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# Obesity and Cardiovascular Disease: Current Knowledge

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## ABSTRACT

Obesity and cardiovascular disease (CVD) remain two of the most consequential noncommunicable diseases worldwide, contributing substantially to global morbidity and mortality. This narrative review synthesizes current evidence on the epidemiology, pathophysiological mechanisms, phenotypic variations, diagnostic assessments, and therapeutic strategies linking obesity and CVD. Global trends indicate a rapid increase in obesity prevalence across age groups, with corresponding rises in CVD incidence, particularly among women, children, and ethnic minorities. Obesity promotes cardiovascular risk through multiple interrelated pathways, including metabolic syndrome, insulin resistance, chronic inflammation, dysregulated adipokine secretion, atherogenic dyslipidemia, hemodynamic overload, and adverse cardiac remodeling. Emerging research highlights the importance of obesity phenotypes such as central adiposity, ectopic fat deposition, and metabolically healthy obesity in refining cardiovascular risk stratification beyond traditional metrics like BMI. Advances in imaging technologies and biomarker profiling have further enhanced noninvasive assessment of cardiometabolic risk. Evidence demonstrates that lifestyle modification remains the cornerstone of obesity management, although pharmacotherapy and bariatric surgery play critical roles in achieving meaningful and sustained weight loss in selected individuals. Special population considerations, including age, sex, ethnicity, and comorbidities such as diabetes, hypertension, and sleep disorders, underscore the heterogeneous nature of obesity-related CVD risk. Public health interventions that modify obesogenic environments, coupled with precision medicine approaches tailored to individual phenotypes, offer promising strategies for prevention. Despite substantial progress, significant knowledge gaps persist regarding long-term outcomes of emerging therapies, interactions between genetic predisposition and environmental exposures, and mechanisms underlying metabolically healthy obesity. Continued multidisciplinary research is essential to improve risk prediction, optimize clinical management, and reduce the growing global burden of obesity-related cardiovascular disease.

**Keywords:** Obesity, Cardiovascular Disease, Metabolic Syndrome, Atherogenic Dyslipidemia, and Cardiometabolic Risk.

## INTRODUCTION

Definition, Prevalence, and Public Health Relevance, Obesity is a chronic disease characterized by excessive abnormal or fat accumulation, with a Body mass index (BMI) of ≥30 kg/m2; Cardiovascular disease is defined as a pathophysiological process affecting the heart or blood vessels; Cardiovascular disorders remain the leading global cause of morbidity and mortality [1]; Obesity-related risks for cardiovascular disease are elevated across all age groups, with an earlier onset observed in females, children, and ethnic minorities [2]. The present review aims to provide healthcare professionals with a rigorous, evidence-based synthesis of current knowledge relating to these two conditions; Main questions considered include pathophysiological conditions linking obesity and cardiovascular disease, the role played by obesity phenotype classification in determining cardiovascular risk or intervention effectiveness, and strategies for assessment, management, and risk reduction[7].

### Epidemiology of Obesity and Cardiovascular Disease

Obesity represents a significant global health concern. In 1990, 857 million adults worldwide were classified as obese, and this figure had risen to 2.52 billion by 2017. By 2030, an estimated 1.12 billion adults globally (11.6% of

the population) will be obese [3]. Obesity is a well-recognized risk factor for the emergence of cardiovascular disease (CVD). The risk attributable to obesity is complex, varying significantly by age, sex, and ethnicity, and differing in the strength and specific nature of the association across circumstances [8]. By understanding the epidemiological patterns and trends of obesity and CVD, it follows that the risk of cardiovascular events among the obese remains the subject of ongoing investigation [4]. CVD is a particularly diverse term [10]. Despite the wide variety of conditions that fall under this heading, they all share a common pattern: some form of obstruction to blood flow to the heart and brain, the two organs that consume by far the highest fraction of the body's total glucose and oxygen. Risk increases steadily with age, diabetes, high blood pressure, elevated levels of circulating lipids, and many other risk factors [7].

**Pathophysiological Links between Obesity and Cardiovascular Disease**

Adiposity is associated with increased risk of metabolic syndrome, which raises cardiovascular risk [2]. The metabolic syndrome is defined by the co-occurrence of central obesity, hyperglycemia, hypertension, and dyslipidemia. The underlying mechanisms are thought to include hyperinsulinemia and dysregulation of insulin-sensitive tissues, such as skeletal muscle and liver. These lead to insulin resistance, increased free fatty acid flow from adipose tissue and deposition in non-adipose tissues, and disruption of glucose homeostasis [5]. Adipose tissue is an active endocrine organ that secretes a range of cytokines and other bioactive factors collectively termed adipokines. Obesity leads to expansion of white, brown, and peri-infarct adipose tissue, which results in altered adipokine secretion and increased vascular risk [7]. Furthermore, adipose tissue is the largest reservoir of pro-inflammatory mediators in the body; obesity causes local and systemic inflammation characterized by infiltration of activated M1 macrophages into visceral adipose tissue [4]. The consequent production of reactive oxygen species and release of pro-inflammatory cytokines, including interleukin-6 and tumor-necrosis factor-alpha, have been implicated in the development of atherosclerosis and acute coronary events [8]. Obesity also causes profound alterations in lipid metabolism, leading to atherogenic dyslipidemia. Triglyceride-rich lipoproteins are elevated owing to increased hepatic production and decreased postprandial lipolysis 6. High-density lipoprotein cholesterol (HDL-C) concentration is often low owing to increased transfer of cholesteryl esters from HDL to triglyceride-rich particles via cholesteryl-ester transfer protein (CETP), high rates of hepatic HDL-C uptake, and abnormal reverse cholesterol transport [8]. Hypertensive heart disease and heart failure are prevalent in obesity, and are progressively more common and occur at a younger age with increasing degrees of adiposity. Obesity exerts direct effects on the myocardium, which augment pressure and volume overload secondary to hypertensive heart disease and cardiac output increase secondary to peripheral vascular resistance decrease [6]. The net effect is an adverse alteration of ventricular structure characterized by hypertrophy and diastolic dysfunction [4].

**Metabolic Syndrome and Insulin Resistance**

Established criteria for metabolic syndrome, which encompasses insulin resistance, visceral obesity, lipid disturbances, and hypertension, delineate a potent cardiovascular risk profile [7]. Insulin resistance impairs suppression of hepatic gluconeogenesis and inhibition of lipolysis in adipocytes, predisposing to elevated serum glucose and free fatty acids (FFA)[8]. Insulin resistance also intensifies the atherogenic dyslipidemia associated with obesity, characterized by increased plasma triglycerides, low high-density lipoprotein (HDL) cholesterol, and a predominance of small dense low-density lipoprotein (LDL) particles [7]. Reduced HDL levels augment the risk of arterial disease, while elevated postprandial lipemia, driven by the secretion of triglyceride-rich very low-density lipoproteins (VLDL) from the liver, further compounds this risk. Within the vascular endothelium, FFAs and their metabolites are implicated in promoting inflammation, thrombosis, and the formation of atherosclerotic plaques, all proatherogenic mechanisms exacerbated by elevated concentrations of circulating nonesterified FFAs [8].

**Inflammatory Pathways and Adipokines**

Obesity-related cardiovascular disease is critically linked to the inflammatory pathways activated by proinflammatory cytokines and adipokines produced by adipose tissue [16]. Obesity triggers a cascade of inflammatory processes involving different cell types, chemokines, and cytokines that, when dysregulated and chronic, facilitate atherosclerosis and endothelial dysfunction, both of which are closely correlated with increased cardiovascular risk [9]. Understanding the relationship between obesity and cardiovascular disease requires a clear appreciation of inflammatory systems and the multiple metabolic effects of the principal adipokines [10]. Adipose tissue has an endocrine function, producing bioactive substances that regulate cardiovascular functions; white adipose tissue, besides storing energy, releases various cytokines and chemokines involved in the control of immune responses, appetite, energy homeostasis, vascular function, and blood pressure [11].

**Lipid Metabolism and Atherogenic Dyslipidemia**

Obesity is associated with an increased risk of cardiovascular disease through various mechanisms, including alterations in lipid metabolism that give rise to a characteristic atherogenic dyslipidemia [15]. Dyslipidemia,

which presents as an excess of triglyceride-rich lipoproteins and a deficiency of high-density lipoprotein (HDL) particles, is recognized as a fundamental cause of vascular disease in obesity [12]. Excess calories and nutrients induce changes in the insulin-glucose-adipokine axis that exacerbate dyslipidemia and rigidly maintain elevated plasma triglyceride (TG)-rich lipoprotein (TGRL) concentrations, even in the fasting state. Body fat mass, ectopic fat accumulation, inflammation, and adverse expansions of the gut and liver microbiomes have all been implicated as contributing determinants of the obesity-related vascular dyslipidemic triad. Obesity exerts additional deleterious effects on the metabolic and functional properties of HDL particles, resulting in the emergence of HDL dysfunction [13]. Accumulating evidence has shown that the quantity and quality of HDL and its associated apolipoprotein A-I (ApoA-I) strongly influence vascular health and the risk of cardiovascular disease. Clinically, a striking and virtually universal decrease in HDL-cholesterol (HDL-C) plasma levels can be observed early in the development of metabolic syndrome and obesity [12]. Furthermore, obesity alters the metabolism of low-density lipoprotein (LDL) particles, producing variations in LDL size and density. The emergence of small, dense LDL particles and the upsurge of non-ApoB lipoproteins contribute to an atherogenic dyslipidemia that remains prevalent even after weight loss [14].

**Hemodynamic Alterations and Cardiac Remodeling**

Obesity has been linked to notable alterations in blood pressure and cardiac geometry [7]. Obese individuals exhibit a higher prevalence of hypertension, characterized by increases in both systolic and diastolic values and elevated resting heart rates compared to normal-weight individuals; these parameters are aggravated by obesity duration. A positive correlation between body mass index (BMI) and mean arterial pressure persists across different ages and populations, suggesting that obesity is a fundamental determinant of higher blood pressure [6]. Furthermore, the recent increase in mean BMI among adolescents may explain the observed rise in hypertension prevalence within this cohort [10]. The development and maintenance of elevated blood pressure during obesity have been associated with increased vascular resistance, although obesity-related variations in cardiac preload and afterload may also play a role [11]. Obese subjects exhibit elevated cardiac output, where both stroke volume and heart rate contribute to the augmented volume. Cardiac output exceeds normal values not only during exercise but also at rest and during sleep, independent of obesity comorbidities [12]. Cardiac volume overload remains a characteristic feature of obesity; left ventricular (LV) volume, end-diastolic anterior–posterior diameter, stroke volume, and cardiac output increase proportionally with BMI elevation, even in the absence of associated comorbidities [13]. The ensuing pressure overload induces concentric hypertrophy, titled "obesity-related left ventricular hypertrophy." Cardiac diastolic function is deteriorated by obesity at an early age; this is aggravated by increased pressure and volume overload sustained throughout the lifetime, leading to heart failure. Obesity affects both cardiac and vascular morbidity and mortality, with men subjected to greater cardiovascular risk and heart failure progressively occupying dominant positions in mortality [14].

**Obesity Phenotypes and Cardiovascular Risk**

Obesity epidemics have reached crisis levels, exacerbating cardiovascular and metabolic diseases. The majority of studies examining the links between obesity and cardiovascular diseases focus on body mass index rather than evaluating meaningful differences in morbidities caused by the different obesity phenotypes [5]. Degree and distribution of fat mass influence cardiovascular risk. Excess fat mass, whether generalised or specifically located in the abdomen, promotes the development of morbidity [2]. Unlike patients with uncomplicated obesity, those with metabolic-complex and obesity-related comorbidities are conventionally deemed to present a potentially more serious condition [6]. For this reason, patients with non-alcoholic fatty liver disease, diabetes mellitus, hypertension, sleep apnoea, dyslipidemia, depressive disorders, and lower limb osteoarthritis, among obesity-related comorbidities, are generally considered at greater cardiovascular risk [7]. Epidemiological studies in different populations have demonstrated a significant reduction in life expectancy associated with cardiovascular disease estimates attributable to obesity, diabetes, and sleep apnoea [1]. Considering the potential adverse cardiovascular effects of obesity and other cardiovascular risk factors, precision medicine has emerged to individualise the treatment approach aimed at the cardiometabolic health of patients affected by obesity [8]. Many genetic and epigenetic determinants of obesity phenotypes have been identified that are more associated with different cardiovascular phenotypes than body mass index measurements [9]. On the basis of clinical and biometric parameters collected over decades, machine learning techniques are being used to divide patients into non-weight-related clusters, revealing that some obesity-related comorbidities occur independently of weight gain. Research using imaging techniques of fat distribution is being employed to predict additional cardiovascular risk in people diagnosed with different obesity phenotypes [3].

**General Obesity versus Central Adiposity**

Waist circumference (WC) and waist-to-hip ratio (WHR) are surrogates that provide additional information on the distribution of body fat and the associated cardiovascular disease (CVD) risk [13]. Hip circumference appears to be protective against general obesity, while larger WC and WHR are additional clear indicators associated with

elevated risk. General obesity, closely related to body mass index (BMI), is a poor risk stratifier since larger body fat mass is frequently offset by concomitant increases in FFM, particularly in body builders, female athletes, and the very young, 15 in whom BMI may be misleading [11]. The interaction between adipose tissue and the cardiovascular system is complex, with the functional quality of adipose tissue rather than the total amount or classical measurements such as BMI emerging as more relevant [10]. Perivascular and other regional fat depots play an important role in CVD risk, both directly and through modulation of metabolic comorbidities 16. Depots of adipose tissue comprise 5–60% of total body weight [15]. Regional distribution, depot size, and depot function are key determinants of cardiometabolic risk. Waist-to-hip ratio conveys direct information about the distribution and functionality of fat depots and is a better predictor of CVD risk than BMI; a 0.1 increase in WHR corresponds to a 5% higher risk for both men and women [16]. Increased waist circumference is associated with higher CVD mortality risk, even in individuals classified "normal-weight" by BMI. General obesity measured by BMI alone poorly reflects CVD risk, and the Framingham Heart Study cites close interlinkage between body weight gain and the subsequent development of heart failure, coronary heart disease, stroke, and total CVD mortality. Consequently, the w ≥ 102 cm / h ≥ 88 cm distinction satisfies risk stratification with great clinical relevance in general obesity when BMI fails to fulfill the task.

**Severity and Distribution of Adiposity**

Abdominal obesity is associated with an increased risk of cardiovascular disease (CVD). Therefore, waist circumference and waist-to-hip ratio (WHR), which are anthropometric indices that measure central fat distribution, have been proposed to be better predictors of CVD risk than body mass index (BMI) [17]. Nonetheless, the discrete assessment of central obesity overlooks how differences in fat accumulation across diverse anatomical areas contribute to risk estimation [27]. Regional fat deposition, depot volume, and local tissue characteristics are important for both cardiometabolic and cardiovascular risk. Mesenteric, epicardial, and perivascular fat are key depots of visceral fat associated with adverse cardiovascular outcomes. Visceral fat accumulation is linked to metabolic dysregulation and inflammation [25]. Consequently, the total volume of MEFP represents a better correlate of CVD risk than simple waist circumference or WHR. Ectopic fat deposition within the liver, pancreas, heart, and skeletal muscle is also associated with cardiovascular [15]. The pathophysiological mechanisms linking ectopic fat deposition to CVD are only partially understood. However, excessive fat accumulation in non-adipose tissues promotes lipotoxicity, steatosis, inflammation, and fibrosis, triggering a cardiovascular cascade [22]. Overall, obesity is characterized not only by excess mass but also by abnormal patterns of fat accumulation. Body shape and the presence of ectopic fat can modify the prognosis in overweight and obese patients. Weight gain in the long term can augment the likelihood of developing type 2 diabetes mellitus or CVD [21].

**Metabolically Healthy Obesity and Cardiovascular Risk**

Individuals meeting the criteria for metabolically healthy obesity remain at increased cardiovascular risk compared to their metabolically healthy nonobese counterparts, with process-driven studies suggesting the presence of unmeasured or insidious metabolic derangements [18]. Metabolically healthy obese men show an excess prevalence of cardiac fatty deposits [19]. Systematic reviews document a lower risk of cardiovascular disease, type 2 diabetes, and premature all-cause mortality among metabolically healthy obese individuals compared to their metabolically unhealthy counterparts, yet a higher risk relative to metabolically healthy normal-weight individuals, underscoring the crucial role of metabolic health independent of weight status [18]. This underscores the necessity of preventing metabolic disturbances among those with excessive adiposity and all individuals with normal weight [13].

**Noninvasive Markers and Diagnostic Assessments**

Paralleling the importance of assessing the obesity phenotype, estimating specific noninvasive markers has proven useful in predicting cardiovascular disease in patients affected by obesity [14]. Cardiovascular risk can be estimated by multiple anthropometric indices, such as body mass index (BMI) and waist circumference, and by imaging measures, including dual-energy X-ray absorptiometry (DXA) and magnetic resonance imaging (MRI) or computer tomography (CT) [15]. Several biomarkers have also emerged to assist with risk stratification, including high-sensitivity C-reactive protein, adipokines (e.g., leptin, resistin), natriuretic peptides, various lipidomic profiles, and the quantification of glucose metabolism [20]. Novel assessments have also provided insight into the presence and severity of atherosclerosis in patients with morbid obesity. An additional dimension of atherosclerosis, extra-media thickness (EMT), has recently been measured along with epicardial (EAT) and periarterial (PAT) adipose tissue parameters [12]. These indices correlate with coronary heart disease incidence and stage [21]. However, cardiovascular risk profiles remain heterogeneous, and routine evaluation of additional phenotypic or noninvasive markers continues to be beneficial to predict cardiovascular disease and to tailor prevention strategies to those most in need [17].

## Anthropometric Indices and Imaging Measures

Body mass index (BMI) is the most widely used measure of general obesity, but its relation to cardiovascular disease (CVD) and its associated disease states and comorbidities (together referred to as CVD risk) depends on many factors, such as race/ethnicity, sex, age, and a variety of clinical covariates and traditional risk factors [20]. Other anthropometric indices, such as waist circumference (WC), waist-to-hip ratio (WHR), and waist-to-height ratio (WHtR), have been proposed as potential markers more closely linked to CVD risk and health consequences related to body fat distribution and central obesity than BMI [22]. Imaging techniques, including dual-energy X-ray absorptiometry (DXA) to measure body composition and quantify fat mass (FM) or fat-free mass, magnetic resonance imaging (MRI) to assess multi-focality of fat depot distribution, and computed tomography (CT) to quantify the abdominal fat area (AFA) have been employed to further identify cardiometabolic risk associated with excess adiposity [23].

## Biomarkers of Cardiovascular Risk in Obesity

Aside from anthropometric parameters and imaging techniques, noninvasive blood-based biomarkers are essential for assessing cardiovascular risk in patients with obesity [4]. Such biomarkers provide insight into the underlying pathophysiological processes driving cardiovascular disease and can indicate treatment response [23]. Several candidate biomarkers show promise for risk stratification [8]. For example, the proinflammatory mediator high-sensitivity C-reactive protein (hs-CRP) is produced by the liver in response to inflammatory cytokines such as interleukin-6. Increased hs-CRP correlates with obesity and metabolic syndrome and independently predicts cardiovascular events in various populations [26]. Increased circulating concentrations of the adipokines leptin and retinol-binding protein 4 also independently associate with cardiovascular risk [22]. Furthermore, elevated circulating levels of natriuretic peptides such as B-type natriuretic peptide (BNP) reflect obesity–related neurohormonal activation and left ventricular stress and independently predict cardiovascular events. Enhanced lipidomic profiling holds promise for identifying potentially hazardous triglyceride-rich lipoprotein subclasses and intermediates of de novo lipogenesis that accumulate in response to obesity [21]. Impairments in glucose homeostasis, reflected by elevated fasting plasma glucose or decreased insulin sensitivity measured using homeostasis model assessment, independently predict cardiovascular events [24].

## Therapeutic Interventions and Outcomes

The obesity crisis threatens optimization of cardiovascular health and disease prevention. Alterations associated with obesity drive progressive impairment of cardiovascular risk burden [1]. For the prevention of cardiovascular disease, caloric restriction and increased physical activity are foundational evidence-based interventions to achieve and sustain weight loss [6]. However, obesity pharmacotherapy and referral for bariatric surgery are crucial adjuncts for obesity treatment when weight loss via lifestyle modifications alone is inadequate [14]. Cardiovascular risk factor and comorbidity improvements have been observed after initiation of obesity pharmacotherapy, and this treatment alone, independent of weight loss, may favorably influence blood pressure and glycemic control. Comprehensive clinical care plans that include implementation of multimodal approaches have improved weight loss maintenance and control of cardiovascular risk factors and comorbidities [16]. Through a multidisciplinary team approach, the complexity of the obesity crisis can be more efficiently addressed [13].

## Lifestyle Modification and Weight Loss

Excess body weight increases cardiovascular disease risk, independently of other risk factors [25]. The most effective approach to weight loss is lifestyle modification, yet obesity is resistant to treatment. The United States Dietary Guidelines for Americans recommend restricting caloric intake and increasing physical activity to achieve a 5% to 10% reduction in body weight [1]. Physical activity should be planned to accomplish 150 to 300 minutes of moderate-intensity activity per week. Bariatric surgery and pharmacotherapy are alternatives for patients with morbid obesity or cardiovascular disease. Risk of type 2 diabetes, hypertension, and cardiovascular disease increases at a body mass index of 25 kg/m2. Education and rewards for lifestyle changes have been shown to enhance success [23].

## Pharmacotherapy for Obesity and Cardiovascular Risk Reduction

Several drugs are approved for weight management and obesity-related cardiovascular risk reduction, and others are in development [17]. The agents currently in use can be broadly classified according to their mechanisms of action. Some drugs stimulate energy expenditure by central nervous system (CNS) mechanisms, while others target peripheral metabolic organs [14]. A few act on glucose-dependent pathways, aimed at improving glycemic control and are considered in the context of diabetes; they induce weight loss and also decrease cardiovascular risk, but were primarily developed for other indications [18]. The selective serotonin-reuptake inhibitor fluoxetine was the first drug approved for obesity treatment. Although the effects on weight are modest, there is some evidence of accompanying risk reduction for type 2 diabetes and related sequelae [19]. Other serotonergic agents associated with weight gain have been withdrawn because of cardiac valvulopathy. Tetrabenazine, approved for hyperkinetic

movement disorders, is a central dopamine-depleting agent that can induce marked weight loss. Its potential for wider obesity treatment is being examined. Other central dopamine receptor antagonists (e.g., haloperidol) act through the aphagia observed as a side effect of their main indications [20]. The clinical use of these drugs for weight control is limited because of ethical, legal, and safety concerns, but clarifying the mechanisms involved could provide new therapeutic avenues [5]. Triolex is an investigational combination of bupropion and naltrexone acting on both the mesolimbic and the hypothalamic pathways [22]. Naltrexone, an opiate antagonist, is a non-selective blocker that, when administered alone, induces weight gain [27]. Its combination with bupropion, a non-selective inhibitor of norepinephrine and dopamine reuptake, increases dopaminergic transmission in the nucleus accumbens and enhances the reward value of food [26]. An associated decrease in body mass index (BMI) was recently observed in a proof-of-principle study on overweight patients with major depressive disorder, supporting the hypothesis of a common pathophysiological component in both conditions characterized by anhedonia[25].

**Bariatric Surgery: Indications and Cardiovascular Benefits**

Obesity, defined as an excessive accumulation of body fat, is commonly diagnosed using the body mass index (BMI), which assesses weight relative to height [26]. The disease constitutes a major global public health concern, as rates have increased worldwide; it is estimated that 39 million children under the age of five were overweight or obese in 2020. Obesity is also linked to reduced quality of life, increased healthcare utilization, and shortened life expectancy [19]. The term cardiovascular disease is used to denote the organization of the cardiovascular system and all pathologies associated with it [27]. Cardiovascular disease is the leading cause of death, global disability, and absence from work, making it a major public health concern. This review focuses on the association between obesity and cardiovascular disease in adults [22].

**Special Populations and Considerations**

Obesity increases the risk of cardiovascular disease through direct and indirect mechanisms [1]. Various comorbidities, such as diabetes, hypertension, and sleep disorders, exacerbate the burden and should therefore be prioritized in treatment [28]. The combination of low mortality with considerable coronary artery disease noted in certain populations undergoing bariatric surgery suggests that obesity's influence is context-dependent. Obesity is a concern across age groups, but the epidemiology of patients younger than 18 remains incomplete [29]. In the absence of overt risk factors, attentiveness remains warranted, owing to emerging evidence that metabolic alterations may develop even before clinical cardiovascular disease appears [27].

**Age, Sex, and Ethnicity**

Age, sex, and ethnicity substantially influence the risk of obesity and associated cardiovascular disease [29]. Although some risk levels are similar among different groups, marked variability exists. The absolute risk associated with obesity remains lower among younger individuals, those who are female, and some racial and ethnic groups [26]. However, individuals in these categories still have increased rates of obesity-related disease compared with their nonobese counterparts [28]. For these populations, managing obesity may be particularly urgent to prevent the development of conditions such as hypertension or metabolic syndrome, which strongly and independently elevate CVD risk [25]. Effects also differ according to metabolic and other individual characteristics, sometimes leading to the contradictory designation of a population as both "at risk of obesity-related CVD" and "less at risk than others"[23]. In these groups, greater emphasis should be placed on monitoring and controlling comorbidities rather than on promoting weight loss as a primary CVD-prevention strategy [27].

**Comorbid Conditions: Diabetes, Hypertension, and Sleep Disorders**

Obesity is associated with an increased risk of type 2 diabetes, hypertension, and sleep disorders, all of which are themselves risk factors for cardiovascular disease [27]. Several studies suggest that sleep disorders may play a direct role in the progression of cardiovascular disease in obese patients, and the prevalence of both sleep disorders and obesity is on the rise globally [28]. In a Danish study, the incidence of type 2 diabetes increased by about 200% among patients with sleep-disordered breathing over the 2000 to 2009 period, and about 80% of type 2 diabetes patients also developed obstructive sleep apnoea (OSA) during the same period. In the year prior to the start of positive airway pressure treatment, the incidence of newly diagnosed type 2 diabetes and hypertension was estimated to be 16% and 47%, respectively [30]. Patients with diabetes may also be at a greater risk of developing severe nocturnal hypoxaemia and compliance with continuous positive airway pressure therapy among those with OSA [31].

**Public Health Implications and Preventive Strategies**

Public health campaigns targeting obesity, like a campaign warning about the dangers of tobacco, could lead to social norms promoting healthy behavior and discouraging unhealthy habits and foods. Effective campaigns seek to create supportive environments and reduce access to unhealthy behaviors [29]. Several of the same steps taken to limit tobacco use can also reduce obesity rates [20]. Many programs can help modify environments, and a mix of approaches has a greater effect. Multi-faceted interventions have been effective in modifying behaviors that people find difficult to change [25].

### Knowledge Gaps and Future Directions

In the past decades, obesity has become a worldwide epidemic, accompanied by a remarkable rise in cardiovascular disease (CVD) morbidity and mortality [30-34]. In parallel with high earlier-life risks for acute coronary syndromes, hypertension, and heart failure, an extended age-related increase in CVD burden has emerged, now extending to late-onset stages in populations that were traditionally CVD-free. Globally, nearly one-third of adults are classified as overweight, representing a dramatic increase compared with earlier periods. Interventions against obesity and associated CVD have become a public health priority [32-35]. A better understanding of obesity and its relationship to cardiovascular risk factors and CVD across the life span is paramount for effective prevention at a population level [36, 37].

## CONCLUSION

Obesity and cardiovascular disease are intimately interconnected public health challenges that continue to expand in prevalence and complexity. Evidence demonstrates that obesity contributes to CVD through diverse biological pathways, including metabolic, inflammatory, hemodynamic, and structural mechanisms. Importantly, cardiovascular risk is not solely determined by total body weight but by patterns of fat distribution, the presence of ectopic fat, and metabolic health status. Central adiposity, visceral fat accumulation, and obesity-related comorbidities such as diabetes, hypertension, and sleep disorders substantially amplify CVD risk, while even individuals categorized as metabolically healthy obese exhibit elevated long-term cardiovascular vulnerability. Improvements in anthropometric assessment, imaging modalities, and biomarker profiling enhance early detection and risk stratification, supporting more precise and individualized management strategies. Therapeutically, lifestyle modification remains foundational, yet is often insufficient alone, necessitating incorporation of pharmacotherapy and bariatric surgery for appropriately selected patients. Public health interventions targeting obesogenic environments, food systems, and health inequities are critical to reducing population-level risk. Despite significant scientific advances, critical gaps persist regarding the long-term cardiovascular outcomes of pharmacological agents, the mechanisms underlying heterogeneity in obesity phenotypes, and the optimal integration of precision medicine approaches. Addressing these gaps will require sustained research, multidisciplinary collaboration, and robust policy action. Ultimately, reducing the global burden of obesity-related cardiovascular disease demands a comprehensive strategy encompassing prevention, early detection, individualized care, and health system strengthening.

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