International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	OBESITY AND TYPE 2 DIABETES: THE ROLE OF LIFESTYLE, BIOLOGICAL MECHANISMS, AND SOCIO-TECHNOLOGICAL DETERMINANTS
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5650
RECEIVED	27 February 2026
ACCEPTED	04 May 2026
PUBLISHED	13 May 2026
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OBESITY AND TYPE 2 DIABETES: THE ROLE OF LIFESTYLE, BIOLOGICAL MECHANISMS, AND SOCIO-TECHNOLOGICAL DETERMINANTS

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ABSTRACT

Objective: The aim of this study is to provide a comprehensive overview of the relationship between obesity and type 2 diabetes (T2D) and to identify the key environmental, behavioral, and biological factors involved in their development. A review of current scientific literature concerning the epidemiology, pathophysiology, and risk factors of both conditions was conducted, with particular emphasis on the roles of diet, physical activity, gut microbiota, and socio-economic determinants.
Analysis: The analysis demonstrated that obesity, particularly visceral adiposity, constitutes the primary risk factor for type 2 diabetes. The central mechanism linking these conditions is insulin resistance, which is exacerbated by chronic low-grade inflammation and adipose tissue dysfunction. Significant importance is attributed to unfavorable dietary patterns, including the high consumption of ultra-processed foods and high-glycemic-load diets, both of which promote metabolic disturbances. Furthermore, the role of gut microbiota in metabolic regulation was highlighted, alongside the impact of physical activity – specifically mechanisms involving GLUT4 translocation and Non-Exercise Activity Thermogenesis (NEAT) on improving glucose homeostasis. Additionally, environmental and socio-economic factors, such as urbanization, food accessibility, and educational attainment, significantly modulate disease risk.
Conclusions: The findings indicate that obesity and type 2 diabetes share a common, multifactorial etiology. Consequently, effective prevention and management require an integrated approach encompassing lifestyle modifications, systemic interventions, and the utilization of modern health-supporting technologies.

KEYWORDS

Obesity, Type 2 Diabetes, Diet, Physical Activity, Insulin Resistance, Public Health

CITATION

Julia Mądrzak, Klaudia Jurkowska, Olga Endler, Mikołaj Dybicz, Marianna Ciastoń, Magdalena Filuk, Jakub Fidelus, Dominika Dutkiewicz, Julia Czerniewska, Marta Handzel. (2026) Obesity and Type 2 Diabetes: The Role of Lifestyle, Biological Mechanisms, and Socio-Technological Determinants. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5650

1. Introduction

Obesity and type 2 diabetes (T2D) represent some of the most significant public health challenges of the 21st century. Their rising prevalence poses a substantial burden on healthcare systems globally. These conditions are intrinsically linked at both the epidemiological and pathophysiological levels. Excess body mass, particularly visceral adiposity, serves as a primary risk factor for the development of T2D and plays a pivotal role in its initiation and progression.

The mechanisms connecting obesity to glucose metabolism disorders are multifaceted, involving the development of insulin resistance, chronic low-grade inflammation, and the dysfunction of adipose tissue as an active endocrine organ. Furthermore, dysregulated secretion of adipokines such as leptin and adiponectin, which modulate metabolism, appetite, and insulin sensitivity plays a critical role. Consequently, a metabolic vicious cycle emerges, wherein obesity and glycemic impairment exacerbate one another.

Despite an expanding body of research, there remains a critical need to integrate biological mechanisms with the influence of environmental, socio-economic, and technological factors that determine the development and clinical course of these diseases. Modern advancements, such as mHealth technologies, telemedicine, and wearable devices, are increasingly significant in supporting both prevention and therapeutic management.

This review aims to provide a comprehensive analysis of the relationship between obesity and type 2 diabetes, with particular focus on pathophysiological mechanisms, the role of adipose tissue, and the impact of socio-economic and technological determinants on disease risk and therapeutic options. Given the complex, multilevel nature of these conditions, effective prevention and management require an integrated approach encompassing lifestyle modifications, environmental initiatives, and systemic interventions within the framework of public health policy.

2. Literature Review Methodology

To systematize current knowledge, a narrative literature review was conducted regarding the impact of diet and physical activity on the development of obesity and type 2 diabetes. The objective of this analysis was to provide a synthetic overview of the contemporary state of research, identify key pathophysiological mechanisms, and highlight existing research gaps.

A literature search was performed across electronic databases, including PubMed and Google Scholar. A total of 106 scientific publications were identified and analyzed. The analysis included articles in both English and Polish, focusing primarily on works published between 2020 and 2025. Selected classic earlier studies were also included to ensure a comprehensive perspective while maintaining a focus on current research trends in metabolic diseases.

The search strategy was based on a combination of keywords such as: obesity, type 2 diabetes, physical activity, diet, insulin resistance, glycemic index, microbiome, and sedentary behavior. These terms were combined using Boolean operators (AND, OR) to enhance search precision.

Inclusion criteria consisted of: original research papers, systematic reviews, and meta-analyses, human-based studies, and where justified experimental animal models, provided they pertained to the pathophysiological mechanisms associated with obesity and type 2 diabetes. Publications focusing on the correlation between diet and/or physical activity and the development of metabolic diseases were prioritized. Exclusion criteria involved non-full-text papers, publications with low methodological quality, and articles not directly relevant to the scope of this review.

This study is a narrative literature review. Its aim is to provide a synthetic and critical presentation of the current state of knowledge, identify the primary mechanisms linking lifestyle to metabolic diseases, and suggest potential directions for future research.

3. Obesity as a Public Health Challenge

Obesity has emerged as one of the most critical global public health challenges of the 21st century. It is no longer viewed merely as an aesthetic concern but as a chronic disease (ICD-10 code: E66), which serves as a foundation for over 200 health complications.

From a public health perspective, obesity generates significant economic burdens, encompassing both direct costs (related to medical treatment) and indirect costs, such as reduced productivity and increased absenteeism. Rapid urbanization and the progressive digitalization of daily life have fostered the creation of an "obesogenic environment," characterized by ubiquitous access to high-calorie foods and a systematic decline in daily physical activity.

3.1 Definition and BMI Criteria

Obesity is a complex, multifactorial disease defined as an excessive accumulation of adipose tissue in the body, resulting from a prolonged imbalance between energy intake and expenditure (Faccioli et al., 2023). According to the World Health Organization (WHO) definition, obesity is a chronic, relapsing disease that significantly increases the risk of developing numerous metabolic complications and organ-specific disorders (World Health Organization, 2025; Ahmed & Mohammed, 2025).

The fundamental mechanism leading to the development of obesity is a positive energy balance, resulting from excessive caloric intake relative to expenditure (World Health Organization, 2025; Zheng et al., 2018). Modern civilizational changes, including rapid urbanization and globalization, promote the reinforcement of unfavorable dietary patterns and the reduction of physical activity, further exacerbating the scale of the problem (Ahmed & Mohammed, 2025).

The etiology of obesity is multifaceted and encompasses individual factors, such as genetic predispositions and established health behaviors, as well as environmental and social determinants. The latter include, among others, improper eating habits, the influence of the cultural environment, and limited access to healthy food a phenomenon referred to as "food deserts" (Williams et al., 2015; Shaharir et al., 2014). Despite the significance of biological factors, the dominant role of lifestyle, particularly excessive energy supply and insufficient physical activity, is emphasized in the development of obesity (Safaei et al., 2021; Zheng et al., 2018). Due to the increasing prevalence of this disease and the need for its standardized assessment in clinical practice and population studies, simple diagnostic methods have been developed for its classification.

The diagnosis of overweight and obesity is most commonly based on measurements of body weight and height and the calculation of the Body Mass Index (BMI). BMI is the most widely used tool for assessing the

nutritional status of a population. It is a simple method based on the relationship between body weight and height, developed by Adolphe Quetelet in the 19th century.

BMI is calculated using the formula: body weight (kg) divided by the square of height (m^2).

According to the World Health Organization (WHO) criteria, the BMI classification is as follows (World Health Organization, 2025):

Table 1. Classification of Body Mass Index (BMI) according to WHO criteria.

BMI Category	BMI (kg/m²)
Underweight	<18.50
Severely Underweight	<16.00
Moderately/Mildly Underweight	16.00–18.49
Normal weight	18.50–24.99
Overweight (Pre-obese)	25.00–29.99
Obese	≥30.00
Class I (Moderate)	30.00–34.99
Class II (Severe)	35.00–39.99
Class III (Morbid/Very Severe)	≥40.00

BMI is widely applied in both clinical practice and population-based research. However, it has significant limitations, as it does not account for body composition, specifically the ratio between adipose tissue and muscle mass nor the distribution of fat tissue. This is particularly important in the case of visceral obesity, which is associated with a higher metabolic risk (Dominguez et al., 2023).

Despite these limitations, BMI remains a useful screening tool, especially in epidemiological studies. Its simplicity, low cost, and ease of application make it universally utilized for assessing nutritional status and monitoring health trends within the population (Wu et al., 2024; Dominguez et al., 2023).

3.2 Epidemiology

Obesity, along with its associated comorbidities, represents one of the most critical health challenges in the modern world. The literature emphasizes that excessive body mass is a significant risk factor for premature mortality and is currently ranked among the leading causes of death globally (Safaei et al., 2021).

The scale of the obesity crisis is systematically increasing across both adult and pediatric populations. Data from the World Health Organization (WHO) indicate that between 1990 and 2022, the prevalence of obesity among children and adolescents aged 5–19 quadrupled, rising from 2% to 8%. During the same period, the proportion of adults living with obesity more than doubled, increasing from 7% to 16% (World Health Organization, 2025).

In 2022, overweight (BMI 25.00-29.99) affected approximately 2.5 billion adults (≥18 years), of whom nearly 890 million met the criteria for obesity (BMI ≥30.0). This issue also extends to the pediatric population; in 2024, it was estimated that approximately 35 million children under the age of 5 were overweight (World Health Organization, 2025).

Epidemiological projections indicate a further intensification of this phenomenon. It is estimated that by 2050, the number of adults with overweight or obesity could reach 3.8 billion, accounting for more than half of the global adult population (Ahmed & Mohammed, 2025).

The rapid increase in obesity prevalence and its global reach have led to this phenomenon being increasingly referred to as the "epidemic of the 21st century."

3.3 Risk Factors

The development of obesity results from a complex interaction of multiple factors, including behavioral, biological, and environmental aspects. Contemporary lifestyle changes promote a positive energy balance, leading to weight gain.

Diet and Physical Inactivity

One of the primary risk factors for obesity is an improper diet, characterized by a high proportion of processed foods and sugar-sweetened beverages. Excessive energy intake, coupled with a simultaneous reduction in energy expenditure, leads to the accumulation of adipose tissue.

A low level of physical activity constitutes another significant risk factor. Limited activity reduces the body's total energy expenditure, which facilitates a positive energy balance and weight gain (Thivel et al., 2012).

Genetic and Metabolic Factors

Genetic, endocrine, and metabolic factors play a crucial role in weight regulation and the predisposition to obesity (Gasmi et al., 2020; Lin et al., 2021). It is estimated that up to 70% of body weight variance may be linked to genetic factors (Loos et al., 2021).

One of the most well-studied genetic factors is the FTO gene, which is associated with an increased risk of developing obesity. Its presence may influence appetite regulation, leading to increased caloric intake and reduced feelings of satiety (Melhorn et al., 2018). Individuals possessing specific variants of this gene exhibit a greater tendency to gain weight during both childhood and adulthood (Ahmed & Mohammed, 2025).

Metabolic disorders, such as insulin resistance, also play a vital role by promoting fat storage, particularly within the abdominal cavity (Ahmed & Mohammed, 2025).

Lifestyle and Socioeconomic Factors

The modern lifestyle is characterized by a significant reduction in physical activity and an increase in sedentary time. Environmental factors, such as prolonged sedentary work, limited access to recreational infrastructure, and the widespread use of motorized transport, further exacerbate this issue (Moschonis & Trakman, 2023; Woessner et al., 2021; Ahmed & Mohammed, 2025).

Socioeconomic determinants are also of significant importance. Numerous studies indicate an inverse relationship between socioeconomic status and obesity prevalence individuals with lower income and education levels more frequently struggle with excessive body weight (Autret & Bekelman, 2024). Limited access to healthy food and the predominance of inexpensive, highly processed products contribute to the reinforcement of poor dietary habits (Ahmed & Mohammed, 2025).

Additionally, psychological factors, such as stress and emotional tension, can lead to "emotional eating" which involves consuming high-calorie products regardless of actual hunger (Ahmed & Mohammed, 2025).

Research also indicates that the most critical behavioral factors promoting obesity development include physical inactivity, poor diet, and excessive consumption of sugar-sweetened beverages, combined with a low intake of vegetables and fruits (Choukem et al., 2020; Sun et al., 2020).

All the risk factors described above are synthetically presented in the figure below, illustrating their interconnections and the multifactorial nature of obesity development.

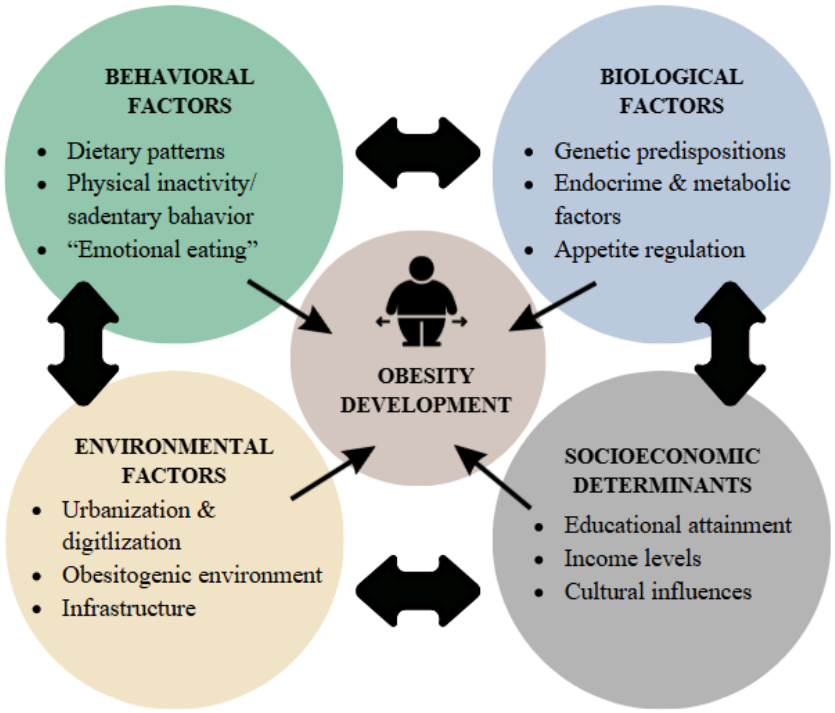

Fig. 1. *Major risk factors contributing to obesity development*

3.4 Health Consequences of Obesity

Obesity is associated with numerous health complications, encompassing both metabolic disturbances and systemic diseases. The literature emphasizes that excessive body mass is a major risk factor for the development of many chronic conditions, including type 2 diabetes, kidney disease, cardiomyopathy, neurodegenerative disorders, and certain autoimmune diseases.

Abdominal obesity is a particularly adverse phenotype, showing a strong correlation with an increased risk of metabolic and cardiovascular diseases, including hypertension, dyslipidemia, asthma, and various types of cancer (Hu et al., 2017; Young et al., 2017).

Metabolic Consequences

The development of type 2 diabetes is among the most significant metabolic consequences of obesity. Excessive accumulation of adipose tissue promotes insulin resistance, which impairs glucose uptake by cells, subsequently leading to elevated blood glucose concentrations (Ahmed & Mohammed, 2025).. Obesity also frequently co-occurs with non-alcoholic fatty liver disease (NAFLD) and dyslipidemia, further exacerbating the risk of metabolic complications (Ahmed & Mohammed, 2025)..

Cardiovascular Consequences

Obesity has a profound impact on the cardiovascular system. One of the earliest and most frequently observed sequelae is hypertension, resulting, among other factors, from increased blood volume and elevated cardiac output (Welsh et al., 2024; Mahesh et al., 2024; Ahmed & Mohammed, 2025).

Long-term strain on the circulatory system may lead to the development of heart failure and impairment of both systolic and diastolic myocardial function. Consequently, individuals with obesity exhibit an increased risk of severe cardiovascular events, such as myocardial infarction and stroke (Ahmed & Mohammed, 2025).

Metabolic Syndrome

Metabolic syndrome constitutes one of the most critical sequelae of obesity and is strongly associated with an increased risk of cardiovascular disease. It is characterized by the clustering of visceral obesity, hypertension, and lipid disturbances (Ahmed & Mohammed, 2025).

Other Health Consequences

Beyond metabolic and cardiovascular disorders, obesity is associated with a range of other health complications. These include osteoarthritis and chronic pain, sleep apnea, fertility disorders, and dermatological issues, such as skin changes within skin folds (Ahmed & Mohammed, 2025). Furthermore, psychological consequences, including an increased risk of depression and reduced quality of life, represent a significant aspect of the condition (Ahmed & Mohammed, 2025).

3.5 Social and Economic Impact

Obesity represents not only a significant health issue but also a substantial social and economic challenge. Its consequences manifest at both individual and systemic levels, generating high costs and necessitating multifaceted intervention strategies (Cerceo et al., 2023).

Economic Costs

Excess body mass is associated with a significant increase in healthcare expenditures and negatively affects the economic status of individuals by reducing both income levels and employment probability (Biener et al., 2018). Healthcare systems worldwide are facing escalating costs related to the treatment of obesity-related comorbidities, including type 2 diabetes, cardiovascular diseases, and various cancers.

The economic burden of obesity encompasses both direct costs (e.g., medical treatment, hospitalization) and indirect costs resulting from reduced productivity, absenteeism, and disability. It is estimated that by 2035, the global costs associated with overweight and obesity could reach $4.32 trillion annually (Mahase, 2023; Ahmed & Mohammed, 2025). Furthermore, analyses conducted in the United States indicate that as early as 2017, losses related to diminished productivity and lower household incomes amounted to approximately $1.4 trillion (Lopez et al., 2020; Tulane University School of Social Work, 2018).

Burden on Healthcare Systems

The rising prevalence of obesity leads to an increased burden on healthcare systems. In the United States, the proportion of medical expenditures allocated to the treatment of obesity-related diseases in adults rose from 6.13% in 2001 to 7.91% in 2015, representing a 29% increase (Biener et al., 2018). This growth reflects the increasing demand for medical services, including the management of chronic conditions, surgical interventions, and long-term care for patients with obesity-related complications.

Impact on Quality of Life

Obesity exerts a profound influence on quality of life and psychosocial functioning. Individuals with obesity often experience weight-related stigma and discrimination, which can lead to severe psychological consequences (Puhl et al., 2020; Mambrini et al., 2023). Negative social stereotypes foster prejudice within the workplace, educational settings, and healthcare environments, which in turn is associated with lowered self-esteem, an increased risk of depression, and social isolation (Hajek et al., 2021).

Moreover, the experience of stigmatization can paradoxically exacerbate the obesity problem; individuals subjected to discrimination are more likely to avoid physical activity or employ maladaptive coping strategies, such as emotional eating (Ahmed & Mohammed, 2025). Children and adolescents constitute a particularly vulnerable group, for whom obesity may lead to social exclusion, experiences of peer bullying, and persistent body image disorders that often persist into adulthood (Ahmed & Mohammed, 2025).

Obesity represents a complex medical, social, and economic challenge. Its growing scale may lead to the overburdening of healthcare systems and negatively impact economic development unless effective preventive and systemic measures are implemented (Ahmed & Mohammed, 2025).

4. The Role of Diet in the Development of Obesity and Diabetes

Unfavorable dietary patterns are among the key factors determining the development of obesity and type 2 diabetes. A diet rich in saturated fats, simple sugars, and high-energy products, combined with a low intake of plant-based foods, promotes a positive energy balance and metabolic disturbances. In conjunction with low levels of physical activity, these factors are currently recognized as the primary determinants of the development and progression of obesity and type 2 diabetes (Ezzati & Riboli, 2013).

4.1 High-Calorie Diet and UPF

Ultra-processed foods (UPF) encompass products subjected to intensive technological processes, often containing numerous additives such as preservatives, emulsifiers, colorants, and flavor enhancers. These products are characterized by high energy density and a high content of simple sugars, saturated fats, and salt, while possessing low nutritional value.

Epidemiological data indicate a significant positive correlation between the consumption of ultra-processed foods and body mass index (BMI), as well as the risk of overweight and obesity (Valicente et al., 2023). Cohort studies confirm that higher UPF intake is associated with greater energy intake and higher BMI values (Chen et al., 2023).

Increasing evidence also points to the biological mechanisms underlying these relationships. Components of UPF may adversely affect gut microbiota, exacerbate chronic low-grade inflammation, and

promote the development of insulin resistance, which increases the risk of cardiometabolic diseases, including type 2 diabetes (Chen et al., 2023; Hall, 2019, Srour et al., 2022).

Results from prospective studies indicate that total UPF consumption is significantly associated with an increased risk of developing type 2 diabetes. The quality of scientific evidence supporting this relationship has been rated as high (NutriGrade: 8/10), underscoring the importance of this factor in the pathogenesis of metabolic diseases (Chen et al., 2023).

Meta-analyses and systematic reviews further confirm that increased UPF consumption is linked to an increased risk of weight gain, overweight, and obesity (Dicken & Batterham, 2024). Prospective studies have demonstrated a positive correlation between UPF intake and both general and abdominal obesity (Pagliai et al., 2020). Similar observations apply to the pediatric population, where higher consumption of ultra-processed food is associated with an increased risk of overweight, obesity, and an elevated waist circumference (Chen et al., 2023; Petridi et al., 2023).

Significant evidence is also provided by interventional studies. In a randomized crossover trial, it was demonstrated that a diet based on UPF led to a mean increase in energy intake of approximately 500 kcal per day, resulting in weight gain (~0.9 kg), whereas a diet based on minimally processed products led to weight reduction (Zakim, 1972).

4.2 Glycemic Index (GI)

The Glycemic Index (GI) is a tool used to classify carbohydrate-containing products based on their impact on postprandial glycemia. The GI value determines the rate and extent of the rise in blood glucose levels compared to a reference product, such as pure glucose or white bread (Sievenpiper et al., 2018).

Based on their GI values, food products are divided into three categories: low (≤55), medium (56-69), and high (≥70) glycemic index, allowing for the assessment of their potential impact on glucose metabolism (Sievenpiper et al., 2018).

The Glycemic Load (GL) complements the GI by accounting for both the quality and the quantity of carbohydrates consumed. It is calculated as the product of the GI and the amount of available carbohydrates per serving (in grams), divided by 100 (Thomas & Wolever, 2006). This indicator is considered more practical for assessing the real-world impact of diet on glycemia.

Available scientific evidence indicates that a low-GI and low-GL diet can yield beneficial metabolic effects. It has been associated with improved glycemic control, a favorable lipid profile, weight reduction, lower blood pressure, and decreased systemic inflammation (Chiavaroli et al., 2021).

These benefits are particularly observed in individuals with type 1 and type 2 diabetes, for whom a low-GI/GL diet can serve as a significant adjunct to pharmacological treatment, supporting metabolic control of the disease (Chiavaroli et al., 2021).

4.3 The Role of the Microbiome

The gut microbiota is increasingly recognized as a functional "metabolic organ" that plays a vital role in regulating the body's energy homeostasis and nutrient metabolism. It is estimated that the gut microbiota can weigh approximately 1 kg, underscoring its biological significance (Geng et al., 2022).

Contemporary research indicates that the gut microbiome participates in the pathogenesis of obesity and type 2 diabetes (T2D) by influencing glucose metabolism, adipose tissue function, and inflammatory processes (Chatelier et al., 2013; Stanislawski et al., 2019). A critical factor modulating microbiota composition is diet, which can lead to both beneficial shifts and microbial imbalances, referred to as dysbiosis (Stanislawski et al., 2019)

Biological Mechanisms

The gut microbiota influences host metabolism through several mechanisms. Among the most important is the production of short-chain fatty acids (SCFAs), such as acetate, propionate, and butyrate, which regulate energy homeostasis, insulin sensitivity, and inflammatory responses (Markowiak-Kopeć & Śliżewska, 2020).

Dysbiosis can lead to increased intestinal barrier permeability and the translocation of lipopolysaccharides (LPS) into the systemic circulation. This process promotes the development of chronic low-grade inflammation, a key mechanism driving insulin resistance and obesity (Acevedo-Román et al., 2024). Furthermore, the microbiota can affect the host's ability to harvest energy from the diet, increasing the efficiency of calorie extraction and promoting a positive energy balance (Geng et al., 2022).

Microbiota Alterations in Obesity

Under physiological conditions, the gut microbiota is characterized by high diversity and stability; disturbances in this balance may facilitate the development of metabolic diseases (Shanahan, 2013). In individuals with obesity, alterations in microbiota composition are observed, including reduced alpha-diversity and shifts in species composition (de La Serre et al., 2010; Million et al., 2011; Chen et al., 2015)

While earlier studies suggested an increased Firmicutes-to-Bacteroidetes ratio in obese individuals, it is now emphasized that this relationship is not unequivocal and may depend on various factors, including diet, lifestyle, and microbiome analysis methodologies. Significant changes also occur at the level of specific bacterial species. Higher concentrations of Lactobacillus reuteri have been associated with obesity, whereas Bifidobacterium animalis and Methanobrevibacter smithii are more frequently found in individuals with a normal body weight (Million et al., 2011).

Association with Type 2 Diabetes

Gut microbiota disturbances may serve as an early risk marker for type 2 diabetes and influence glucose metabolism. Specific microbial profiles have been linked to insulin resistance and systemic inflammation. Certain bacteria, such as Bacteroides faecalis, may promote the development of metabolic disorders by affecting glucose regulation and inflammatory pathways (Wei et al., 2021; Brunkwall & Orho-Melander, 2017).

Clinical Significance

Given the critical role of the microbiota in metabolic regulation, it represents a potential target for therapeutic interventions. Modifying microbiome composition through diet, probiotics, or prebiotics may play an important role in the future prevention and treatment of obesity and type 2 diabetes.

4.4. Dietary Patterns

Dietary patterns, understood as a holistic approach to nutrition, play a crucial role in regulating metabolism and tissue sensitivity to hormones. An increasing body of research suggests that it is not only individual dietary components but also their mutual proportions and interactions that determine the risk of developing metabolic diseases.

Energy restriction exerts a beneficial effect on physiological functioning, contributing not only to weight reduction but also to the improvement of metabolic parameters and hormonal regulation (Mazza et al., 2024).

Health-Promoting Patterns

Dietary patterns based on high consumption of vegetables, fruits, and plant-based products are associated with a lower risk of developing obesity (Ledoux et al., 2010). The benefits of the Mediterranean diet, characterized by a high intake of unsaturated fatty acids, dietary fiber, and bioactive compounds, are particularly well-documented. Its adherence is linked to a reduced risk of both obesity and type 2 diabetes.

Meta-analyses of randomized controlled trials (RCTs) have demonstrated that the Mediterranean diet leads to greater reductions in body weight and BMI compared to other nutritional models, while cohort studies indicate a decreased risk of obesity and weight gain (Dominguez et al., 2023).

The mechanisms responsible for these beneficial effects include improved insulin sensitivity, reduction of chronic inflammation, a favorable impact on the gut microbiota, and lower dietary energy density (Dominguez et al., 2023). Additionally, high dietary fiber content promotes appetite regulation and the stabilization of postprandial glycemia.

The Western Dietary Pattern

Conversely, the so-called Western diet characterized by high intake of ultra-processed foods, including fast food, fried dishes, processed meats, and sugar-sweetened beverages is associated with an increased risk of overweight and metabolic disturbances (Pao Ying Hsiao et al., 2011).

This diet facilitates a positive energy balance, exacerbates insulin resistance, increases systemic inflammation, and disrupts the gut microbiota. The high energy density and low nutritional value of these products lead to excessive caloric intake and the impairment of satiety regulation mechanisms.

Excessive intake of simple sugars, particularly fructose, can further disrupt hepatic metabolism, promoting insulin resistance and leading to increased synthesis of very-low-density lipoproteins (VLDL), hypertriglyceridemia, as well as heightened oxidative stress and low-grade inflammation (Mambrini et al., 2023). Furthermore, ultra-processed foods serve as a significant source of trans and saturated fatty acids, contributing to the development of dyslipidemia.

Significance for Type 2 Diabetes

Dietary patterns play a vital role not only in the development of obesity but also in type 2 diabetes. A high-quality diet based on plant-based and minimally processed products is associated with a lower risk of disease onset and better glycemic control in those already diagnosed (Dominguez et al., 2023).

In contrast, dietary patterns based on ultra-processed foods and a high glycemic load promote the development of insulin resistance and glucose metabolism disorders. Results from prospective studies indicate that high consumption of ultra-processed foods is associated with an increased risk of obesity, hypertension, type 2 diabetes, and dyslipidemia (Mambrini et al., 2023).

Analysis of dietary patterns indicates that the overall dietary model is of greater importance for metabolic health than individual nutrients. Consequently, prevention strategies for obesity and type 2 diabetes should focus on promoting healthy dietary patterns rather than the exclusive elimination of single dietary components.

5. Physical Activity, Body Weight, and Metabolism

5.1 Physical Activity and Sedentary Lifestyle

Insufficient physical activity represents one of the key global public health challenges. It affects approximately 27.5% of the global population, with higher rates observed in high-income countries (42.3%). Crucially, no significant improvement in these figures has been recorded in recent years (Guthold et al., 2018; World Health Organization, 2020).

A sedentary lifestyle and an insufficient level of moderate-to-vigorous physical activity (MVPA) constitute independent risk factors for cardiometabolic disorders. Their co-occurrence leads to an increased risk of developing type 2 diabetes, cardiovascular diseases, and premature mortality (Chen et al., 2024).

Increasing evidence also indicates that prolonged periods of sedentary behavior represent a distinct risk factor, independent of the total level of physical activity (Owen et al., 2014; Manson et al., 2004). Consequently, physical inactivity has been recognized as one of the leading modifiable risk factors for mortality worldwide, holding a high position in the global classification of risk factors (World Health Organization, 2020).

At the same time, a growing number of studies indicate that regular physical activity can partially compensate for the negative effects of a sedentary lifestyle. It has been demonstrated that meeting physical activity guidelines (at least 150 minutes of moderate-to-vigorous activity per week) is associated with a reduction in mortality risk, even among individuals leading a sedentary lifestyle (Zhu et al., 2025). This suggests that physical activity and sedentary behavior should be treated as related yet distinct constructs in the analysis of metabolic health.

5.2. Molecular Mechanisms: The Role of GLUT4

One of the key mechanisms responsible for the beneficial impact of physical activity on glucose metabolism is the regulation of glucose transporter type 4 (GLUT4). This protein plays a fundamental role in glucose uptake by skeletal muscles, both in response to insulin and muscle contraction induced by physical exertion (Zheng et al., 2018).

During physical activity, there is an increased translocation of GLUT4 from intracellular compartments to the cell membrane (sarcolemma) and T-tubules, enabling more efficient glucose transport into muscle cells. Furthermore, regular physical exercise leads to an increase in the total expression of the GLUT4 protein, which further enhances the muscles' capacity for glucose utilization (Zheng et al., 2018; Williams et al., 2015).

This mechanism is of particular significance in the context of insulin resistance and type 2 diabetes. In individuals with impaired insulin sensitivity, the GLUT4 response to insulin is diminished; however, the activation of this transporter in response to muscle contraction remains largely preserved (Christ-Roberts et al., 2004; Hussey Roberts et al., 2012). This means that physical activity provides an alternative, insulin-independent pathway for increasing glucose uptake, which is essential for improving glycemic control and metabolic flexibility (Williams et al., 2015).

5.3. NEAT (Non-Exercise Activity Thermogenesis)

Non-Exercise Activity Thermogenesis (NEAT) is a significant component of total energy expenditure and plays a crucial role in regulating the body's energy balance. It encompasses all forms of spontaneous physical activity not related to planned exercise, such as walking, gesturing, changing posture, or performing daily chores.

NEAT is defined as any bodily movement generated by skeletal muscles that results in energy expenditure exceeding resting levels (typically above 1.6 METs) (Rizzato et al., 2022). Unlike structured physical activity, NEAT is often subconscious and can vary significantly between individuals.

Research indicates that NEAT levels can exhibit extreme variability up to approximately 2000 kcal per day between individuals of similar body mass and composition (Charan et al., 2023). In individuals leading a sedentary lifestyle, this value is considerably lower, usually not exceeding 700 kcal per day, which facilitates a chronic positive energy balance and the development of obesity (Rizzato et al., 2022).

Increasing NEAT levels can be a vital element in the prevention and treatment of obesity, particularly for those with limited structured physical activity. It has been shown that higher levels of spontaneous activity can counteract weight gain by increasing total daily energy expenditure (Loeffelholz & Birkenfeld, 2018).

Importantly, the significance of NEAT extends beyond simple energy balance regulation. An increasing number of studies suggest that higher levels of daily, low-intensity activity can beneficially influence insulin sensitivity, glucose metabolism, and lipid profiles, which is of particular importance in the prevention of type 2 diabetes.

Furthermore, unlike organized forms of physical activity, increasing NEAT does not require the implementation of structured training programs, making it more accessible and potentially a more sustainable component of lifestyle modification. In a population context, this can be significantly meaningful, especially in environments that promote sedentary behavior.

5.4 Urbanization and Environmental Factors

Progressive urbanization and civilizational development significantly shape population lifestyles, influencing physical activity levels, dietary patterns, and exposure to environmental factors. These changes facilitate the adoption of pro-obesogenic behaviors, such as sedentary lifestyles, increased consumption of ultra-processed foods, and chronic stress (Chandrasekaran & Weiskirchen, 2024).

The urbanized environment, specifically its infrastructure and spatial organization, plays a key role in shaping daily health choices. Research indicates that the availability of grocery stores offering fresh products promotes healthier dietary choices, whereas a high concentration of fast-food outlets is associated with greater consumption of energy-dense, nutrient-poor foods (Bernsdorf et al., 2017; Burgoine et al., 2016; Moore et al., 2008).

The so-called "built environment," which includes access to spaces conducive to physical activity such as parks, cycling paths, and green areas also plays a vital role. The presence of these areas shows a positive correlation with physical activity levels within the population, underscoring the importance of urban planning in obesity prevention (Cohen et al., 2007).

High levels of urbanization are also linked to changes in the structure of daily activity. In highly developed countries, lower levels of physical activity and more hours spent in sedentary positions are observed, which are closely related to the nature of modern work and a dependence on motorized transport. Conversely, populations residing in rural areas, where daily functioning often involves greater physical exertion (e.g., agricultural work, commuting by foot or bicycle), are characterized by higher activity levels (Ezzati & Riboli, 2013).

Dependence on car transport and limited access to infrastructure promoting physical activity further exacerbate the problem of hypokinesia in urban environments. Consequently, urbanization represents a significant environmental factor influencing the development of obesity and metabolic diseases, including type 2 diabetes.

5.5 WHO Recommendations

According to the recommendations of the World Health Organization (WHO), regular physical activity is a cornerstone in the prevention of metabolic diseases, including obesity and type 2 diabetes. To achieve significant health benefits, adults should engage in at least 150–300 minutes of moderate-intensity aerobic physical activity per week, or 75-150 minutes of high-intensity activity. Alternatively, an equivalent combination of both intensities is recommended (World Health Organization, 2020).

These guidelines also emphasize the necessity of limiting sedentary time and increasing overall daily activity levels, which includes spontaneous physical activity (NEAT). Adhering to these recommendations is associated with improved weight management, increased insulin sensitivity, and a reduced risk of cardiovascular and metabolic diseases.

However, it must be noted that despite clearly defined recommendations, a significant portion of the population fails to reach the suggested physical activity levels. This is due to various factors, including environmental elements such as urbanization, limited access to supportive infrastructure, and the nature of the modern lifestyle, which promotes sedentary behavior.

Consequently, the effective implementation of WHO recommendations requires a multilevel approach, encompassing not only individual lifestyle changes but also environmental interventions and public health policies. This underscores the need to integrate actions aimed at increasing both structured physical activity and daily energy expenditure, including NEAT, as complementary components in the prevention of obesity and type 2 diabetes.

6. The Link Between Obesity and Type 2 Diabetes

Obesity is recognized as one of the most significant risk factors for the development of type 2 diabetes (T2D) and represents a key element of its pathogenesis (Chandrasekaran & Weiskirchen, 2024). It is estimated that over 80% of individuals with T2D are overweight or obese, which underscores the strong association between these conditions (García-Molina et al., 2020).

The link between obesity and T2D is based on complex and interconnected pathophysiological mechanisms, among which insulin resistance plays a central role. Excessive body mass, particularly in the form of visceral obesity, leads to increased release of free fatty acids and their ectopic deposition in organs such as the liver and muscles, which impairs insulin signaling pathways.

A common denominator for both conditions is chronic, low-grade inflammation, insulin resistance, lipid accumulation in non-adipose tissues, and progressive dysfunction of pancreatic β-cells (Artasensi et al., 2023). Excessive body mass plays a significant role in both the initiation and progression of T2D, regardless of the patient's age (Chandrasekaran & Weiskirchen, 2024).

Type 2 diabetes is a chronic metabolic disease with a complex etiology, characterized by persistent hyperglycemia resulting from impaired insulin secretion, impaired insulin action, or both (Chandrasekaran & Weiskirchen, 2024). During the course of the disease, reduced insulin secretion by β-cells and exacerbated insulin resistance in peripheral tissues are observed, leading to increased levels of free fatty acids, intensified lipolysis, and enhanced glucose production in the liver (Acevedo-Román et al., 2024).

Obesity further exacerbates glucose homeostasis disturbances by reducing glucose uptake in tissues, including muscles and the brain. These disturbances can affect central mechanisms regulating appetite and satiety, which facilitates a further positive energy balance. Consequently, a metabolic "vicious cycle" is created, where obesity worsens insulin resistance and hyperglycemia, while disturbances in glucose-lipid metabolism promote further weight gain.

Therefore, dietary interventions, including the restriction of carbohydrate intake, can support improved glycemic control and weight reduction, thereby positively influencing the course of the disease (Magkos et al., 2020; Chandrasekaran & Weiskirchen, 2024).

6.1 Insulin Resistance

Insulin resistance (IR) is a key mechanism linking obesity to the development of type 2 diabetes. It is a state in which peripheral tissues such as skeletal muscle, the liver, and adipose tissue exhibit reduced sensitivity to insulin action, leading to glucose metabolism disturbances (Artasensi et al., 2023).

IR plays a central role in the pathophysiology of type 2 diabetes and often develops long before the onset of overt clinical symptoms. In the initial stages, the body compensates for the diminished effectiveness of insulin by increasing its secretion, which leads to hyperinsulinemia. However, as the disorder progresses, there

is a gradual exhaustion of pancreatic β-cell function, resulting in the transition from a pre-diabetic state to full-blown type 2 diabetes (Artasensi et al., 2023).

Obesity, particularly when associated with an excess of visceral adipose tissue, promotes the development of insulin resistance by disrupting lipid metabolism and increasing the release of free fatty acids (FFAs). This leads to their ectopic deposition in the liver and muscles, which impairs insulin signaling pathways, limits glucose uptake in peripheral tissues, and exacerbates hepatic gluconeogenesis (Klein et al., 2022).

At the cellular level, inflammatory processes and oxidative stress also play a significant role. Consequently, insulin resistance is not only a key element in the pathogenesis of type 2 diabetes but also the primary mechanism mediating the relationship between excessive body mass and the development of metabolic disorders. Its presence precedes the clinical manifestation of the disease, making it a critical target for preventive and therapeutic interventions.

6.2 Chronic Low-Grade Inflammation

Chronic low-grade inflammation is one of the key mechanisms linking obesity to the development of insulin resistance and type 2 diabetes. This process directly contributes to impaired insulin signaling, leading to the exacerbation of insulin resistance in peripheral tissues. Obesity-related inflammation promotes metabolic disturbances, including reduced glucose uptake by muscles, increased hepatic glucose production, and the gradual dysfunction of pancreatic β-cells. Additionally, it leads to the impairment of the endocrine function of adipose tissue (Chandrasekaran & Weiskirchen, 2024).

Pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α), interleukins (IL-1β, IL-6), and monocyte chemoattractant protein-1 (MCP-1), play a critical role in the pathogenesis of metabolic inflammation. These substances are secreted by both adipocytes and activated macrophages present within the adipose tissue. Their action affects multiple organs, including the liver, skeletal muscles, and the pancreas, where they contribute to insulin signaling interference, impaired glucose metabolism, and the progression of insulin resistance (Chandrasekaran & Weiskirchen, 2024).

At the molecular level, pro-inflammatory cytokines activate signaling pathways such as NF-κB and JNK, which inhibit proper insulin signal transduction and increase tissue resistance to insulin.

Consequently, chronic inflammation creates a positive feedback loop in which obesity exacerbates insulin resistance, and metabolic disturbances further promote the activation of inflammatory processes. This cycle plays a crucial role in the transition from obesity to full-blown type 2 diabetes, facilitating the consolidation of metabolic disorders.

6.3 Visceral Adipose Tissue

Visceral adipose tissue (VAT) is currently viewed not merely as an energy reservoir but as a metabolically active organ that plays a pivotal role in the pathogenesis of type 2 diabetes. Unlike subcutaneous adipose tissue, visceral fat exhibits a stronger association with metabolic disturbances, including insulin resistance and pancreatic β-cell dysfunction. It has been demonstrated that both an increase in Body Mass Index (BMI) and the accumulation of adipose tissue within the abdominal cavity are linearly correlated with an increased risk of developing type 2 diabetes (Chandrasekaran & Weiskirchen, 2024).

The accumulation of fat within internal organs, such as the liver or pancreas, further exacerbates this risk. Individuals with higher amounts of visceral and ectopic fat are characterized by a higher probability of developing metabolic disorders compared to those with lower accumulation levels (Manolopoulos et al., 2010).

Under conditions of obesity, adipose tissue undergoes significant structural and functional changes. These include adipocyte hypertrophy, ectopic lipid deposition, and the development of visceral obesity, all of which promote cardiometabolic disorders (Iacobini et al., 2019). Simultaneously, an increased infiltration of macrophages into the adipose tissue is observed, which participates in the development of insulin resistance (Wu & Ballantyne, 2020). Changes in their polarization reflect the immune response disturbances accompanying metabolic syndrome (Artasensi et al., 2023).

Adipose tissue dysfunction is also associated with an altered adipokine secretion profile. In obesity, there is an increase in the concentration of pro-inflammatory factors such as TNF-α, IL-1β, IL-6, IL-8, and leptin, alongside a simultaneous reduction in adiponectin and other anti-inflammatory factors. These imbalances promote chronic inflammation and exacerbate insulin resistance (Lumeng et al., 2007; Xu et al., 2013; Artasensi et al., 2023).

Excess adipose tissue can initiate the inflammatory process through various mechanisms, including ectopic lipid deposition leading to lipotoxicity and impaired adipokine secretion. These factors negatively affect glucose homeostasis and lipid metabolism, contributing to the development of type 2 diabetes (Whitehead et al., 2006). Furthermore, dysfunctional adipose tissue can lead to structural changes such as fibrosis, hypoxia, and mitochondrial dysfunction, further deepening metabolic disturbances (Artasensi et al., 2023).

Consequently, visceral adipose tissue constitutes the central link connecting excessive body mass with chronic inflammation and insulin resistance, playing a crucial role in the pathogenesis of type 2 diabetes.

6.4 Adipokines (Leptin, Adiponectin)

In addition to its energy storage function, adipose tissue plays a vital endocrine role by secreting numerous biologically active molecules known as adipokines. These molecules regulate metabolic processes, inflammatory responses, and the functioning of various organs, including the liver, skeletal muscles, the pancreas, and the central nervous system (Chandrasekaran & Weiskirchen, 2024; Liu et al., 2022).

Adipokines can be classified into protective factors (e.g., adiponectin, FGF-21, CTRP9) and pro-inflammatory factors (e.g., leptin, resistin, chemerin, IL-6). The disruption of the balance between these groups plays a pivotal role in the development of obesity and its metabolic complications (Kumar et al., 2022).

Leptin is a hormone primarily produced by adipocytes, and its circulating concentration correlates with the amount of stored adipose tissue. Under physiological conditions, it regulates energy balance by acting on the central nervous system to reduce appetite and increase energy expenditure (Bays, 2004).

However, individuals with obesity often exhibit leptin resistance, a phenomenon characterized by an impaired biological response to the hormone despite elevated blood levels. This results in disrupted appetite regulation, increased energy intake, and the maintenance of a positive energy balance (Bays, 2004). Furthermore, leptin exerts pro-inflammatory and immunomodulatory effects, enhancing the activation of immune cells and contributing to chronic inflammation (Liu et al., 2022). It has also been demonstrated that factors such as chronic stress can modulate leptin levels and facilitate the development of leptin resistance (Kumar et al., 2022; Chao et al., 2017).

Adiponectin is a protective adipokine with anti-inflammatory properties that increases tissue insulin sensitivity and enhances fatty acid oxidation (Whitehead et al., 2006). In the liver, it limits glucose production, while in skeletal muscles, it promotes lipid utilization, partly through the activation of AMP-activated protein kinase (AMPK) (Chandrasekaran & Weiskirchen, 2024).

Unlike most adipokines, adiponectin concentration is inversely proportional to the amount of adipose tissue. In individuals with obesity and type 2 diabetes, its levels are reduced, which promotes the development of insulin resistance and chronic inflammation (Whitehead et al., 2006). Additionally, adiponectin participates in the regulation of mitochondrial function and muscle energy homeostasis (Kim et al., 2025).

Disruptions in adipokine secretion, resulting from excessive lipid storage and adipocyte dysfunction, lead to a predominance of pro-inflammatory signals over protective ones. Consequently, this exacerbates insulin resistance, consolidates chronic inflammation, and impairs metabolic control.

In conclusion, the dysregulation of the adipokine axis represents a significant mechanism linking excess adipose tissue to the development of type 2 diabetes.

7. Technological and Socio-Economic Perspective

7.1. mHealth and Wearables

The development of digital technologies in healthcare, including mHealth (mobile health) solutions and wearable devices, represents a promising direction for supporting the prevention and treatment of obesity and type 2 diabetes. These technologies increase the accessibility of dietary interventions and lifestyle modification programs while streamlining their implementation and monitoring (Spinean et al., 2024).

mHealth applications allow for the real-time monitoring of health behaviors and provide access to personalized support and education. Their functionality includes logging food intake, analyzing physical activity, tracking body weight, monitoring blood glucose or fluid intake, and providing real-time feedback (Istepanian & Al-Anzi, 2018). This enables users to better identify unfavorable habits and make more informed nutritional decisions.

The literature emphasizes that dietary apps are widely available and frequently used as tools to assist in diet and physical activity control (Fakih El Khoury et al., 2019). Particular importance is attributed to features such as goal setting, progress tracking, and reminders, which strengthen motivation and facilitate the maintenance of healthy behavior changes (Spinean et al., 2024).

Available research indicates that mHealth-based interventions can contribute to weight reduction (Sakane et al., 2023) and improve adherence to dietary recommendations, especially in the case of personalized interventions (Allman-Farinelli & Chen, 2017). With the increasing ubiquity of smartphones, these technologies may play an ever-greater role in the care model for patients with chronic diseases, positively impacting health outcomes and quality of life (Merck, 2017).

Wearable technologies, such as smartwatches and fitness trackers, serve as an essential complement to mobile applications. They enable continuous tracking of health parameters, including physical activity levels, heart rate, and sleep patterns (Charan et al., 2023). This data can be utilized for both diagnostic and therapeutic purposes, supporting the individualization of health interventions (Lewczak & Mitchell, 2024; Xie et al., 2021).

Of particular importance is sleep monitoring, as sleep plays a vital role in metabolic and weight regulation. Sleep disorders are linked to the development of obesity and metabolic disturbances; their identification and modification can be a crucial component of therapeutic interventions.

These solutions are further complemented by telemedicine, defined as the use of telecommunications technologies to provide healthcare services remotely. This includes video consultations, remote monitoring of health parameters, and integration with mobile apps (Ezeamii, 2024).

The application of telemedicine in the treatment of obesity and type 2 diabetes facilitates continuous dietary and psychological support, increasing the accessibility of care and reducing geographical and organizational barriers (Ma, et al., 2022). Telemedicine interventions can enhance patient engagement in the treatment process and improve clinical indicators, such as glycated hemoglobin (HbA1c) levels (Ezeamii, 2024). Research also indicates that glycemic telemonitoring can lead to a significant reduction in HbA1c values in diabetic patients (Hu, et al., 2020). Furthermore, regular remote monitoring and consultations have been shown to improve the effectiveness of treatment for other chronic conditions, such as hypertension (Ma, et al., 2022).

In conclusion, the integration of mobile technologies, wearable devices, and telemedicine creates a new healthcare model based on continuous monitoring, personalized interventions, and active patient engagement in the therapeutic process.

7.2. Social Determinants of Health

Socio-economic factors play a crucial role in shaping health behaviors and the risk of developing obesity and type 2 diabetes. In many cases, health education alone proves insufficient because individual decisions are strongly conditioned by the social, economic, and spatial environment, which can either promote or restrict the adoption of healthy choices.

A significant phenomenon in this context includes "food deserts" – areas with limited access to fresh, wholesome food, particularly fruits and vegetables. Simultaneously, researchers describe the concept of "food swamps," characterized by a high concentration of outlets offering ultra-processed, energy-dense, and nutrient-poor food. In such environments, individuals are more likely to make suboptimal dietary decisions.

Research indicates that elements of the built environment significantly influence diet quality and physical activity levels. Greater access to supermarkets is associated with better dietary quality, while a high concentration of fast-food restaurants correlates with increased consumption of low-nutritional-value foods (Bernsdorf, et al., 2017; Burgoine, et al., 2016; Moore, et al., 2008). Conversely, the availability of recreational areas fosters physical activity (Cohen, et al., 2007).

Economic conditions also remain a significant factor influencing dietary choices. Research suggests that food price may have a greater impact on obesity risk than the physical availability of products (Carlson & Frazão 2014). Cheaper products are often more caloric and less nutritious, which encourages excessive energy intake.

Psychosocial factors, including chronic stress-particularly job-related stress are also highly significant. Stress affects cognitive functions, including self-regulation, which can lead to less favorable dietary decisions and increased consumption of high-calorie products (Kumar, et al., 2022). These mechanisms are further modulated by hormonal changes involving leptin, ghrelin, and neuropeptide Y (Kumar, et al., 2022).

Cortisol plays a key role in the body's stress response, a glucocorticoid synthesized in the adrenal cortex that participates in regulating metabolism and homeostasis (Erceg, et al., 2025). Long-term elevated cortisol levels promote increased glucose production, insulin resistance, and the redistribution of adipose tissue, particularly within the abdominal area (Spinean, et al., 2024; Kamin & Kertes, 2017). Furthermore, chronic activation of the stress axis affects the brain's reward system, increasing susceptibility to emotional eating and promoting a positive energy balance (Lewczak & Mitchell, 2024; Chao et al., 2017).

Another important determinant is the level of education and health literacy. Individuals with higher education levels typically exhibit better diet quality and more pro-health dietary habits Kuczmarski, 2016).

Conversely, lower education levels are associated with more frequent consumption of ultra-processed food and higher usage of fast-food outlets (Miura et al., 2011; Miura & Turrell, 2014). These disparities translate into a higher risk of overweight and obesity (Public Health England, 2014). These correlations can vary by gender, as some populations show a stronger link between socio-economic status and Body Mass Index (BMI) in women than in men (Miura & Turrell, 2014).

The significance of the socio-economic environment is confirmed by observations indicating that improving living conditions e.g., moving to a more privileged neighborhood can be associated with favorable changes in health indicators, including lower BMI (Tulane University School of Social Work, 2018)

In summary, socio-economic determinants constitute a key element in the pathogenesis of obesity, influencing both individual behaviors and the surrounding living environment. These factors intensify the biological mechanisms described in previous sections, contributing to the development of metabolic disorders. The complexity of these relationships suggests that effective obesity prevention requires not only individual-level interventions but also systemic actions and appropriately targeted public health policies.

8. Results

The analysis of the available literature demonstrates a consistent and robust association between obesity and the development of type 2 diabetes (T2D), supported by both epidemiological and mechanistic evidence.

A key finding across studies is the high prevalence of excess body weight among individuals with T2D, with more than 80% of patients presenting with overweight or obesity. This relationship is particularly pronounced in cases of visceral obesity, which shows a strong correlation with metabolic disturbances.

The reviewed studies consistently identify insulin resistance as the central mechanism linking obesity to impaired glucose metabolism. Increased levels of circulating free fatty acids, resulting from excessive adipose tissue accumulation, lead to ectopic fat deposition in the liver and skeletal muscles. This process disrupts insulin signaling pathways, reduces glucose uptake, and enhances hepatic glucose production.

In addition, a chronic low-grade inflammatory state is frequently reported in obese individuals. Elevated levels of pro-inflammatory cytokines, including TNF-α, IL-6, and MCP-1, contribute to the impairment of insulin sensitivity and the progressive dysfunction of pancreatic β-cells.

The role of adipose tissue as an active endocrine organ is also strongly supported. Studies indicate a shift in adipokine secretion patterns, characterized by increased leptin levels and decreased adiponectin concentrations. This imbalance promotes inflammation, insulin resistance, and metabolic dysregulation.

Furthermore, the literature highlights the significant contribution of visceral adipose tissue to disease progression. Compared to subcutaneous fat, visceral fat exhibits higher metabolic activity and a greater capacity to induce inflammatory and endocrine disturbances. A structured summary of the key pathophysiological mechanisms linking obesity and T2D is presented in Table 2.

Table 2. Summary of key pathophysiological links between obesity and type 2 diabetes.

Factor	Change in Obesity	Impact on Metabolism
Visceral Fat	Increase (↑)	Direct link to portal circulation and liver IR
Free Fatty Acids	Increase (↑)	Ectopic deposition in muscle and liver
Adiponectin	Decrease (↓)	Reduced insulin sensitivity and fatty acid oxidation
TNF-α / IL-6	Increase (↑)	Disruption of insulin receptor signaling
GLUT4 Activity	Decrease (↓)	Impaired glucose uptake in skeletal muscle

Finally, emerging evidence suggests that socio-economic factors and environmental conditions, including limited access to healthy foods and reduced opportunities for physical activity, contribute to the increasing prevalence of obesity and T2D. Emerging evidence also highlights the potential of technological interventions, such as mHealth and wearable devices, in improving lifestyle behaviors and supporting disease management.

9. Discussion

The findings of this review confirm a strong and multidimensional relationship between obesity and the development of type 2 diabetes (T2D), driven by a complex interplay of metabolic, inflammatory, and hormonal mechanisms.

Key mechanistic insights indicate that three interrelated processes play a central role in this relationship: insulin resistance, chronic low-grade inflammation, and adipose tissue dysfunction. Insulin resistance emerges as the primary mediator, developing as a consequence of lipid metabolism disturbances, ectopic fat accumulation, and impaired insulin signaling. These processes are further exacerbated by adipokine imbalance, characterized by elevated leptin levels accompanied by leptin resistance, and reduced concentrations of adiponectin, a protective anti-inflammatory factor.

A pivotal role is attributed to visceral adipose tissue (VAT), which, due to its anatomical location and high metabolic activity, exerts a more pronounced pathogenic effect than subcutaneous fat. VAT promotes the secretion of pro-inflammatory cytokines such as TNF-α and IL-6, leading to disruption of insulin signaling pathways and progressive β-cell dysfunction. These findings support the contemporary view of obesity as a systemic, multifactorial disease rather than a simple consequence of excessive energy intake.

Importantly, the progression of obesity and T2D is not determined solely by biological mechanisms but is intensified by a "perfect storm" of socio-economic and environmental factors. Limited access to healthy foods (food deserts), high availability of energy-dense processed products (food swamps), and built environments that discourage physical activity significantly shape health behaviors. Additionally, chronic stress, mediated by cortisol, contributes to visceral fat accumulation and promotes maladaptive eating patterns, further reinforcing metabolic dysregulation.

In this context, emerging digital health technologies, including mHealth applications, wearable devices, and telemedicine, represent a promising avenue for intervention. These tools enable continuous monitoring of lifestyle behaviors, glucose levels, and physiological parameters, facilitating personalized and more effective management of chronic metabolic conditions. Their integration into healthcare systems may significantly enhance patient engagement and long-term treatment outcomes.

10. Conclusions

Obesity remains one of the most significant modifiable risk factors for type 2 diabetes, exerting its effects through a multifaceted network involving insulin resistance, chronic inflammation, and adipose tissue dysfunction. In particular, visceral adiposity and the associated imbalance in adipokine secretion play a decisive role in the deterioration of metabolic homeostasis.

The evidence highlights the necessity of adopting a comprehensive and integrated approach to prevention and treatment. Effective strategies should combine:

1. Individual lifestyle modifications, including dietary changes, increased physical activity, and enhancement of non-exercise activity thermogenesis (NEAT)
2. Systemic public health interventions addressing environmental and socio-economic determinants
3. The implementation of digital health technologies to support monitoring, adherence, and patient engagement.

Future research directions should focus on further elucidating the molecular mechanisms linking obesity to T2D, identifying novel biomarkers and therapeutic targets (e.g., myokines, gut microbiota-derived metabolites), and evaluating the long-term effectiveness of digital health interventions. Additionally, greater emphasis should be placed on understanding how socio-economic factors influence the success of prevention strategies, in order to develop more equitable and effective public health policies.

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