errors are most consequential near treatment or screening thresholds (47, 48, 53). WHtR inherits all WC errors plus height misreporting (54-56). Because the Commission scheme links anthropometric indices to organ-specific evaluation, measurement error becomes a source of misclassification and inequity.

Many primary-care settings lack training, time, and infrastructure for accurate anthropometry (57, 58). A staged implementation—adopting improved measures where feasible while retaining simple BMI-plus-comorbidity criteria in resource-limited settings—may be more realistic than mandating comprehensive anthropometry universally.

Operational and policy implications of the Commission's framework

Coverage and affordability in US payer systems: In cash-pay markets, demand for weight loss, convenience, and price will dominate, and detailed phenotyping is unlikely to shape practice. In reimbursed systems, however, the Commission criteria may restrict eligibility for obesity treatments to individuals with demonstrable functional impairment, excluding those with preclinical obesity and limiting early pharmacologic intervention. Because detailed assessment is less available in low-income, rural, and minority-serving settings, stringent criteria may exacerbate existing disparities.

In Medicare, broad BMI-based eligibility (BMI $\geq$30 kg/m^2 or 27-29.9 kg/m^2 with an obesity-related condition) identifies the largest pool of nondiabetic beneficiaries (59, 60). Limiting coverage to individuals who meet the Commission's definition of clinical obesity reduces this population by roughly one-fifth (12). Yet when the Congressional Budget Office's (CBO's) assumptions about treatment initiation, discontinuation, and long-term adherence are applied, these large eligibility differences yield only modest differences in realized medication use (59). With a CBO estimated 13% of newly eligible beneficiaries still on therapy by year 10 (59), Medicare's projected spending varies by about 20% between the broadest and most restrictive frameworks.

A similar pattern appears in commercially insured populations. Under broad BMI-based criteria, roughly 45% of diabetes-excluded commercially insured adults (approximately 63-70 million people) would qualify for treatment, although meaningful access is largely confined to employer-sponsored insurance. Using the Commission's clinical obesity definition reduces eligibility by about half, with BMI-plus-comorbidity

criteria falling in between (4). These estimates scale All of Us prevalence to the insured population, introducing uncertainty, but the core conclusion remains.

It is reasonable to interpret part of the Commission's intent as prioritizing treatment for individuals with the greatest demonstrable health burden, particularly in the context of constrained payer resources. Targeting therapy toward those most likely to benefit is a defensible policy goal. Our concern is less about prioritization itself and more about whether the proposed diagnostic structure is the optimal mechanism for achieving that goal, particularly given the central role of price and affordability in determining real-world access.

When Institute for Clinical and Economic Review's (ICER's) annual affordability threshold of $880 million is applied, eligibility differences have minimal practical impact (61-63). At current net prices, <1% of eligible individuals could be treated before exceeding this threshold; the affordable share increases only slightly under the narrower clinical-obesity criteria (0.14% to 0.28%). Thus, regardless of definition, more than 99.7% of eligible adults would remain untreated.

Taken together, the Medicare and commercial analyses both demonstrate that eligibility definitions influence the size of the potential treatment population but have limited impact on actual use or spending once real-world uptake and affordability constraints are incorporated. The Administration's recently proposed most-favored-nation (MFN) pricing initiative (64) would cut net prices by roughly half for Medicare and by two-thirds for many commercially purchased GLP-1-based medications, substantially reducing projected federal spending. However, even if these historic price reductions are realized, affordability constraints remain severe: the MFN cuts lower total costs but do not expand affordable treatment capacity meaningfully under ICER's threshold, leaving more than 99% of eligible commercial-market adults still unable to be treated at scale. Across both settings, the core policy insight is the same: the binding constraint is price, not eligibility.

Finally, the Commission's framework presumes routine confirmation of excess adiposity and systematic assessment of organ or functional limitations—requirements that exceed the diagnostic capacity of many primary-care settings. As a result, BMI combined with comorbidity status will remain the default classification tool because of its simplicity, scalability, and integration into electronic health records. Moreover, real-world data and electronic documentation rarely include the standardized adiposity or functional metrics necessary for strict implementation, reinforcing disparities between data-rich and data-poor systems.

Comparing the Commission with staging systems used in practice

Several staging systems already characterize obesity severity, guide treatment decisions, and communicate risk. These frameworks predate or parallel the Commission work and highlight both convergences and divergences.

The Edmonton Obesity Staging System (EOSS) similarly shifts focus from adiposity quantity to illness severity (46). Using stages 0 through 4, it incorporates clinical risk factors, established comorbidities, end-organ damage, functional limitations, and mental health. EOSS shows strong predictive validity for mortality, cardiovascular events, and health care utilization, outperforming BMI alone. It is explicitly prognostic and treatment-guiding rather than definitional. This outcome-based orientation is important: it treats obesity staging as a prognostic tool rather than a definitional one.

The AACE Adiposity-Based Chronic Disease (ABCD) model labels obesity as chronic disease and divides it into stages based on the presence and severity of complications (65). The Commission's preclinical obesity corresponds roughly to an early ABCD stage without complications, and clinical obesity to more advanced stages. AACE emphasizes evidence-based interventions at all stages, including prevention and early obesity.

The European Association for the Study of Obesity (EASO) diagnostic and treatment framework defines obesity as BMI $\geq$30 kg/m^2 or BMI 25 to 29.9 kg/m^2 with WHtR $\geq$0.5 plus medical, functional, or psychological impairment (10). Its management algorithm separates body weight management from complications management and recognizes both "fat mass disease" (eg, sleep apnea, osteoarthritis) and "sick fat disease" (eg, T2DM, CVD, heart failure, metabolic dysfunction, steatotic liver disease).

Compared with the Commission's binary preclinical/clinical model, these staging systems offer several advantages. They provide graded severity across the full spectrum of risk rather than a sharp health-illness divide. They avoid causal adjudication, thereby eliminating the need to determine whether each comorbidity is mechanistically attributable to adiposity. They are more directly actionable because staging maps onto treatment pathways and intensity. They also integrate psychosocial and functional domains more fully, reflecting obesity's multidimensional nature.

From a policy perspective, frameworks that anchor diagnosis in demonstrable medical, functional, or psychological impairment are easier to implement across diverse settings and less dependent on resource-intensive diagnostics. Their impairment-based criteria align more closely with evolving coverage decisions for pharmacologic and surgical treatments, which increasingly depend on documented functional impact rather than BMI thresholds alone.

Overall, staging systems such as EOSS and EASO remain, in many respects, better suited for routine clinical use and more empirically grounded than an etiologically stringent, 2-state structure that demands extensive causal adjudication.

Achieving harmonization across frameworks will require structured collaboration among professional societies, patient representatives, payers, and regulators. Practical steps could include: (1) development of shared minimum diagnostic data elements; (2) agreement on standardized anthropometric measurement protocols; (3) creation of cross-walk tables aligning stages across systems (eg, EOSS, ABCD, Commission categories); and (4) prospective validation studies applying multiple staging systems within the same cohorts to compare prognostic performance. Pragmatic alignment may be more achievable than full definitional consensus and would reduce variability in clinical practice.

Conclusion

The Lancet Commission framework represents a constructive shift toward diagnosing obesity by assessing whether excess adiposity is contributing to clinically meaningful illness.

However, its dependence on detailed causal adjudication, the conceptual ambiguity of the preclinical obesity category, and the extensive diagnostic requirements limit its feasibility and carry a risk of exacerbating existing inequities. A more sustainable approach would prioritize early identification of central adiposity through simple, scalable measures; emphasize functional impairment and quantifiable risk rather than rigid diagnostic thresholds; and align treatment access with expected clinical benefit and safety rather than with definitive causal attribution. Future diagnostic frameworks should integrate biological nuance with operational practicality to ensure that refinements in classification broaden, rather than restrict, equitable access to effective, evidence-based obesity care.

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Disclosures

See Appendix.

Data availability

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Appendix

Endocrine Society's Guideline Communication Work Group
Roles, disclosures, and management plans

SUMMARY

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	Dimpi Desai	No
	Roma Gianchandani	No
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NOTES:

- Total number of work group members & 7
- Percent of total GDP members with relevant (or potentially-relevant) COI & 14%

INDIVIDUAL DISCLOSURES, CONFLICTS, AND MANAGEMENT STRATEGIES

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- American Association of Cliinical Endocrinology: Vice Chair of Obesity and Nutrition DSN, speaker honorarium
- The Obesity Society: Chair of CME Oversight Committee
- Journal of Clinical Endocrinology and Metabolism (JCEM): Editorial Board
- Endocrine Society: Pharmacological Management of Obesity Guideline Update Panel Member

Open Payments Database: https://openpaymentsdata.cms.gov/physician/8795412

ASSESSMENT AND MANAGEMENT

- No industry relationships relevant to this manuscript in past 24 months.
- No COI management required.

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Disclosures (2024-2026):

- Endocrine Society: Clinical Guidelines Committee Chair

Open Payments Database: https://openpaymentsdata.cms.gov/physician/371886

ASSESSMENT AND MANAGEMENT

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- National Institutes of Health: Research funding
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- The Obesity Society: Finance Committee Chair
- The Hormone Foundation: Trustee
- University of Michigan: Clinical Excellence Society Treasurer/Secretary

Open Payments Database: https://openpaymentsdata.cms.gov/physician/164497

ASSESSMENT AND MANAGEMENT

- Dr. Rothberg has industry relationships relevant to this CPG.
- Dr. Rothberg was invited to participate on this work group because she is a renowned expert in the area of obesity.
- Since Endocrine Society Guideline Communications serve as scholarly commentaries and strategic updates, and do not make clinical recommendations or rely on formal voting, management of these relationships emphasized full transparency and disclosure rather than divestment or recusal.

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- Endocrine Society: Speaker honorarium, Pharmacological Management of Obesity Guideline Update Panel Chair
- Cornell Qatar Medicine: Speaker honorarium
- Metabolic Care Today: Editor

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ASSESSMENT AND MANAGEMENT

- No industry relationships relevant to this manuscript in past 24 months.
- No COI management required.

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