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REVIEW ARTICLE

Body composition and cardiovascular disease: Clinical implications beyond body mass index

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ABSTRACT

Cardiovascular diseases (CVDs) remain the leading cause of morbidity and mortality worldwide, and excess adiposity is a major contributor to cardiovascular (CV) risk. However, obesity is a heterogeneous condition, and body mass index (BMI) alone fails to capture important differences in fat distribution and lean mass that substantially influence CV outcomes. Growing clinical and epidemiological evidence indicates that body composition, rather than body weight per se, provides a more accurate and biologically meaningful framework for CV risk assessment. This narrative review summarizes clinical evidence linking key components of body composition, including total and regional adiposity, skeletal muscle mass, and ectopic fat depots, to CV risk and prognosis. Central and visceral adiposity are consistently shown to be more strongly associated with cardiometabolic dysfunction, atherosclerosis, and CV events than generalized obesity. In parallel, reduced lean mass and sarcopenia emerge as independent predictors of adverse CV outcomes, particularly in older adults and patients with established CVD. Ectopic fat depots, such as epicardial and hepatic fat, further contribute to CV pathology through local and systemic mechanisms. Collectively, these findings highlight the limitations of BMI-centered approaches and support the integration of body composition measures into CV risk stratification. Emphasizing body recomposition, with reduction of harmful fat depots and preservation of skeletal muscle, may enable more precise, individualized strategies for CV prevention and management.

Keywords: body composition, cardiovascular disease, visceral adiposity, sarcopenia, ectopic fat

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INTRODUCTION

Cardiovascular diseases (CVD) remain the leading contributor to global morbidity and mortality, with a sustained rise in absolute burden driven by population growth, aging, and persistence of modifiable risk factors [1]. In parallel, overweight and obesity have become highly prevalent worldwide and contribute to cardiovascular (CV) risk through both indirect pathways including promotion of hypertension, dyslipidemia, and type 2 diabetes, and direct adverse effects on cardiac structure, vascular function, and inflammation [2]. However, "obesity" is a biologically heterogeneous state: individuals with similar body mass index (BMI) may have markedly different distributions of adipose tissue, different amounts of skeletal muscle, and different cardiometabolic risk profiles.

A major limitation of BMI is that it does not distinguish fat mass from lean mass and does not capture regional adiposity. Clinical and epidemiologic evidence increasingly indicates that where fat is stored is at least as important as how much fat is present. Central (abdominal) obesity, often reflecting excess visceral adipose tissue and ectopic fat, shows stronger associations with insulin resistance, atherogenic dyslipidemia, systemic inflammation, and incident cardiometabolic disease than generalized adiposity [3]. Mechanistically, visceral adipose tissue is metabolically active and contributes to adverse endocrine and inflammatory signaling, including altered

adipokines and increased pro-inflammatory mediators that may accelerate atherosclerosis and vascular dysfunction [4].

These limitations have motivated broader use of alternative anthropometric indices (e.g., waist circumference and waist-to-height ratio) and imaging or device-based body composition assessments. Systematic reviews suggest that central obesity metrics often predict cardiometabolic outcomes more consistently than BMI, supporting their use in risk stratification [5]. At the same time, the clinical literature includes apparent paradoxes, most notably the "obesity paradox," where overweight/obesity by BMI may associate with better outcomes in some established CVD populations, raising the possibility that body composition (fat distribution, muscle mass) and confounding (fitness, unintentional weight loss, disease severity) materially influence observed associations [6].

Accordingly, this review examines clinical evidence linking body composition, including total and regional adiposity, skeletal muscle mass, and ectopic fat depots, to CV risk and outcomes. Differences in fat distribution and lean mass help explain the heterogeneity of CV risk observed among individuals with similar BMI. By integrating evidence from population-based and clinical studies, the review aims to clarify why traditional weight-based metrics may be insufficient for CV risk assessment and to underscore the potential value of body composition-oriented approaches in prevention and clinical practice.

A narrative literature review was conducted using PubMed/MEDLINE and Scopus databases to identify relevant studies on body composition and CVD. The search covered publications from January 2000 to March 2025. Key search terms included "body composition," "visceral adiposity," "central obesity," "sarcopenia," "ectopic fat," "epicardial fat," and "cardiovascular disease," used alone and in combination. Priority was given to large cohort studies, systematic reviews, meta-analyses, and clinically relevant observational studies published in English. Additional references were identified through manual screening of reference lists from selected articles.

CONCEPTS AND MEASUREMENT OF BODY COMPOSITION

Body composition describes the relative proportions of fat mass, lean mass, and bone mass in the human body and represents a more biologically meaningful construct than body weight alone. In CV research and clinical practice, this concept emphasizes not only the quantity of adipose tissue but also its distribution and the amount of skeletal muscle. Adipose tissue is a heterogeneous organ composed of subcutaneous fat, visceral fat, and ectopic fat depots, each with distinct metabolic and CV implications. Visceral adipose tissue, located within the abdominal cavity, is strongly associated with insulin resistance, atherogenic dyslipidemia, low-grade systemic inflammation, and increased CV risk, whereas subcutaneous fat appears to have a weaker association with adverse cardiometabolic outcomes [7, 8].

Lean mass, primarily represented by skeletal muscle, is increasingly recognized as a key determinant of cardiometabolic health. Skeletal muscle plays a central role in glucose uptake, insulin sensitivity, basal metabolic rate, and physical performance. Reduced muscle mass may therefore predispose individuals to metabolic dysfunction and adverse CV outcomes, particularly in older populations. These differences help explain why individuals with similar BMI values may exhibit markedly different CV risk profiles depending on their underlying body composition [9].

Several methods are available to assess body composition, each with specific advantages and limitations. Anthropometric indices such as waist circumference and waist-to-hip ratio are widely used as surrogate markers of central adiposity but cannot directly quantify visceral fat or lean mass. Bioelectrical impedance analysis (BIA) provides a practical and non-invasive method for estimating fat mass and lean mass in both research and clinical settings, although its accuracy may be influenced by hydration status, age, and comorbid conditions [10]. Dual-energy X-ray absorptiometry allows relatively precise assessment of total and regional fat and lean mass with low radiation exposure and is frequently used in epidemiological studies. Computed tomography (CT) and magnetic resonance imaging (MRI) permit direct quantification of visceral and ectopic fat depots and are considered reference standards, but their routine clinical use is limited by cost, accessibility, and radiation exposure in the case of CT [11].

From a clinical perspective, the choice of body composition assessment method reflects a balance between feasibility and precision. While advanced imaging techniques are valuable in research and selected clinical scenarios, simpler methods such as BIA or DXA-derived indices may provide incremental information beyond traditional anthropometric measures for CV risk assessment [8]. The major components of body composition, their commonly used assessment methods, and their CV relevance are summarized in **Table 1**.

ADIPOSITY AND CV RISK

Excess adiposity is a well-established determinant of CV risk, yet its impact varies substantially according to the amount and distribution of fat tissue. Epidemiological studies have consistently shown that increased adiposity is associated with higher incidence of hypertension, dyslipidemia, type 2 diabetes, and atherosclerotic CVD. However, measures of total body fat, most commonly approximated by BMI, incompletely capture the complexity of adiposity-related risk. Individuals with similar BMI values may differ markedly in fat distribution, metabolic profile,

Table 1. Body composition components and their CV implications

Body composition component	Definition	Assessment methods	CV relevance
Total adiposity	Overall body fat mass	BMI, BIA, DXA	Associated with cardiometabolic risk but limited prognostic precision
Visceral adipose tissue	Fat within the abdominal cavity	CT, MRI, waist circumference	Strongly linked to insulin resistance, inflammation, atherosclerosis
Subcutaneous adipose tissue	Fat beneath the skin	DXA, CT, MRI	Weaker or neutral association with CV outcomes
Skeletal muscle mass	Lean mass contributing to movement and metabolism	BIA, DXA, CT	Protective role; low levels associated with higher CV mortality
Ectopic fat depots	Fat accumulation in non-adipose tissues	CT, MRI, echocardiography	Contribute to local and systemic CV dysfunction

Note. DXA: Dual-energy X-ray absorptiometry

Table 2. Associations between body composition phenotypes and CV outcomes

Body composition phenotype	Key characteristics	Associated cardiovascular outcomes
General obesity	Elevated BMI	Increased risk of hypertension, diabetes, and CVD; heterogeneous prognosis
Central obesity	Increased waist circumference or waist-to-hip ratio	Higher risk of myocardial infarction, stroke, and heart failure
Visceral obesity	Excess intra-abdominal fat	Coronary atherosclerosis, cardiometabolic dysfunction, CV mortality
Sarcopenia	Low muscle mass and function	Increased CV events, frailty, and all-cause mortality
Sarcopenic obesity	Coexistence of excess fat and low muscle	Highest cardiometabolic and CV risk

and CV outcomes, underscoring the importance of distinguishing between generalized and central adiposity [12].

Central obesity, reflecting increased abdominal and visceral fat accumulation, has emerged as a stronger predictor of CVD than overall adiposity. Large prospective cohort studies demonstrate that waist circumference and waist-to-hip ratio are more closely associated with incident myocardial infarction, stroke, and CV mortality than BMI alone [13]. Visceral adipose tissue is metabolically active and characterized by increased lipolytic activity, leading to elevated free fatty acid flux to the liver and promotion of insulin resistance, atherogenic dyslipidemia, and systemic inflammation [14]. These mechanisms provide a biological basis for the stronger association between central obesity and CV risk.

Beyond traditional anthropometric indices, imaging-based studies have further clarified the role of visceral fat in CVD. Quantification of visceral adipose tissue using CT or MRI has shown independent associations with coronary artery calcification, subclinical atherosclerosis, and incident CV events, even after adjustment for BMI and other risk factors [15]. In contrast, subcutaneous adipose tissue appears to exert a weaker association with CV outcomes and, in some contexts, may even display neutral or protective metabolic characteristics compared with visceral fat depots [16].

The relationship between adiposity and CV outcomes is further complicated by observations commonly referred to as the "obesity paradox," in which overweight or mildly obese individuals with established CVD sometimes exhibit better short- and medium-term outcomes than their normal-weight counterparts. Contemporary analyses suggest that this paradox may be partly explained by limitations of BMI, residual confounding, reverse causation, and the failure to account for body composition and cardiorespiratory fitness [17]. When measures of central adiposity or fat distribution are considered, the apparent protective associations of excess body weight are substantially attenuated or abolished.

Overall, current evidence indicates that adiposity-related CV risk is driven predominantly by central and visceral fat accumulation rather than excess body weight per se. These findings support a shift toward adiposity distribution–focused assessment in CV risk stratification and reinforce the limitations of BMI as a standalone metric [12, 13]. Distinct body composition phenotypes and their associated CV outcomes are summarized in **Table 2**.

LEAN MASS, SARCOPENIA, AND CV OUTCOMES

Lean mass, largely composed of skeletal muscle, is a critical but often underappreciated determinant of CV health. Beyond its mechanical role in movement, skeletal muscle functions as a major metabolic organ involved in glucose uptake, insulin sensitivity, lipid oxidation, and energy homeostasis. Reduced muscle mass is associated with

impaired metabolic regulation and diminished physical capacity, both of which may contribute to adverse CV outcomes independent of adiposity [18].

Sarcopenia, defined as a progressive loss of skeletal muscle mass and function, is increasingly prevalent with aging and chronic disease. According to the updated European Working Group on Sarcopenia in Older People (EWGSOP2) consensus, low muscle strength is considered the primary indicator of probable sarcopenia, while the diagnosis is confirmed by reduced muscle quantity or quality, and severe sarcopenia is defined by the presence of impaired physical performance in addition to low strength and low muscle mass [19].

Consensus definitions emphasize not only low muscle mass but also reduced muscle strength and physical performance. Epidemiological studies demonstrate that sarcopenia is associated with increased prevalence of CV risk factors, including hypertension, insulin resistance, and systemic inflammation, as well as higher rates of CV events and mortality [20, 21]. These associations persist after adjustment for traditional risk factors and BMI, highlighting the independent prognostic significance of muscle loss.

Low lean mass has been linked to unfavorable CV outcomes across multiple clinical contexts. In population-based cohorts, reduced appendicular lean mass is associated with higher all-cause and CV mortality. In patients with established CVD, particularly heart failure and coronary artery disease (CAD), diminished muscle mass and strength correlate with poorer functional status, increased hospitalization rates, and worse survival [22]. Mechanistically, skeletal muscle wasting may exacerbate CV risk through reduced insulin-mediated glucose disposal, increased oxidative stress, and impaired exercise tolerance.

The coexistence of excess adiposity and low muscle mass, commonly referred to as sarcopenic obesity, represents a particularly high-risk phenotype. Individuals with sarcopenic obesity exhibit a combination of adverse metabolic features, including increased inflammation, insulin resistance, and physical inactivity. Clinical studies suggest that this phenotype confers greater CV risk than either obesity or sarcopenia alone, especially in older adults [23]. These findings underscore the importance of assessing both fat and lean compartments when evaluating CV risk.

Overall, accumulating evidence indicates that lean mass and muscle function are key determinants of CV health and prognosis. Consideration of skeletal muscle status alongside measures of adiposity may improve CV risk stratification and help identify individuals who could benefit from targeted lifestyle and rehabilitative interventions.

ECTOPIC FAT DEPOTS AND CVC

Ectopic fat refers to lipid accumulation in non-adipose tissues or in adipose depots surrounding vital organs, including the heart, liver, skeletal muscle, and blood vessels. Unlike subcutaneous fat, ectopic fat depots are closely linked to local and systemic metabolic dysfunction and exert direct effects on CV structure and function. Growing evidence indicates that ectopic fat contributes to CVD independently of overall and central adiposity, highlighting its clinical relevance in CV risk assessment [24].

Epicardial adipose tissue, located between the myocardium and the visceral pericardium, has attracted particular interest due to its anatomical proximity to the coronary arteries and myocardium. Epicardial fat is metabolically active and capable of secreting pro-inflammatory cytokines, adipokines, and free fatty acids that may promote coronary atherosclerosis and myocardial dysfunction through paracrine mechanisms. Clinical imaging studies have demonstrated strong associations between increased epicardial fat volume and CAD severity, coronary calcification, and adverse CV outcomes, independent of traditional risk factors [25, 26].

Hepatic fat accumulation, commonly manifesting as non-alcoholic fatty liver disease, is another important ectopic fat depot with CV implications. NAFLD is strongly associated with insulin resistance, systemic inflammation, and atherogenic dyslipidemia, and numerous cohort studies have shown that it predicts incident CVD and CV mortality, often exceeding the risk of liver-related complications [27]. Importantly, the CV risk associated with NAFLD persists after adjustment for BMI and other metabolic risk factors, suggesting a direct link between hepatic steatosis and CV pathology [28].

Perivascular and intramuscular fat depots represent emerging areas of interest. Perivascular adipose tissue surrounding large and small arteries may influence vascular tone, endothelial function, and arterial stiffness through local inflammatory signaling. Increased perivascular fat has been associated with impaired vasodilation and subclinical atherosclerosis, supporting a potential mechanistic role in vascular disease development [29]. Although these depots are not routinely assessed in clinical practice, advances in imaging techniques continue to improve their characterization and relevance.

Collectively, evidence on ectopic fat depots underscores the importance of fat location, rather than total fat mass alone, in CVD pathogenesis. Assessment of ectopic fat provides mechanistic insight into obesity-related CV risk and may help refine risk stratification beyond conventional anthropometric measures. The adverse CV effects of visceral adiposity, reduced skeletal muscle mass, and ectopic fat depots are mediated through interconnected pathophysiological mechanisms, including insulin resistance, chronic inflammation, endothelial dysfunction, and neurohormonal activation (**Figure 1**).

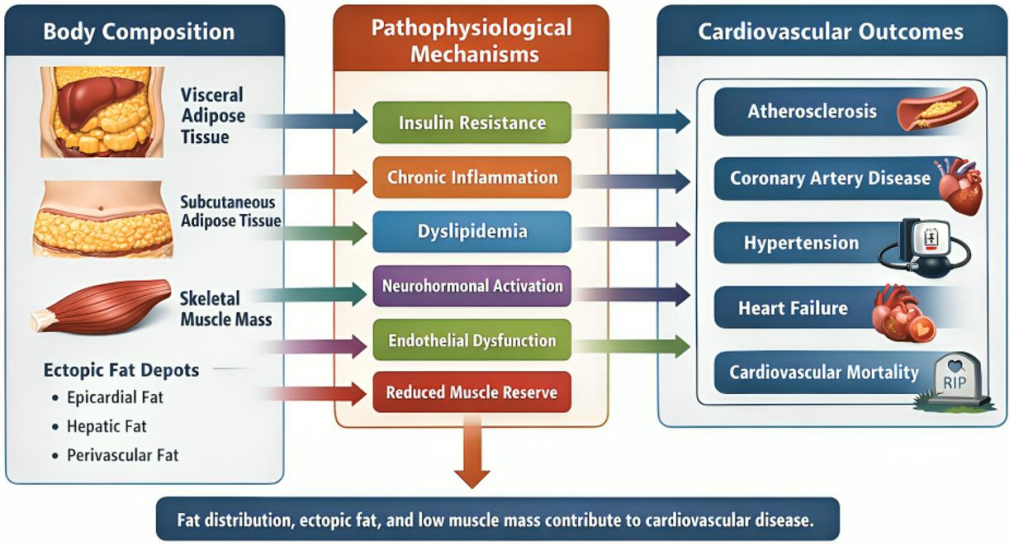

Figure 1. Pathophysiological mechanisms linking body composition to cardiovascular outcomes (Source: Authors' own elaboration)

BODY COMPOSITION AND SPECIFIC CV CONDITIONS

Hypertension

Adiposity is closely linked to blood pressure regulation, but accumulating evidence indicates that this relationship is driven predominantly by central and visceral fat rather than total body weight. Visceral adipose tissue contributes to hypertension through multiple mechanisms, including activation of the renin–angiotensin–aldosterone system, sympathetic nervous system stimulation, endothelial dysfunction, and chronic low-grade inflammation [30]. These processes promote sodium retention, increased vascular resistance, and arterial stiffness, all of which favor the development and progression of hypertension.

Importantly, BMI often fails to identify individuals with excess visceral fat and elevated blood pressure risk. For this reason, expert consensus statements advocate routine assessment of waist circumference in clinical practice as a simple and effective marker of visceral obesity and cardiometabolic risk [31]. Incorporating measures of central adiposity may therefore improve early identification of hypertension risk, particularly in individuals with normal or mildly elevated BMI.

CAD

In CAD, body fat distribution appears to be more relevant than total adiposity. Large-scale observational studies and meta-analyses consistently show that waist-to-hip ratio and other indices of central obesity are stronger predictors of myocardial infarction than BMI across diverse populations [32]. These findings are supported by genetic studies using Mendelian randomization approaches, which suggest a causal association between increased waist circumference and coronary heart disease risk [33].

Beyond fat distribution, lean mass also plays a role in coronary outcomes. Reduced skeletal muscle mass has been associated with worse prognosis following acute coronary syndromes, including higher mortality and adverse clinical outcomes after ST-segment elevation myocardial infarction [34]. These observations suggest that muscle mass may influence recovery, functional capacity, and resilience after coronary events, reinforcing the need for a comprehensive body composition perspective in patients with established coronary disease.

Heart Failure

The relationship between adiposity and heart failure is complex and strongly influenced by fat distribution. Prospective cohort studies indicate that abdominal obesity, measured by waist circumference, is independently associated with incident heart failure even after adjustment for BMI, highlighting the pathogenic role of central fat accumulation [35]. Mechanistically, visceral adiposity may promote myocardial remodeling, diastolic dysfunction, and systemic inflammation, thereby increasing susceptibility to heart failure.

Evidence from specific high-risk populations further supports the importance of central obesity. In individuals with type 1 diabetes, central adiposity has been associated with a significantly increased risk of heart failure hospitalization or death over long-term follow-up [36]. Notably, recent population-based analyses demonstrate that normal-weight individuals with central obesity have a higher risk of developing heart failure and atrial fibrillation compared with those without central fat accumulation, underscoring the limitations of BMI-centered risk assessment [37].

Taken together, these findings indicate that body composition, particularly central adiposity and lean mass, modifies the risk and clinical course of major CV conditions. Assessment strategies that move beyond BMI may therefore provide more accurate risk stratification and inform targeted preventive and therapeutic approaches.

Table 3. Clinical applications of body composition assessment in CV practice

Clinical setting	Limitation of BMI	Added value of body composition assessment
Primary prevention	Underestimates risk in normal-weight central obesity	Improved identification of high-risk individuals
Hypertension management	Poor discrimination of visceral fat	Better prediction of blood pressure risk
CAD	Limited prognostic value	Risk stratification using fat distribution and muscle mass
Heart failure	Obesity paradox with BMI	Identification of sarcopenia and adverse prognosis
Cardiac rehabilitation	Weight change poorly reflects benefit	Monitoring fat loss and muscle preservation

CLINICAL IMPLICATIONS AND RISK STRATIFICATION

Growing evidence indicates that incorporating body composition into CV risk assessment provides incremental information beyond traditional measures such as BMI. Reliance on BMI alone may lead to misclassification of risk, particularly in individuals with normal-weight central obesity or those with reduced skeletal muscle mass. Clinical studies demonstrate that indices of central adiposity, including waist circumference and waist-to-height ratio, improve identification of individuals at high cardiometabolic risk who might otherwise be considered low risk based on BMI [38]. As a result, several expert groups now recommend routine assessment of central obesity in clinical practice. Potential clinical applications of body composition assessment across different CV settings are outlined in **Table 3**.

From a practical clinical perspective, simple anthropometric thresholds can be readily applied to improve CV risk identification beyond BMI. Commonly used cut-off values for central obesity include a waist circumference ≥ 102 cm in men and ≥ 88 cm in women, with lower thresholds proposed for certain ethnic populations [31]. Waist-to-height ratio values ≥0.5 have also been widely suggested as a pragmatic indicator of increased cardiometabolic risk across diverse populations [38]. For assessment of sarcopenia, EWGSOP2 recommends low handgrip strength as the initial screening parameter, followed by confirmation with reduced skeletal muscle mass measured by DXA or BIA, using population-appropriate cut-offs [19]. In selected settings, echocardiographic measurement of epicardial fat thickness has been proposed as a feasible marker of ectopic fat burden, with values above approximately 5 mm associated with increased CV risk [25].

Beyond anthropometric indices, device- and imaging-based assessments of body composition may further refine CV risk stratification. Measures of visceral fat, lean mass, and ectopic fat depots have been shown to predict CV events and mortality independently of conventional risk factors. In particular, low skeletal muscle mass and poor muscle quality have emerged as markers of vulnerability and adverse prognosis in both primary and secondary prevention settings [39]. These findings support a shift toward integrated risk models that account for both adipose tissue distribution and muscle mass.

Body composition assessment also has practical implications for preventive strategies. Identification of individuals with excess visceral fat or sarcopenic obesity may guide personalized lifestyle interventions emphasizing not only weight reduction but also muscle preservation or augmentation. In patients with established CVD, body composition metrics may help identify those who are likely to benefit most from structured exercise programs and comprehensive cardiac rehabilitation [40]. Importantly, such approaches align with evidence showing that improvements in fitness and muscle strength are strongly associated with reduced CV and all-cause mortality, often independent of weight loss.

Overall, integrating body composition into CV risk stratification supports a more individualized and biologically relevant approach to prevention and management. While widespread implementation of advanced imaging may not be feasible, the use of simple central adiposity measures and selected body composition assessments could meaningfully enhance clinical decision-making and risk prediction [38, 41]. Accordingly, assessment of body composition phenotypes provides incremental information for CV risk stratification beyond BMI (**Figure 2**).

THERAPEUTIC AND LIFESTYLE INTERVENTIONS

Recognition of body composition as a determinant of CV risk has important implications for therapeutic and lifestyle interventions. Traditional strategies have focused primarily on weight reduction, yet growing evidence suggests that improvements in fat distribution and preservation or enhancement of skeletal muscle mass may be equally, if not more, relevant for CV risk reduction. Interventions that target body recomposition rather than weight loss alone may therefore provide greater cardiometabolic benefit [42].

Lifestyle modification remains the cornerstone of therapy. Aerobic exercise is effective in reducing visceral adiposity and improving cardiometabolic risk factors, even in the absence of substantial weight loss. Resistance training, in contrast, plays a critical role in maintaining or increasing skeletal muscle mass and strength, thereby counteracting

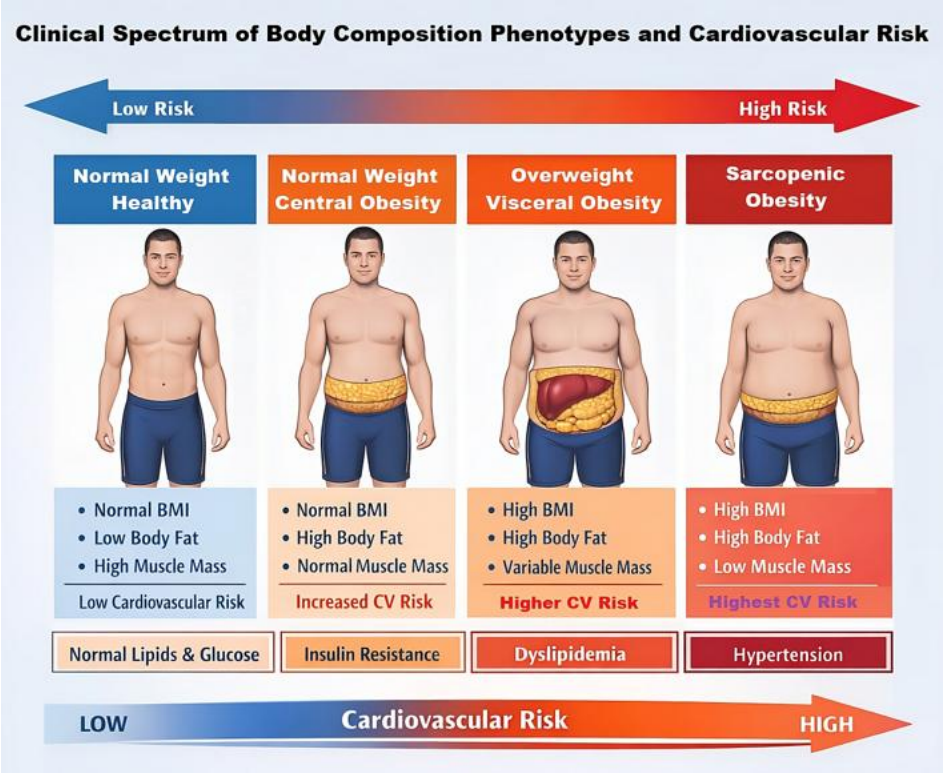

Figure 2. Clinical spectrum of body composition phenotypes and cardiovascular risk (Source: Authors' own elaboration)

sarcopenia and improving insulin sensitivity and functional capacity [43]. Combined aerobic and resistance exercise programs have been shown to produce favorable changes in both fat and lean compartments, supporting their use in CV prevention and rehabilitation settings.

Nutritional strategies also influence body composition and CV risk. Diets emphasizing adequate protein intake are important for muscle preservation, particularly in older adults and patients with chronic CVD. Caloric restriction without attention to protein intake or resistance exercise may lead to unintended loss of lean mass, potentially attenuating CV benefit. Evidence suggests that dietary patterns promoting negative energy balance while preserving muscle mass, such as Mediterranean-style diets combined with structured exercise, are associated with improved cardiometabolic outcomes [44].

Pharmacologic interventions may also affect body composition. Some glucose-lowering agents, including glucagon-like peptide-1 receptor agonists and sodium–glucose cotransporter 2 inhibitors, have been shown to reduce visceral adiposity and improve CV outcomes in high-risk populations. However, the effects of these agents on lean mass are variable and remain an area of ongoing investigation [45]. These considerations highlight the need to integrate pharmacologic therapy with lifestyle interventions to optimize body composition and CV risk reduction.

Overall, therapeutic approaches that address both adipose tissue distribution and skeletal muscle mass align with emerging evidence and support a more comprehensive strategy for CV prevention and management.

LIMITATIONS OF CURRENT EVIDENCE AND RESEARCH GAPS

Despite substantial progress, several limitations constrain the interpretation and clinical application of existing evidence linking body composition to CV risk. First, there is considerable heterogeneity in body composition assessment methods across studies. Anthropometric indices, BIA, DXA, CT, and MRI differ in precision, availability, and biological relevance, making comparisons across cohorts challenging and limiting the establishment of universally accepted thresholds for CV risk [46]. Variability in measurement protocols and analytic approaches further complicates synthesis of findings.

Second, many studies are observational in design, which restricts causal inference. Although associations between visceral adiposity, low lean mass, and CV outcomes are consistent, residual confounding, reverse causation, and selection bias cannot be fully excluded. For example, unintentional weight loss or muscle wasting due to subclinical disease may precede CV events, thereby exaggerating observed associations [47]. Randomized trials specifically targeting body composition components with CV endpoints remain limited.

Third, population heterogeneity is insufficiently addressed. Sex, age, ethnicity, and comorbid conditions influence body fat distribution and muscle mass, yet many

studies apply uniform cut-off values that may not be appropriate across diverse populations. Older adults and women, in particular, are underrepresented in imaging-based studies of visceral and ectopic fat, while functional measures of muscle quality are often omitted [48]. These gaps limit generalizability and clinical translation.

Finally, longitudinal data on changes in body composition over time are sparse. Most studies rely on single baseline measurements, despite evidence that dynamic changes in fat distribution and lean mass may be more informative for CV risk prediction than static values. Standardized definitions, repeated measurements, and integration with functional and biomarker data are needed to advance the field [49].

FUTURE DIRECTIONS

Future research on body composition and CV risk should move toward more precise, standardized, and longitudinal assessment strategies. A major priority is the development of harmonized definitions and cut-off values for visceral adiposity, ectopic fat depots, and low lean mass that are applicable across sexes, age groups, and ethnicities. Without standardized thresholds, translation of body composition metrics into routine CV risk prediction remains limited [50].

Longitudinal studies with repeated body composition measurements are particularly needed. Dynamic changes in visceral fat, skeletal muscle mass, and ectopic fat over time may provide stronger prognostic information than single baseline assessments. Such approaches could clarify whether improvements in body composition, independent of weight loss, translate into meaningful reductions in CV events [51]. Integration of body composition trajectories with biomarkers of inflammation, insulin resistance, and myocardial stress may further enhance mechanistic understanding.

Advances in imaging and digital health technologies also offer new opportunities. Automated analysis of CT and MRI images, often obtained for other clinical indications, may allow opportunistic assessment of visceral fat and muscle mass at scale. In parallel, the application of artificial intelligence and machine learning techniques could facilitate individualized CV risk prediction models that incorporate body composition alongside traditional risk factors [52].

Finally, future interventional studies should prioritize body recomposition as a therapeutic target. Randomized trials designed to increase skeletal muscle mass and reduce visceral or ectopic fat, rather than focusing solely on weight loss, are needed to establish causal relationships and inform clinical guidelines. Such evidence would support a shift toward tissue-specific, personalized strategies for CV prevention and management [53].

CONCLUSION

Body composition represents a clinically relevant and biologically meaningful determinant of CV risk that extends beyond traditional measures of body weight. Accumulating evidence demonstrates that fat distribution, particularly visceral and ectopic adiposity, and the quantity of skeletal muscle independently influence the development, progression, and prognosis of CVD. These findings help explain the substantial heterogeneity in CV risk observed among individuals with similar BMI values.

Assessment strategies focused solely on BMI may therefore overlook high-risk phenotypes, such as individuals with normal-weight central obesity or those with low lean mass. Incorporating measures of central adiposity and muscle mass into clinical evaluation offers the potential to improve CV risk stratification and to better identify patients who may benefit from targeted preventive and therapeutic interventions.

From a clinical perspective, shifting attention from weight loss alone toward body recomposition, reducing harmful fat depots while preserving or enhancing skeletal muscle, may represent a more effective and individualized approach to CV prevention and management. Future integration of body composition metrics into routine practice and risk prediction models may support more precise, patient-centered CV care.

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AI statement: The authors stated that generative AI tools (such as ChatGPT and OpenAI GPT-5) were used only to assist with language refinement, grammar checking, and formatting consistency. No AI system contributed to the conception, analysis, interpretation, or validation of the scientific content. The authors reviewed and verified all text to ensure accuracy and accountability for the final version of the manuscript.

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