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Obesity and Metabolic Syndrome: Interconnections, Mechanisms, and Implications for Health

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ABSTRACT

Obesity and metabolic syndrome (MetS) are two interrelated global health challenges whose prevalence continues to rise across all age groups and socioeconomic settings. Obesity, particularly central adiposity, plays a critical pathophysiological role in the development and progression of MetS, which is defined by the clustering of abdominal obesity, dyslipidemia, hypertension, and impaired glucose regulation. This narrative review synthesizes current scientific evidence on the definitions, diagnostic criteria, epidemiology, and mechanistic links between obesity and MetS. Key interconnected mechanisms include adipose-tissue dysfunction, chronic inflammation, insulin resistance, lipotoxicity, and obesity-related vascular alterations. Together, these pathways promote a spectrum of clinical consequences, notably cardiovascular disease, type 2 diabetes, non-alcoholic fatty liver disease, obstructive sleep apnea, osteoarthritis, and certain cancers. The review further discusses therapeutic strategies including lifestyle modification, pharmacological interventions, bariatric surgery, and emerging precision-medicine approaches. Public health implications underscore the need for multisectoral policies that address obesogenic environments and health inequities. Finally, future research directions highlight the importance of longitudinal studies, biomarker discovery, and implementation science to strengthen prevention and management strategies. Understanding the complex interplay between obesity and metabolic syndrome is essential to mitigating their global burden and improving long-term population health outcomes.

Keywords: Metabolic Syndrome, Adipose Tissue Dysfunction, Insulin Resistance, Lipotoxicity, and Obesity-Related Inflammation

INTRODUCTION

Obesity and metabolic syndrome represent two of the greatest public health challenges of the 21st century. Their consequent health impacts have extensive repercussions for economic productivity and human capital [1-6]. Growing evidence indicates that obesity is central to the development and progression of metabolic syndrome. Hence, elucidating the interrelationship between these conditions, along with the mechanisms that underlie them, is critical in mitigating their emergence and curtailing their spread [7-9]. Obesity is generally defined as an excessive accumulation of fat that impairs health. Although it can be quantified through direct measurements or imaging techniques, it is routinely assessed using the widely adopted body mass index (BMI) classification established by the World Health Organization (WHO) [10-14]. According to this standard, a BMI ≥30 kg/m2 indicates obesity. Additionally, waist circumference (WC) provides an estimate of abdominal (visceral) fatness, which is a key determinant of obesity-related health risks [4, 5]. To date, recommendations suggest a WC cutoff point of >102 cm for men and >88 cm for women [15-19]. The major component of the metabolic syndrome is abdominal obesity, which is represented as a separate diagnostic criterion in most widely used definitions. Other components include dyslipidemia (triglycerides and/or high-density lipoprotein cholesterol), hypertension, and glucose intolerance [20-24].

Definitions and Diagnostic Criteria

Body weight is a widely used measure of overweight or obesity. Body mass index (BMI), defined as weight in kilograms divided by the square of height in meters, serves as a measure of relative body weight and is heavily used in epidemiological research and clinical settings [25-29]. The World Health Organization defines the following BMI categories (underweight: < 18.5 kg/m2, normal range: 18.5–24.9 kg/m2, overweight: ≥ 25 kg/m2, and obesity: ≥ 30 kg/m2) [30-36]. The American Heart Association also states that a BMI ≥ 25 kg/m2 qualifies as overweight, while ≥ 30 kg/m2 indicates class I obesity [37-40]. BMI correlates well with total body fat in most populations, with the correlation only declining above class II obesity (BMI ≥ 35 kg/m2) or when BMI is low (< 20 kg/m2) 3. However, it remains suboptimal at the individual level since the BMI value does not distinguish between lean mass and fat mass, nor does it indicate regional fat distribution, which could be fat in non-adipose tissue (visceral fat). Therefore, additional measures may be needed to evaluate body composition and fat distribution for individuals with obesity complications or those at very low risk [41-46]. Waist circumference (WC) is used both as an independent risk factor and as an indicator of abdominal (central) obesity. Certain guidelines have proposed obesity thresholds based on WC alone [47-49]. For example, WC ≥ 102 cm in men and ≥ 88 cm in women indicates abdominal obesity according to the International Diabetes Federation criteria. Others, such as waist-to-height ratio (WHtR) or waist-to-hip ratio (WHR), are also considered markers of diabetes and cardiovascular disease, but their associations are weaker than waist alone [50-57]. Body composition from dual-energy X-ray absorptiometry can provide fat mass, fat-free mass, and trunk fat. Fat mass percentage is recommended as an additional measure to define different obesity classes [58-63]. A fat mass percentage of≥ 25% in men and ≥ 35% in women qualifies as obesity according to the National Institutes of Health definition. Fat mass cannot be estimated from standard clinical data, and thus BMI remains the most widely used classification criterion [64-68]. The metabolic syndrome (MetS) was first defined in 1923, characterizing a syndrome with hypertension, hyperglycemia, and hyperuricemia. Reaven's late 1980s description of syndrome X, describing insulin resistance, included impaired glucose tolerance, hyperinsulinemia, high triglycerides, low HDL, and hypertension [8]. The concept of visceral adiposity was added later [69-74]. Different criteria have been proposed, including WHO's 1999 definition focusing on insulin resistance and obesity, and the ATP III criteria requiring three out of five factors: high waist circumference, elevated triglycerides, low HDL, high blood pressure, and fasting glucose. The IDF aimed to unify definitions, emphasizing central obesity [75-80]. Currently, the consensus diagnosis involves any three of these criteria, with specific thresholds depending on populations. Abdominal obesity (waist and/or WHtR), dyslipidemia (TG, HDL), blood pressure (systolic and/or diastolic), and glucose (fasting) determine 4 out of the 5 criteria, wherein body mass index (BMI) and waist circumference (WC) help assess adiposity [81].

Epidemiology and Global Trends

Obesity and its health-related sequelae, including metabolic syndrome, have become a global health crisis [4]. The prevalence of obesity in both developed and developing countries has doubled over the past decade. While genetic factors, sedentary lifestyles, and overnutrition are critical contributors, evidence indicates that prenatal and perinatal developmental factors significantly influence obesity risk [5]. Research shows that conditions of nutritional surplus or deficiency during in utero and early postnatal life predispose individuals to obesity and related health issues later, with the potential for epigenetic transmission to future generations [4]. Obesity affects at least 400 million adults worldwide with a BMI of 30 or greater; over 6 billion are overweight with a BMI of 25 or greater [3]. It poses a major threat to global health, with metabolic syndrome characterized by abdominal obesity, insulin resistance, hypertension, and dyslipidemia, leading to increased cardiovascular disease and diabetes risk [6]. Obesity is a significant risk factor for metabolic syndrome, typically manifesting as abdominal obesity, insulin resistance, dyslipidemia, and hypertension [5]. Development of insulin resistance due to excess central adiposity is a key event in syndrome origin and progression, involving impaired insulin action and external factors like genetics [6]. Relevant molecular elements underlying insulin resistance, partly mediated by nuclear PPAR, may contribute to clinical components, although associations remain weakly understood [7]. Genetic predisposition has a significant social and cultural component; knowledge on interactions of genetic susceptibility with environmental influence on obesity and related disorders is often limited [4].

Pathophysiological Links between Obesity and Metabolic Syndrome

Obesity and its related disorders may reflect impaired adipose tissue homeostasis. Adipose tissue dysfunction can trigger metabolic syndrome with excess weight, and both conditions can occur independently [7]. Adiposity promotes ectopic fat accumulation, liver steatosis, dyslipidemia, hyperglycemia, β-cell stress, and impairment of glucose homeostasis. Adipose tissue regulates metabolic homeostasis by secreting adipokines. Obesity-associated changes in adipose tissue structure, biochemistry, and cell composition lead to abnormal adipokine secretion and the inflammatory macrophage cholesterol deposit [9]. Metabolically activated macrophages secrete proinflammatory cytokines that induce inflammation and insulin resistance in adjacent tissues, leading to further

dysregulation of metabolism and a vicious cycle of chronic inflammation [8]. Cyclic adenosine monophosphate, diacylglycerol, diacylglycerol, and ceramide are among the signalling lipids stimulated by obesity-related insulin resistance that are uniquely generated in an anomalous way by several organs, including the pancreas, liver, and adipose tissue[7]. These aberrant lipids are harmful to both the generation and the growth of nonalcoholic fatty liver disease, atherogenic dyslipidemia, and hypertriglyceridemia, ultimately promoting cardiovascular disease, type 2 diabetes, and related mortality [3]. Substantial medial hypertrophy, an early indicator of hypertension, has been observed. Overweight and obesity activate the renin–angiotensin–aldosterone system, which elicits arterial changes that lead to hypertension [2]. Acute and chronic local tissue inflammation stimulate cytokine secretion, which induces proatherogenic changes in lipoprotein metabolism and amplifies the major metabolic dysregulations associated with obesity [6]. The proliferation of inflammation-related pathways disrupts cardiovascular, metabolic, and homeostatic system functions and modifies the sequence of tissue-cell specialization. In particular, the ability of standpoints that contribute to tissue cell specialization to regulate pro- and anti-inflammatory signals is markedly disrupted [7].

Adipose Tissue Dysfunction

Obesity brings about adverse changes in adipose tissue characterized by ectopic fat accumulation, hypertrophied adipocytes, and inflammation [4]. In clinical settings, these changes are frequently observed, and their extent is directly associated with the presence of metabolic syndrome-associated disorders. Particularly, the dysregulation of adipokines secreted by adipose tissue redistributes fat in a manner that promotes these disorders, creating a progressive cycle hostile to metabolic wellbeing [5]. The hypertrophy of adipocytes activates dedifferentiation pathways whereby preadipocytes convert into fibroblast-like cells, leading to excessive deposition of fibrous matrix proteins and ultimately fibrosis [5]. Co-incident activation of immune processes induces switching of macrophage activity from diffuse tissue homeostasis to focal inflammatory activity, triggering further adipocyte dysregulation and broad disruption of metabolic processes [9].

Insulin Resistance and Glucose Homeostasis

Obesity is a key health problem with interconnections to metabolic syndrome and type 2 diabetes; as rising adiposity continues to affect populations globally, understanding how the condition operates pathophysiologically is essential[8]. Major mechanisms involved include adipose tissue dysfunction, insulin resistance and glucose homeostasis, inflammation and immune activation, lipotoxicity and dyslipidemia, and hypertension and vascular alterations [6]. Adipose tissue dysfunction includes excess adipose tissue (abdominal fat mass) and alterations to adipokine profiles [10]. Expansion of adipose tissue mass during weight gain generates ectopic fat deposition in the liver (steatosis), pancreas (islets), heart (disease), skeletal muscle (lipotoxicity), and kidneys (interstitial), triggering secondary disturbances [11]. Also, ectopic deposition of toxic ceramides, diacylglycerol, and cytokines occurs in the liver and muscle, promoting steatosis, dyslipidemia, inflammation, and insulin resistance, respectively [3]. The condition of insulin resistance is further characterized by β-cell stress from failure to adequately suppress hepatic glucose production during fasting, hyperinsulinemia from increased secretory demands on β-cells to maintain normal glucose levels during postprandial states, and decoupling of insulin dynamics in genetic, physiological, and pathological conditions [12]. Its understanding is relevant in the study of the underlying links between obesity and metabolic syndrome.

Inflammation and Immune Activation

The onset of obesity-associated metabolic dysfunction is accompanied by altered gene expression and secretion of a variety of factors by adipose tissue, which subsequently contributes to the development of metabolic syndrome [6]. An early change in the adipokine milieu is the decreased secretion of the anti-inflammatory adipokine adiponectin [7]. A number of other factors also begin to change at this stage; an example is lipocalin-2 (also called neutrophil gelatinase-associated lipocalin), an adipocyte-secreted protein that promotes leukocyte recruitment into inflamed tissues [13]. The infiltration of activated immune cells and the release of pro-inflammatory cytokines that follow become the basis for an aggravation of the pathological state of adipose tissue. Monocyte recruitment is promoted through the increased expression of adhesion molecules such as vascular cell adhesion molecule-1 (VCAM-1) and intracellular adhesion molecule-1 (ICAM-1) by adipocytes, which is induced by inflammatory signals such as tumor necrosis factor alpha (TNF-α). The monocytes that infiltrate adipose tissue subsequently differentiate into macrophages and become activated [14]. Statins were perceived primarily as cholesterol-lowering agents, but a better understanding of their broader biological effects highlighted mechanisms contributing to an extensive range of benefits that are independent of cholesterol lowering [16]. As a consequence, statins have been proposed for chronic inflammatory diseases and, more recently, for obesity control. During the last decade, numerous studies have found that statins prevent or alleviate obesity-induced inflammation at the tissue, organ, and systemic levels and that such actions are mediated by the modulation of specific cytokines, chemokines, and other inflammatory mediators[12].

Lipotoxicity and Dyslipidemia

The lipid storage capacity of adipose tissue is limited, such that excessive caloric intake and the resultant positive energy balance lead to ectopic fat deposition in non-adipose tissues [13]. The accumulation of harmful lipid metabolites, including metabolites derived from excess ceramide and diacylglycerol (DAG) synthesis, induces hepatic steatosis [7]. In addition, insulin resistance appears to arise from the accumulation of fatty acid-derived lipid intermediates, in particular DAG [15]. Increased delivery of saturated fatty acids (SFAs) to non-adipose tissues (e.g., liver and muscle) augments ceramide and DAG synthesis, which, in turn, perturbs lipid metabolism, promotes sterol regulatory element-binding protein (SREBP-1) activation, and ultimately enhances de novo lipogenesis[11]. Obesity-associated fatty liver also results in excessive production of serum triglyceride-rich very-low-density lipoproteins (VLDLs) and, consequently, atherogenic dyslipidaemia, characterized by raised fasting triglyceride and remnant lipoprotein levels and lowered high-density lipoprotein (HDL) cholesterol. Insulin-sensitizing agents that improve peripheral fat storage while ameliorating hepatosteatosis are expected to exert favourable effects on overall cardio-metabolic risk [15, 8].

Hypertension and Vascular Alterations

Accumulating evidence indicates that obesity is contributing to the increased frequency and severity of hypertension observed in the metabolic syndrome [16]. Hypertension associated with obesity involves volume-overload mechanisms and a large number of vascular alterations. An expansion of the bloodstream occurs through increased blood flow and cardiac output, and the combination of increased plasma volume and cardiac output enhances arterial wall stresses that provoke structural and elastic vascular remodeling [15]. Preclinical studies demonstrate that, during the development of obesity or increased body weight, vascular alterations can be detected before insulin resistance and dyslipidemia occur [10]. Thus, obesity induces the activation of steps in the metabolic syndrome signal pathway that are independent of the insulin-resistant state [16]. In particular, the activation of the renin-angiotensin-aldosterone system contributes to blood-pressure elevation and endothelial dysfunction in resistant forms of obesity [9].

Clinical Consequences and Associated Diseases

Obesity and metabolic syndrome represent major global health challenges. Crude prevalence estimates suggest that, in 2022, approximately 716 million adults aged 18 years and older were living with obesity, defined by a body mass index (BMI) of 30 kg/m2 or higher [13]. This number is projected to exceed 1.5 billion by 2035. Metabolic syndrome, which substantially increases the risk of cardiovascular disease (CVD), type 2 diabetes (T2DM), and other conditions, is also increasing in parallel [15]. In recognition of the interrelationship between the two conditions, the International Federation of Obesity and Non-Communicable Diseases has recently proposed an overweight-and-obesity syndrome. Emerging evidence indicates that obesity influences metabolic syndrome through five interconnected pathophysiological mechanisms: adipose-tissue dysfunction, insulin resistance, inflammation, lipotoxicity, and hypertension. By elucidating these connections and the clinical consequences of obesity and metabolic syndrome, opportunities for preventive and therapeutic interventions can be identified [17]. Obesity and metabolic syndrome represent major global health challenges that have risen steeply in prevalence over several decades [15]. Crude estimates suggest that, in 2022, approximately 716 million adults aged 18 years and older were living with obesity, defined by a body mass index (BMI) of 30 kg/m2 or higher [13]. This estimate is projected to exceed 1.5 billion by 2035. At the same time, metabolic syndrome, defined as a clustering of abdominal obesity, dyslipidemia, hypertension, and glucose intolerance, has been gaining attention [12]. Individuals with metabolic syndrome have an approximately fivefold increased risk of developing cardiovascular disease (CVD) and a similar increase in the risk of developing type 2 diabetes (T2DM), along with elevated risks of fatty liver disease, polycystic ovarian syndrome, cancer, obstructive sleep apnea, and osteoarthritis [13]. A recent survey of international guidelines identified substantial variability in the criteria used to define metabolic syndrome disruption, and individuals may therefore differ in their clinical diagnoses of the condition [11]. In recognition of the interrelationship between Metabolic Syndrome and Obesity, the International Federation of Obesity and Non-Communicable Diseases has recently proposed an Overweight-and-Obesity Syndrome [14]. Emerging research indicates that obesity influences metabolic syndrome through five interconnected pathophysiological mechanisms: adipose-tissue dysfunction, insulin resistance and glucose homeostasis, inflammation and immune-system activation, lipotoxicity and dyslipidemia, and hypertension and vascular alterations [16]. By elucidating these connections and the clinical consequences of obesity and metabolic syndrome, opportunities for preventive and therapeutic interventions can be identified [15].

Therapeutic Approaches and Intervention Strategies

The cornerstone of efforts to combat obesity and metabolic syndrome is the baton of caloric loading. The quantity of energy consumed through macronutrients is represented by the caloric balance equation, and various lifestyle and behavioral modifications can be employed to curb unfavorable energy intake [11]. However, maintaining good adherence to often tedious caloric barrier programs proved difficult over time [10]. Therefore, the potential

for developing one-off chemical solutions has far transcended, leading to in-depth investigations of pharmacological agents that cover meticulous mechanisms of the onset and progression of obesity-related metabolic syndrome, as well as surgical procedures that procure substantial loss of body mass through different pathways[12]. Nevertheless, the physiological electrochemical profiles of different medication modules up until now exhibited the limitations of effectiveness and safety profile, whereas particular compounds enumerated or combinations thereof continue to remain of great interest [12]. Further, big enlightenments on genomics have fortuitously emerged, endorsing the possibility of matched therapeutic regimens specifically designed for each individual according to the genetically determined profile of the metabolic syndrome [13]. The metabolic syndrome incorporates interrelated clusters of cardiovascular risk factors encompassing hypertension, dyslipidemia, type 2 diabetes mellitus (T2DM), and central obesity [13]. The combination of obesity together with periodical unhealthy eating coexists widely nowadays, driving adaptation of visceral fat and progressive structural and functional deterioration of the corresponding metabolically active hepatic differentiative adipocytes. In that, the risk of transition from a clinically resolvable individual to a metabolic syndrome patient rises and becomes challenging to reverse [12]. Consequently, the regimen of health-promoting lifestyles in order to maintain or reduce caloric loading signifies the demand to reverse any cuboidal paracrine influence directly on the surrounding organs governing systemic energy homeostasis and to recover the dysfunctional characteristic back to the basal state, so as to halt the aging pro-resistance status [13]. Thus, glycolipid–normalizing agents having extra-efficaciousness against these transitional pathologies toward the metabolic syndrome remain enormously coveted in societies afflicted deeply by slimming problems [18]. The strategic approach of weight-loss induction through dietary volume reduction, another 10% weight in T2DM-enabling metabolic syndrome candidates, could already potentially ameliorate several detrimental side effects and is still worldwide highly valued [1].

Lifestyle and Behavioral Interventions

The interplay between caloric energy consumption and energy expenditure governs body weight and body composition in the long term [10]. In the case of overweight and obesity, caloric intake exceeds expenditure, resulting in an increase in fat mass and nutrient stores [11]. The World Health Organization (WHO) recommends that the global population engage in at least 150 minutes of moderate-intensity aerobic activity per week [12]. This correlates with the age-adjusted prevalence of obesity by sex. It was noted that the number of gym members increased the most in countries with the highest obesity prevalence. Successful behavioral weight control interventions specify clear, valid, and easily obtainable goals concerning energy consumption and physical exercise [19]. Because adherence to change generally declines over time, earlier intervention is deemed preferable [16]. Other support mechanisms, such as personalized planning that encourages positive feedback, have been developed, possibly based on digital platforms such as SMS, email, or phone [18].

Pharmacological Treatments

Pharmacological treatments for obesity can link to metabolic syndrome but do not directly prevent or lessen its severity [20]. Pharmacotherapy may reduce weight or help maintain weight loss and can be an appropriate adjunct to lifestyle changes when those options fail or when patients have excess weight-related risks [20]. A variety of medications can produce weight loss through differing mechanisms, and the selection of one over another should consider both efficacy and safety [20]. Treatment should complement ongoing lifestyle change. Indications for drug use include metabolic syndrome, type 2 diabetes, and specific obesity-related conditions [21]. Of the weight-reducing medications approved for use in the United States since the 1940s, only three agents remain indicated for long-term treatment of obesity: orlistat, phentermine/topiramate extended-release, and naltrexone/bupropion. Other products under investigation may merit interest [1].

Surgical Options

Bariatric procedures are highly effective for treatment in severe obesity and for second-line treatment in type 2 diabetes [19]. Such interventions generate sustained weight loss and improve metabolic comorbidities via hormonal changes, disruption of inflammatory signaling, and alteration of gut microbiota composition [22]. Long-term outcomes show that gastric bypass patients experience greater weight loss and less comorbidity than controls, highlighting the benefits of surgical intervention in metabolic dysfunction. Metabolic surgery is initiated when patients reach and do not achieve a satisfactory reduction in body weight and comorbidity improvement [24].

Emerging Therapies and Precision Medicine

Comprehensive evidence underscores significant pathophysiological and clinical links between obesity and metabolic syndrome [21]. Population-level intervention strategies addressing both the obesogenic environment and healthcare access to effective prevention, treatment, and management are essential to mitigate concomitant disease risk and improve health equity [23]. The precision medicine framework aids targeted primary prevention of metabolic abnormalities, incorporating factors such as genotype, adipotype, microbiome, and exposome [17]. Further investigation into early-stage biomarkers enables objective staging of metabolic deterioration and

increased lifespan intervention opportunities. Combating obesity and metabolic disease sequelae represents a critical priority [23].

Public Health Implications and Policy Considerations

Obesity and metabolic syndrome exert a considerable impact on health, increasing risks of diabetes, cardiovascular disease, certain cancers, and other serious conditions [20]. These associations lead to substantial morbidity, decreased health-related quality of life, diminished productivity, and, ultimately, excess mortality [1]. The economic burden of obesity and its related diseases imposes a significant drain on health-care expenditure and productivity. Public health interventions aimed at population-level prevention and treatment of obesity and metabolic syndrome can help alleviate these burdens [6]. Obesity prevention is particularly urgent in high-income countries where national obesity rates have stabilized at alarming levels. Yet the conditions of obesity and metabolic syndrome also warrant attention as they represent the most common chronic disease among patients with obesity and expand beyond high-income nations [24]. Temporal trends further illuminate the rising health risks associated with obesity and metabolic syndrome. Several high-level interventions can support population-level prevention and management of obesity and metabolic syndrome. Shaping the surrounding environment is a key strategy [13]. Policies that prevent the production or marketing of calorie-dense foods for infants can minimize the risk of inappropriate early feeding practices that promote obesity [21]. A comprehensive and sustainable approach to the food supply may include making foods containing refined sugars and carbohydrates less accessible and affordable, while promoting the intake of fruit and vegetables. Camera surveillance equipment may be placed in shopping locations to track purchasing behaviours and compliance with policies [25]. Direct government or aimed subsidies that make food shoppers more favourably inclined to obtain dietary products beneficial to obesity or full utilisation of other preventive phenomena reduce obesity as well, such as disabling sugar-wasted energy beverages from vending machines, exempt tax on certain health food instead of refreshment at school, such as wholegrain food, milk, and fruit [24]. Widespread, visible pricing indicates decreased energy products, and promotion in mass media is less effective. Obesity incidence and co-morbidities impose massive economic and social consequences on health care systems [21]. Business excess and company taxes taken will generate a more direct income to support the welfare of society. The rising and explosive trend towards obesity will induce detrimental economic consequences as well. Wide price and subsidy would be attracted to businesses to earn profit and create a win–win situation [26]. Efforts to mitigate or reverse these adverse trends can enhance and increase productivity growth, economic growth, and welfare. Pre-conditional task of providing a convincing argumentation way toward to congregation with stakeholders and market enterprise. Stopping the activity of publishing products that nourish refined sugar among youth appears to be an attractive approach. Monopolistic tendency will occur later in market activity driven by price [24]. Aiming at health promotion and technology development boosts added value in economic activity. Early research stage revolving around this subject concentrates strongly on the business marketing aspect of active health sources. The sustained emphasis of hunger and refreshment suppression contributes towards continuous rather hunk of solid food at frequent intervals [20]. Price subsidy counteracts economics to benefit competitive status, travelling numerous fields including business productivity and reduction of sickness [25-30].

Future Research Directions

Obesity and metabolic syndrome (MetS) represent two key public health challenges of the twenty-first century, with their global prevalence continuously rising and their associated health consequences multiplying [24]. Progress has been made in elucidating their epidemiology, aetiology, shared pathophysiology, and clinical sequelae, but important gaps in knowledge remain. Improved understanding of these disorders and their interconnections, particularly regarding mechanisms linking differences in adipose tissue distribution and ectopic fat storage to their development and clinical manifestations, is urgently needed to inform effective prevention or treatment strategies [25, 26]Tenenbaum & Z Fisman, 2011. Consequently, future research should focus on five priority areas. Improved mechanistic studies of adipose biology are required to assess how changes in regional or ectopic fat stores drive the progression to MetS and related disorders [31-34]. The development of longitudinal cohorts assessing the progression of obesity and MetS, their driver diseases, and the impact of interventions on both, will enable better mechanistic understanding, especially in historically understudied paediatric populations. Additional biomarkers of disease status or driver pathways would facilitate improved identification of individuals at greatest risk of developing MetS and associated conditions [25]. Clinical trials assessing the effects of combination therapies or precision treatment approaches that consider genetic, epigenetic, microbiome, or metabolomic differences can further advance understanding and inform targeted strategies. Finally, implementation science will help determine the extent to which discoveries translate from controlled settings to real-world effectiveness, informing strategies to maximise population health gains [24].

CONCLUSION

Obesity and metabolic syndrome are deeply interconnected public health concerns whose prevalence and associated disease burden continue to increase on a global scale. Evidence presented in this review demonstrates that obesity, particularly central adiposity, is a major driver of the metabolic disturbances characteristic of MetS. The shared pathophysiological mechanisms linking both conditions include adipose-tissue dysfunction, chronic systemic inflammation, impaired insulin signaling, lipotoxicity, and obesity-related vascular changes. These mechanisms form a reinforcing cycle that accelerates the progression of cardiovascular disease, type 2 diabetes, non-alcoholic fatty liver disease, hypertension, reproductive disorders, and other obesity-related comorbidities. Effective management requires a comprehensive approach that integrates lifestyle and behavioral interventions, evidence-based pharmacotherapy, and bariatric or metabolic surgery for individuals with severe disease. Emerging precision-medicine strategies, including genotype-informed treatments, microbiome-targeted therapies, and tailored intervention algorithms, offer promise for personalized prevention and management. On a population level, policy interventions that reshape food environments, promote physical activity, reduce socioeconomic disparities, and ensure equitable access to preventive and treatment services are essential. Future research should deepen mechanistic insights into adipose biology, ectopic fat deposition, and metabolic pathway dysregulation, while also expanding longitudinal cohort studies and improving translation of clinical advances into real-world settings. Ultimately, understanding and addressing the multifaceted relationship between obesity and metabolic syndrome will be central to reducing global morbidity and mortality, enhancing quality of life, and strengthening economic and health-system resilience worldwide.

REFERENCES

1. van Greevenbroek MM, Schalkwijk CG, Stehouwer CD. Dysfunctional adipose tissue and low-grade inflammation in the management of the metabolic syndrome: current practices and future advances. F1000Research. 2016 Oct 13;5:F1000-aculty.
2. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, Gordon DJ, Krauss RM, Savage PJ, Smith Jr SC, Spertus JA. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. Circulation. 2005 Oct 25;112(17):2735-52.
3. Ugwu OP, Ogenyi FC, Ugwu CN, Basajja M, Okon MB. Mitochondrial stress bridge: Could muscle-derived extracellular vesicles be the missing link between sarcopenia, insulin resistance, and chemotherapy-induced cardiotoxicity?. Biomedicine & Pharmacotherapy. 2025 Dec 1;193:118814.
4. Ambroselli D, Masciulli F, Romano E, Catanzaro G, Besharat ZM, Massari MC, Ferretti E, Migliaccio S, Izzo L, Ritieni A, Grosso M. New advances in metabolic syndrome, from prevention to treatment: the role of diet and food. Nutrients. 2023 Jan 26;15(3):640.
5. C. Benyshek D. The Developmental Origins of Obesity and Related Health Disorders – Prenatal and Perinatal Factors. 2007. [PDF]
6. Pillon NJ, Loos RJ, Marshall SM, Zierath JR. Metabolic consequences of obesity and type 2 diabetes: Balancing genes and environment for personalized care. Cell. 2021 Mar 18;184(6):1530-44.
7. Paul-Chima UO, Nneoma UC, Bulhan S. Metabolic immunobridge: Could adipose-derived extracellular vesicles be the missing link between obesity, autoimmunity, and drug-induced hepatotoxicity?. Medical Hypotheses. 2025 Sep 28:111776.
8. Tenenbaum A, Z Fisman E. The metabolic syndrome. is dead: These reports are an exaggeration. 2011. ncbi.nlm.nih.gov
9. Muñoz Contreras AM, Bedoya Berrío G, Velásquez R CM. An approach to the etiology of metabolic syndrome. Colombia Médica. 2013 Jan;44(1):57-63.
10. Ju Jung U, Choi MS. Obesity and Its Metabolic Complications: The Role of Adipokines and the Relationship between Obesity, Inflammation, Insulin Resistance, Dyslipidemia and Nonalcoholic Fatty Liver Disease. 2014. ncbi.nlm.nih.gov
11. Paul-Chima UO, Nnaemeka UM, Nneoma UC. Could dysbiosis of urban air microbiota be an overlooked contributor to pediatric asthma and neurodevelopmental disorders?. Medical Hypotheses. 2025 Sep 12:111758.
12. Lee E, Nam JO. Anti-Obesity and Anti-Diabetic Effects of Ostericum koreanum (Ganghwal) Extract. International Journal of Molecular Sciences. 2024 Apr 30;25(9):4908.
13. P. Czech M. Mechanisms of insulin resistance related to white, beige, and brown adipocytes. 2020. ncbi.nlm.nih.gov
14. Ugwu OP, Ogenyi FC, Ugwu CN, Ugwu MN. Gut microbiota-derived metabolites as early biomarkers for childhood obesity: A policy commentary from urban African populations. Obesity Medicine. 2025 Sep 1;57:100641.

15. Kumar Posa D, P. Baba S. Intracellular pH Regulation of Skeletal Muscle in the Milieu of Insulin Signaling. 2020. ncbi.nlm.nih.gov
16. Pearson T, Wattis JA, King JR, MacDonald IA, Mazzatti DJ. The effects of insulin resistance on individual tissues: an application of a mathematical model of metabolism in humans. Bulletin of mathematical biology. 2016 Jun;78(6):1189-217.
17. Liu R, S. Nikolajczyk B. Tissue Immune Cells Fuel Obesity-Associated Inflammation in Adipose Tissue and Beyond. 2019. ncbi.nlm.nih.gov
18. Ugwu OP, Okon MB, Alum EU, Ugwu CN, Anyanwu EG, Mariam B, Ogenyi FC, Eze VH, Anyanwu CN, Ezeonwumelu JO, Egba SI. Unveiling the therapeutic potential of the gut microbiota–brain axis: Novel insights and clinical applications in neurological disorders. Medicine. 2025 Jul 25;104(30):e43542.
19. Meiliana A, Wijaya A. Macrophage polarization in metabolism and metabolic disease. The Indonesian Biomedical Journal. 2013 Aug 1;5(2):81-90.
20. Klop B, Willem F. Elte J, Castro Cabezas M. Dyslipidemia in Obesity: Mechanisms and Potential Targets. 2013. ncbi.nlm.nih.gov
21. Lobato ND, Filgueira FP, Akamine EH, Tostes RC, Carvalho MH, Fortes ZB. Mechanisms of endothelial dysfunction in obesity-associated hypertension. Brazilian journal of medical and biological research. 2012;45:392-400.
22. M. Perdomo C, Avilés-Olmos I, Dicker D, Frühbeck G. Towards an adiposity-related disease framework for the diagnosis and management of obesities. 2023. ncbi.nlm.nih.gov
23. Mallappa RH, Rokana N, Duary RK, Panwar H, Batish VK, Grover S. Management of metabolic syndrome through probiotic and prebiotic interventions. Indian journal of endocrinology and metabolism. 2012 Jan 1;16(1):20-7.
24. Ugwu CN, Ugwu OP, Alum EU, Eze VH, Basajja M, Ugwu JN, Ogenyi FC, Ejemot-Nwadiaro RI, Okon MB, Egba SI, Uti DE. Medical preparedness for bioterrorism and chemical warfare: A public health integration review. Medicine. 2025 May 2;104(18):e42289.
25. Kaur R. Impact of a Lifestyle Modification Intervention on Health Behaviors and Health Outcomes in a Mexican American population: A Mixed-methods Study. 2018. ⌈PDF⌉
26. Narayanaswami V, Dwoskin LP. Obesity: Current and potential pharmacotherapeutics and targets. Pharmacology & therapeutics. 2017 Feb 1;170:116-47.
27. Ugwu CN, Ugwu OP, Alum EU, Eze VH, Basajja M, Ugwu JN, Ogenyi FC, Ejemot-Nwadiaro RI, Okon MB, Egba SI, Uti DE. Sustainable development goals (SDGs) and resilient healthcare systems: Addressing medicine and public health challenges in conflict zones. Medicine. 2025 Feb 14;104(7):e41535.
28. Bapatla N. An evaluation and comparison of current FDA-approved treatments for obesity. 2018. ⌈PDF⌉
29. Cordero P, Li J, Oben JA. Bariatric surgery as a treatment for metabolic syndrome. Journal of the Royal College of Physicians of Edinburgh. 2017 Dec;47(4):364-8.
30. Hocking S, Sumithran P. Individualised prescription of medications for treatment of obesity in adults. Reviews in Endocrine and Metabolic Disorders. 2023 Oct;24(5):951-60.
31. Rush EC, Yan MR. Evolution not revolution: nutrition and obesity. Nutrients. 2017 May 20;9(5):519.
32. Ongesa TN, Ugwu OP, Ugwu CN, Alum EU, Eze VH, Basajja M, Ugwu JN, Ogenyi FC, Okon MB, Ejemot-Nwadiaro RI. Optimizing emergency response systems in urban health crises: A project management approach to public health preparedness and response. Medicine. 2025 Jan 17;104(3):e41279.
33. Meng Q, Mäkinen VP, Luk H, Yang X. Systems biology approaches and applications in obesity, diabetes, and cardiovascular diseases. Current cardiovascular risk reports. 2013 Feb;7(1):73-83.
34. Sadler JR, Thapaliya G, Ranganath K, Gabay A, Chen L, Smith KR, Osorio RS, Convit A, Carnell S. Paediatric obesity and metabolic syndrome associations with cognition and the brain in youth: Current evidence and future directions. Pediatric obesity. 2023 Aug;18(8):e13042.
35. Isaac Edyedu PMA, Ugwu OPC, Ugwu CN, Alum EU, et al. The role of pharmacological interventions in managing urological complications during pregnancy and childbirth: A review. Medicine. 2025;104(7):e41381.
36. Alum EU, Ugwu OPC, Obeagu EI, et al. Nutritional care in diabetes mellitus: A comprehensive guide. Int J Innov Appl Res. 2023;11(12):16-25.
37. Obeagu EI, Ahmed YA, Obeagu GU, Bunu UO, Ugwu OPC, Alum EU. Biomarkers of breast cancer: Overview. Int J Curr Res Biol Med. 2023;1:8-16.
38. Uti DE, Alum EU, Atangwho IJ, Ugwu OPC, et al. Lipid-based nano-carriers for the delivery of anti-obesity natural compounds: Advances in targeted delivery and precision therapeutics. J Nanobiotechnol. 2025;23:336.

39. Ugwu CN, Ugwu OPC, Alum EU, Eze VHU, Basajja M, Ugwu JN, Ogenyi FC, et al. Medical preparedness for bioterrorism and chemical warfare: A public health integration review. Medicine. 2025;104(18):e42289.
40. Obeagu EI, Scott GY, Amekpor F, Ugwu OPC, Alum EU. COVID-19 infection and diabetes: A current issue. Int J Innov Appl Res. 2023;11(1):25-30.
41. Offor CE, Ugwu OPC, Alum EU. Anti-diabetic effect of ethanol leaf extract of *Allium sativum* on albino rats. Int J Pharm Med Sci. 2014;4(1):1-3.
42. Asogwa FC, Okechukwu PCU, Esther UA, Chinedu OE, Nzubechukwu E. Hygienic and sanitary assessment of street food vendors in selected towns of Enugu North District, Nigeria. Am-Eurasian J Sci Res. 2015;10(1):22-26.
43. Alum EU, Uti DE, Agah VM, Orji OU, Nkeiru N, et al. Physico-chemical and bacteriological analysis of water used for drinking and domestic purposes in Amaozara Ozizza, Afikpo North, Nigeria. Niger J Biochem Mol Biol. 2023;38(1):1-8.
44. Ugwu OPC, Alum EU, Okon MB, Obeagu EI. Mechanisms of microbiota modulation: Implications for health, disease, and therapeutic interventions. Medicine. 2024;103(19):e38088.
45. Ezekwe CI, Uzomba CR, Ugwu OPC. Effect of methanol extract of *Talinum triangulare* on hematology and liver parameters in rats. Glob J Biotechnol Biochem. 2013;8(2):51-60.
46. Alum EU, Inya JE, Ugwu OPC, Obeagu EI, Aloke C, Aja PM, Okpata MG, et al. Ethanolic leaf extract of *Datura stramonium* attenuates methotrexate-induced biochemical alterations in Wistar rats. RPS Pharmacol Rep. 2023;2(1):1-6.
47. Ugwu OPC, Erisa K, Inyangat R, Obeagu EI, et al. Indigenous medicinal plants for managing diabetes in Uganda: Ethnobotanical and pharmacotherapeutic insights. INOSR Exp Sci. 2023;12(2):214-224.
48. Alum EU, Aja W, Ugwu OPC. Vitamin composition of ethanol leaf and seed extracts of *Datura stramonium*. Avicenna J Med Biochem. 2023;11(1):92-97.
49. Ezenwaji CO, Alum EU, Ugwu OPC. Digital health in pandemic preparedness and response: Securing global health? Glob Health Action. 2024;17(1):2419694.
50. Adonu CC, Ugwu OP, Bawa A, Ossai EC, Nwaka AC. Intrinsic blood coagulation studies in patients with diabetes and hypertension. Int J Pharm Med Bio Sci. 2013;2(2):36-45.
51. Offor CE, Ugwu PC, Okechukwu PM, Igwenyi IO. Proximate and phytochemical analyses of *Terminalia catappa* leaves. Eur J Appl Sci. 2015;7(1):9-11.
52. Enechi YS, Ugwu OC, Ugwu KK, Ugwu OPC, Omeh N. Evaluation of antinutrient levels of *Ceiba pentandra* leaves. IJRRPAS. 2013;3(3):394-400.
53. Alum EU, Uti DE, Ugwu OPC, Alum BN, Edeh FO, Ainebyoona C. Microbiota in cancer development and treatment. Discov Oncol. 2025;16(1):646.
54. Asogwa FC, Okoye COB, Ugwu OPC, Edwin N, Alum EU, Egwu CO. Phytochemistry and antimicrobial assay of *Jatropha curcas* extracts. Eur J Appl Sci. 2015;7(1):12-16.
55. Enechi OC, Oluka HI, Ugwu PCO. Acute toxicity and ameliorative properties of *Alstonia boonei* leaf extract on diabetic rats. Afr J Biotechnol. 2014;13(5).
56. Alum EU, Obeagu EI, Ugwu OPC. Enhancing water, sanitation, and hygiene for diarrhoea control and SDGs: A review. Medicine. 2024;103(38):e39578.
57. Odo CE, Nwodo OFC, Joshua PE, Ugwu OPC, Okonkwo CC. Anti-diarrhoeal effect of chloroform–methanol extract of *Persea americana* seeds in rats. J Pharm Res. 2013;6(3):331-335.
58. Ugwu OPC, Obeagu EI, Alum EU, Michael M, et al. Effect of ethanol leaf extract of *Chromolaena odorata* on hepatic markers in diabetic rats. IAA J Appl Sci. 2023;9(1):46-56.
59. Ibiam UA, Alum EU, Orji OU, Aja PM, Nwamaka EN, Ugwu OPC, et al. Anti-inflammatory effects of *Buchholzia coriacea* leaf extract in arthritic rats. Indo Am J Pharm Sci. 2018;5(7):6341-6357.
60. Obeagu EI, Obeagu GU, Odo EO, Alum EU. Nutritional approaches for enhancing immune competence in HIV-positive individuals. IDOSR J Appl Sci. 2024;9(1):40-50.
61. Obeagu EI, Alum EU, Ugwu OPC. Hepcidin: Gatekeeper of iron in malaria resistance. Newport Int J Res Med Sci. 2023;4(2):1-8.
62. Nyamboga TO, Ugwu OPC, Ugwu JN, et al. Biotechnological innovations in soil health management: a systematic review of integrating microbiome engineering, bioinformatics, and sustainable practices. Cogent Food Agric. 2025;11(1):2519811.
63. Madu ANB, Alum EU, Aloh HE, Ugwu OPC, Obeagu EI, Uti DE, Egba SI, Ukaidi CUA. The price of progress: Assessing the financial costs of HIV/AIDS management in East Africa. Medicine. 2025;104(18):e42300.

64. Alum EU, Ugwu OPC. Beyond pregnancy: Understanding long-term implications of gestational diabetes mellitus. INOSR Sci Res. 2024;11(1):63-71.
65. Ugwu OPC, Alum EU, Okon MB, Aja PM, Obeagu EI, Onyeneke EC. Anti-nutritional and GC-MS analysis of ethanol root extract and fractions of *Sphenocentrum jollyanum*. RPS Pharmacol Pharm Rep. 2023;2(2):rqad007.
66. Eze VHU, Eze CE, Mbabazi A, Ugwu CN, Ugwu PO, Ogenyi CF, Ugwu JN, et al. Qualities and characteristics of a good scientific research writing: Step-by-step approaches. IAA J Appl Sci. 2023;9(2):71-76.
67. Igwenyi IO, Nchi PO, Okechukwu UPC, Igwenyi IP, Obasi DC, Edwin N. Nutritional potential of *Azadirachta indica* seeds. Indo Am J Pharm Sci. 2017;4(2):477-482.
68. Enechi OC, Oluka IH, Ugwu OPC, Omeh YS. Effect of ethanol leaf extract of *Alstonia boonei* on lipid profile of alloxan-induced diabetic rats. Afr J Biotechnol. 2013;24.
69. Ugwu OPC. Anti-malaria effect of ethanol extract of *Moringa oleifera* leaves on malaria-induced mice. University of Nigeria Nsukka; 2011:39.
70. Alum EU, Ugwu OPC, Obeagu EI. Nutritional interventions for cervical cancer patients: Beyond conventional therapies. J Cancer Res Cell Ther. 2024;8(1):1-6.
71. Obeagu EI, Obeagu GU. Advancements in immune augmentation strategies for HIV patients. IAA J Biol Sci. 2024;11(1):1-11.
72. Okechukwu PU, Nzubechukwu E, Ogbanshi ME, Ezeani N, Nworie MO. Effect of ethanol leaf extract of *Jatropha curcas* on chloroform-induced hepatotoxicity in albino rats. Glob J Biotech Biochem. 2015;10:11-15.
73. Ilozue NM, Ikezu UP, Okechukwu PCU. Antimicrobial and phytochemical screening of *Persea americana* seed extracts. IOSR J Pharm Biol Sci. 2014;9(2):23-25.
74. Onyeze R, Udeh SM, Akachi B, Ugwu OP. Isolation and characterization of fungi associated with spoilage of corn (*Zea mays*). Int J Pharm Med Biol Sci. 2013;2(3):86-91.
75. Obeagu EI, Alum EU, Ugwu OPC. Hepcidin: The gatekeeper of iron in malaria resistance. Newport Int J Res Med Sci. 2023;4:1-8.
76. Obeagu EI, Alum EU, Obeagu GU, Ugwu OPC. Prostate cancer: Review on risk factors. Eurasian Exp J Public Health. 2023;4(1):4-7.
77. Offor CE, Okaka ANC, Ogbugo SO, Egwu CO, Okechukwu PC. Effects of ethanol leaf extract of *Pterocarpus santalinoides* on haemoglobin, packed cell volume and platelets. IOSR J Nurs Health Sci. 2015;4:108-112, 93.
78. Offor C, Aja PC, Ugwu O, Agbafor KN. Effects of ethanol leaf extract of *Gmelina arborea* on serum proteins in albino rats. Glob J Environ Res. 2015;9(1):1-4.
79. Alum EU, Uti DE, Obeagu EI, Ugwu OPC, Alum BN. Cancer's psychosocial aspects: Impact on patient outcomes. Elite J Med. 2024;2(6):32-42.
80. Alum EU, Ugwu OPC, Egba SI, Uti DE, Alum BN. Climate variability and malaria transmission: Unravelling the complex relationship. INOSR Sci Res. 2024;11(2):16-22.
81. Alum EU, Obeagu EI, Ugwu OPC, Egba SI, EjimUti DE, Ukaidi CUA, et al. Confronting dual challenges: Substance abuse and HIV/AIDS. Elite J HIV. 2024;2(5):1-8.

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