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OBESITY IN CHILDREN – EPIDEMIOLOGY, CAUSES, AND HEALTH CONSEQUENCES - A LITERATURE REVIEW

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ABSTRACT

Introduction: Childhood obesity is one of the most serious challenges facing public health today. Its prevalence is steadily increasing worldwide, affecting both developed and developing countries. This disease is chronic, progressive, and multifactorial. Its development is the result of complex interactions between genetic, environmental, behavioral, psychosocial, and neurohormonal factors. The main causes include poor eating habits, low levels of physical activity, a sedentary lifestyle, socioeconomic factors, and the influence of the family environment. Obesity that develops in childhood often persists into adulthood, leading to a significantly increased risk of premature onset of chronic diseases, reduced quality of life, and shortened life expectancy.

Aim of the study: The aim of this study was to present a comprehensive review of the problem of obesity in children and adolescents by analyzing current epidemiological data, etiology, health complications, and contemporary treatment strategies.

Material and methods: This paper is a review of the literature covering current experimental, observational, and randomized controlled trials on childhood obesity. Data on the prevalence of the disease, risk factors, metabolic, cardiovascular, bone and psychosocial disorders, as well as the effectiveness of non-pharmacological interventions, pharmacotherapy and surgical treatment were analyzed.

Results and conclusions: A review of the literature indicates that the prevalence of obesity among children and adolescents is steadily increasing, posing a significant clinical and social problem. Childhood obesity is associated with an increased risk of developing insulin resistance, type 2 diabetes, dyslipidemia, hypertension, and premature atherosclerotic changes, which can lead to cardiovascular disease in adulthood. Excessive body weight also negatively affects the musculoskeletal system and is associated with psychosocial consequences such as low self-esteem, anxiety, and depression. Effective treatment requires early diagnosis and an individually tailored, interdisciplinary approach based primarily on lifestyle modification, including healthy eating habits, increased physical activity, and behavioral interventions involving the whole family. Long-term and comprehensive therapeutic management is crucial to reducing the risk of complications later in life.

KEYWORDS

Childhood Obesity, Overweight, Epidemiology, Risk Factors, Metabolic Complications, Prevention, Treatment

CITATION

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Introduction

The problem of obesity among children and adolescents is growing and represents one of the key challenges to public health on a global scale. Epidemiological data indicate that the prevalence of these disorders varies between countries and regions of the world, but in the vast majority of cases, a steady increase in their prevalence is observed [1]. Between 2011 and 2020, there was a marked increase in the prevalence of obesity in children and adolescents aged 2 to 19 years. The highest prevalence was observed in the 2–5 and 12–19 age groups, among both girls and boys. However, no significant changes in the prevalence of obesity were found in the group of children aged 6–11 [2].

The prevalence of obesity varies significantly between ethnic groups. This condition is more common among African American and Hispanic children than among white and Asian children of non-Hispanic origin [3]. These differences are even more pronounced in the case of severe obesity, which affects 12.8% of African American children and 12.4% of Hispanic children, compared to only 5.0% of white children of non-Hispanic origin [4]. Obesity is a distinct disease entity, and its treatment is particularly difficult in the case of comorbidities such as diabetes, cardiovascular disease, or mental disorders. Numerous studies also indicate that excessive weight and obesity developing in childhood often persist into adulthood. Their consequences are not limited to health problems at a young age, but are associated with an increased risk of early onset of chronic diseases [1,4,5,6], such as type 2 diabetes, cardiovascular disease, hypertension, lipid disorders, metabolic fatty liver disease, obstructive sleep apnea, musculoskeletal disorders, and mental disorders. In addition, overweight individuals are at increased risk of developing certain cancers, including breast cancer, endometrial cancer, and gastrointestinal cancers [5,6]. Obesity is also recognized as a significant contributor to premature puberty in children and adolescents [5]. Despite widespread international awareness that childhood obesity is a serious public health problem, no region of the world has yet managed to effectively curb its growth. Current projections based on simulation models indicate a further increase in its prevalence as well as up to a threefold rise in the risk of obesity in adulthood over the next 5–10 years [7].

Epidemiology

Over the past few decades, there has been an unprecedented increase in the prevalence of overweight children and adolescents worldwide. Studies published between 1975 and 2016 show that, after standardization for age, the prevalence of obesity in the population aged 5–19 has increased many times over. Among girls, the prevalence has increased from less than 1% to over 5%, while among boys it has increased from around 1% to almost 8%. Since the beginning of the 21st century, high-income countries have seen a slowdown in the further increase in average BMI values among children, which have reached a relatively stable, albeit still alarmingly high, level. However, a different trend can be seen in low- and middle-income countries, where body mass indices in the pediatric population continue to rise steadily. In 2016, a particularly high prevalence of childhood obesity – exceeding 30% – was reported in numerous island countries in the Pacific region. Values exceeding 20% were also found in selected countries in the Middle East and North Africa, as well as in Micronesia and Polynesia, the Caribbean, and the United States [6].

The diagnosis of overweight and obesity in the pediatric population is based on the assessment of body mass index relative to the child's age and gender. The World Health Organization (WHO) uses standard deviations (SD) of BMI relative to the population median for this purpose. In children under 5 years of age, according to WHO standards from 2006, overweight is defined as a BMI at least 1 SD above the median, while obesity is defined as a value equal to or exceeding 2 SD. The same diagnostic thresholds apply to children and adolescents aged 5–19 years according to WHO references from 2007. In the United States, however, CDC percentile charts are commonly used, in which overweight corresponds to a BMI between the 85th and 95th percentiles, and obesity to values equal to or higher than the 95th percentile [6,8].

The latest data from 2024 indicate that globally, approximately 35 million children under the age of 5 were overweight. This problem, which for a long time mainly affected highly industrialized countries, is also increasingly observed in lower-income regions. On the African continent, the number of overweight children in the youngest age group has increased by more than 12% since 2000. At the same time, almost half of all children under the age of 5 who were overweight or obese in 2024 lived in Asian countries.

In 2022, more than 390 million children and adolescents aged 5 to 19 were overweight. The percentage of overweight or obese people in this age group increased from about 8% in the early 1990s to 20% in 2022, with a comparable rate of increase among boys and girls. During this period, 21% of boys and 19% of girls were found to be overweight. The prevalence of obesity alone increased even more dramatically. In 1990, it affected about 2% of children and adolescents aged 5–19, which was about 31 million people. By 2022, this percentage had quadrupled, reaching 8%, which corresponded to more than 160 million children and young people worldwide [8].

Etiology of obesity in children

Excessive weight gain in children is most often the result of a prolonged positive energy balance, in which the amount of energy supplied exceeds the amount expended. The result of this process is a gradual increase in body weight and excessive accumulation of adipose tissue. This phenomenon is not caused by a single factor — it is the interaction of many behaviors and various conditions that promote obesity, often coexisting and mutually reinforcing each other's effects [4]. The most commonly observed behavioral factors include regular consumption of sweetened beverages and highly processed foods, which are high in calories and low in nutritional value, as well as insufficient physical activity, a predominantly sedentary lifestyle, reduced sleep time, and circadian rhythm disorders [3,4]. Abnormal sleep patterns promote weight gain by disrupting the secretion of appetite-regulating hormones such as leptin, ghrelin, and peptide YY, as well as by negatively affecting food choices and physical activity levels [3].

The first two years of life are a particularly critical stage for the development of obesity risk. During this time, both genetic factors, which determine metabolic rate, appetite regulation, and hormonal axis function, and rare forms of monogenic obesity resulting from mutations in genes involved in energy balance control are important. Equally important are perinatal and early developmental factors, including cesarean delivery, prematurity, high birth weight, and rapid weight gain in the first months of life, which increase the likelihood of obesity in later years. [9]. An important factor in reducing the risk of excess weight is how the infant is fed. Shorter breastfeeding duration is associated with a higher risk of developing overweight, while prolonged breastfeeding has a protective effect [3], promoting proper appetite regulation, favorable development of the gut microbiota, and influencing epigenetic mechanisms. Conversely, excessive protein intake in the first years of life, especially from cow's milk or high-protein milk substitutes, can lead to accelerated growth and increased fat deposition [9].

The way In Ih an Infant's diet Is expanded also plays a huge role, especially when complementary foods are rich in simple sugars and fats [3]. The risk of obesity is also influenced by low nutritional knowledge among caregivers, unfavorable socioeconomic conditions, and the reinforcement of poor eating habits [10]. A growing body of research indicates that early exposure to antibiotics may increase the predisposition to obesity by disrupting the composition and function of the gut microbiota. In addition, excessive maternal weight may promote the transfer of a microbiome that promotes obesity to the child, and this risk is modified by the mode of delivery and the woman's diet during pregnancy and lactation [9,10]. The development of obesity is also influenced by factors related to the growth and health of the child. Children with growth retardation have a reduced ability to oxidize fatty acids, which can lead to excessive weight gain after nutritional improvement. Damage to the hypothalamic structures, for example as a result of craniocerebral injuries or central nervous system tumors, can result in the development of hypothalamic obesity, characterized by increased appetite, behavioral disorders, and often the need for long-term hospitalization. Importance is also attributed to environmental factors, including exposure to chemicals with obesity-promoting potential, such as phthalates or bisphenol A, contact with tobacco smoke, and air pollution in early life. These factors may influence adipocyte differentiation and the regulation of metabolic processes through epigenetic mechanisms [11].

Psychosocial and behavioral factors are also important elements in the etiology of obesity. Self-regulation disorders, specific temperament traits, excessive screen time, and intense exposure to advertisements for high-calorie foods contribute to the development of unhealthy eating behaviors. Paradoxically, restrictive dietary control strategies used by caregivers can lead to episodes of compensatory consumption of previously restricted foods [9]. Children growing up in lower socioeconomic status environments are particularly vulnerable to developing obesity, which is associated with chronic stress, limited access to high-quality food, lack of safe places for physical activity, and less social support. Negative experiences in early childhood and family instability can contribute to emotional eating and reduced physical activity as early as preschool age [12].

The genetic basis of obesity is complex and multifactorial, and its expression is closely related to environmental, behavioral, and epigenetic factors [9,13]. The development of genome-wide association studies (GWAS) [13] and next-generation sequencing techniques (NGS) [14] has enabled the identification of numerous genetic loci associated with a predisposition to obesity, including both rare monogenic forms and more common polygenic forms. Scientists estimate that approximately 127 regions of the human genome are significantly associated with the development of this disease [13].

Neuroendocrine mechanisms play a central role in the regulation of appetite and energy balance. Ghrelin acts as an orexigenic hormone, increasing the feeling of hunger, while leptin, produced mainly by white adipose tissue adipocytes, affects the hypothalamus by stimulating anorexigenic POMC neurons and inhibiting orexigenic

NPY/AgRP neurons. Leptin is involved not only in the control of food intake, but also in the regulation of glucose and lipid metabolism, immune response, hematopoiesis, neuroprotective processes, and reproductive functions. Its congenital deficiency leads to severe hyperphagia, massive obesity, metabolic disorders, and hypogonadism, while treatment with exogenous leptin allows for effective normalization of these disorders in people affected by this rare defect [14]. Endocrine disorders are a relatively rare cause of obesity, accounting for approximately 2–3% of cases, and are usually accompanied by growth retardation. Untreated or poorly controlled hypothyroidism, hypercortisolism in Cushing's syndrome, and growth hormone deficiency can contribute to the development of excess body weight. A special form is hypothalamic obesity, which develops as a result of damage to the hypothalamic structures following trauma, neurosurgery, radiotherapy, inflammatory processes, tumor infiltration, or congenital genetic disorders affecting this area [15].

Complications

Insulin resistance

Insulin resistance (IR) is a metabolic condition characterized by a weakened response of tissues to insulin, despite normal or elevated blood insulin levels, resulting in limited glucose transport into cells [16]. Insulin, as the only hormone that lowers glucose levels, plays a key role in regulating carbohydrate and lipid metabolism. In IR, peripheral tissues, most commonly skeletal muscle, liver, and adipose tissue, lose their sensitivity to insulin, leading to impaired glucose utilization [17]. Secondary insulin resistance in children is largely the result of environmental factors [16], with excess body weight being one of the most significant risk factors. It is estimated that IR occurs in approximately 38.7% of overweight or obese children, and the presence of increased visceral adipose tissue further exacerbates this disorder [18]. Although both subcutaneous and visceral fat are associated with the development of insulin resistance, visceral fat shows a stronger correlation with its severity [16]. Children with obesity often experience a compensatory increase in insulin secretion. Chronic exposure to hyperglycemia and elevated free fatty acid concentrations leads to gradual overload of pancreatic β cells, limiting their secretory capacity, and subsequently to their damage and apoptosis. This process promotes the development of type 2 diabetes at an early age [17].

Atherogenic dyslipidemia

Atherogenic dyslipidemia is the most common lipid disorder in overweight children and remains closely linked to obesity and metabolic syndrome. It occurs in about one-third of overweight children and in more than 40% of obese children [19]. An abnormal lipid profile often coexists with insulin resistance, type 2 diabetes, and non-alcoholic fatty liver disease [18]. In the case of so-called mixed dyslipidemia, a moderate increase in LDL cholesterol and triglyceride levels is observed, with HDL cholesterol levels usually remaining normal. This picture cannot always be clearly attributed to specific lifestyle factors or classic genetic mutations, such as those found in familial hypercholesterolemia. Nevertheless, early diagnosis is clinically important because environmental factors often play a dominant role in children, and the lack of improvement after modifying these factors may indicate the involvement of genetic determinants [19].

Hypertension

Obesity during childhood significantly increases the risk of hypertension, the pathogenesis of which is multifactorial. One of the key mechanisms is insulin resistance leading to hyperinsulinemia. Elevated insulin concentrations cause sodium and water retention in the kidneys and increase the synthesis of vasoconstrictive factors, leading to an increase in blood pressure [16,20]. Chronic inflammation associated with excess adipose tissue also plays an important role. Adipocytes secrete numerous pro-inflammatory mediators that disrupt blood vessel function, causing them to narrow and limiting their ability to relax. Children with obesity also have endothelial dysfunction, manifested by reduced nitric oxide production and increased secretion of vasoconstrictive substances [20,21]. Factors such as dyslipidemia and increased sympathetic nervous system activity may exacerbate the increase in blood pressure. The basis for the treatment of hypertension in obese children remains a comprehensive lifestyle change, including weight reduction, regular physical activity, sodium restriction, and control of other cardiovascular risk factors. In selected cases, it is also necessary to include pharmacological treatment to protect target organs [21].

Type 2 diabetes

In recent decades, there has been a marked increase in the incidence of type 2 diabetes in the pediatric population. Between 2001 and 2017, the incidence of this disease nearly doubled, rising from 0.34 to 0.67 cases per 1,000 people. Type 2 diabetes is more common among non-Hispanic black adolescents, Native Americans, and those with lower socioeconomic status. Following the COVID-19 pandemic, there has been an increase in the number of new diagnoses and a more severe course of the disease in this age group. Obesity, as a major risk factor for type 2 diabetes, is associated with an increase in the concentration of free fatty acids and pro-inflammatory adipokines, such as leptin, resistin, interleukin 6, and TNF-α. These changes increase insulin resistance, intensify hepatic glucose production, and reduce its uptake by skeletal muscles. At the same time, there is a decrease in the concentration of adiponectin, a hormone that improves insulin sensitivity [22]. Many children and adolescents already have microvascular complications or hypertension at the time of diagnosis of type 2 diabetes, and the risk of developing these complications exceeds 60% after 10–12 years of disease duration [26]. The course of type 2 diabetes in young patients is usually more aggressive than in adults, which is associated with deeper insulin resistance and faster depletion of pancreatic β-cell reserves [22].

Cardiovascular complications

Childhood obesity is considered to be a major factor accelerating the development of atherosclerosis, a process that can begin in early life [7]. Studies show that obese children and adolescents exhibit signs of premature aging of blood vessels, regardless of the presence of type 2 diabetes. These include both structural and functional changes leading to increased vascular stiffness [7,23,24]. A particularly strong correlation has been found between the amount of visceral adipose tissue and pulse wave velocity, an indicator of vascular wall stiffness and increased cardiovascular risk [24]. These data emphasize the importance of early identification of modifiable factors and implementation of preventive measures [23,24].

Impact on the skeletal system

Excessive body weight during growth affects bone metabolism through complex hormonal, inflammatory, and cellular interactions. Adipose tissue acts as an endocrine organ, secreting pro-inflammatory cytokines that promote chronic low-grade inflammation. This leads to an imbalance between bone formation and resorption processes, resulting in increased osteoclast activity and inhibition of osteoblast function [25,26]. Although greater body weight increases the mechanical load on bones, which in theory may promote their adaptation and strength, simultaneous inflammation and potential nutritional deficiencies may impair bone tissue quality. As a result, despite the observed acceleration of skeletal maturation and larger bone size, childhood obesity may increase the risk of fractures and other skeletal pathologies in the long term [26].

Psychosocial consequences

Obesity in childhood and adolescence significantly affects psychological and social functioning. Overweight children are more likely to experience low self-esteem, anxiety and depression, and a poorer quality of life in subjective terms. These difficulties can negatively affect peer relationships and daily functioning [27]. Excessive weight is also associated with a higher risk of school problems, eating disorders, and chronic stress. For this reason, it is particularly important to provide children and adolescents with obesity, especially severe obesity, with comprehensive psychological support that can reduce the long-term emotional and social consequences of the disease and improve the overall well-being of patients [15].

Therapeutic approach

As with other chronic conditions that arise during adolescence, the choice of treatment should be tailored not only to the patient's chronological age, but also to their biological development, the severity of their excess weight, their psychosocial context, and the presence of comorbidities. The therapeutic process should not be carried out according to a rigid scheme or divided into clearly separated stages, but should constitute a fluid, flexible model of care that can be modified according to the patient's needs. It is recommended to start treatment with the least invasive interventions, while ensuring their appropriate intensity. An important element is to discuss all available treatment options with the patient and their family, using language appropriate for the adolescent's age. It is preferable to use neutral terms, such as BMI, and to avoid phrases that could lead to stigmatization, including elaborate diagnostic names [28]. In the case of simple obesity, the basis of treatment remains a multidimensional intervention involving a change in diet, behavioral modification, and increased daily physical activity. These activities should be carried out under the supervision of an

interdisciplinary team of specialists with appropriate training in the treatment of children and adolescents. Nutritional strategies focus mainly on limiting uncontrolled food consumption. This is achieved by reducing free access to food, introducing regular meal times, and giving them a predictable structure. This model promotes a reduction in impulsivity, which often underlies overeating and food-seeking behaviors [29].

Regular moderate- to high-intensity physical activity is one of the key elements in both preventing and treating excess weight. It has a beneficial effect on insulin metabolism, facilitates the body's use of energy rather than storing it, and promotes muscle mass development, which further aids weight control. It is recommended to introduce physical activity gradually, starting with about 20 minutes a day and increasing the time to 60 minutes, in accordance with WHO and CDC recommendations. Regular physical activity not only improves cardiorespiratory fitness and promotes weight loss, but also reduces the concentration of inflammation markers associated with obesity [30]. In addition, it has a beneficial effect on the mental health of children and adolescents, strengthening their self-esteem and promoting the consolidation of healthy habits [31]. Interventions aimed at changing behavior, based on current scientific evidence, are also an extremely important element of long-term obesity treatment. Cognitive-behavioral therapy helps to address overeating related to emotional arousal and difficulties in self-control and motivation by teaching how to identify triggers and implement effective coping strategies [32]. Motivational techniques, setting realistic goals, and family involvement also increase the effectiveness of lifestyle changes [33]. Incorporating these methods into clinical practice translates into better long-term results, both in terms of weight control and improved mental well-being, especially in patients with eating disorders, anxiety symptoms, or low self-esteem [32].

Pharmacotherapy

In some cases, the treatment of obesity in children and adolescents may be supplemented with pharmacological therapy, especially when the disease is severe and accompanied by serious health complications. However, the use of drugs in this age group requires great caution due to the potential impact of long-term treatment on growth and development. For this reason, pharmacotherapy is usually reserved for adolescents with advanced obesity and comorbidities. Until recently, the number of drugs approved for the treatment of obesity in young patients was very limited. In clinical practice, orlistat and phentermine were mainly used. Orlistat, which inhibits pancreatic lipase activity, can lead to a reduction in BMI in adolescents over 12 years of age, but its use is often associated with adverse gastrointestinal symptoms and liver function disorders, resulting in frequent discontinuation of therapy. Long-term treatment with this drug may impair the absorption of fat-soluble vitamins and minerals, posing a potential risk to normal development [34]. A modern example of targeted therapy is setmelanotide, a melanocortin 4 receptor agonist approved by the FDA in 2020. This drug is intended for the treatment of rare, monogenic forms of obesity resulting from mutations in genes such as POMC, PCSK1, or LEPR and is an example of an approach based on the molecular mechanisms of the disease [35]. GLP-1 receptor agonists, such as liraglutide and semaglutide, are becoming increasingly important in the treatment of obesity. Initially developed for the treatment of type 2 diabetes, they have shown significant efficacy in weight reduction, which has expanded their clinical application. These drugs mimic the action of natural intestinal incretins and affect the body through a number of complementary mechanisms [34]. Their action includes the regulation of central appetite control mechanisms by influencing the hypothalamus and brainstem structures, leading to a reduction in hunger and an increase in satiety. At the same time, they slow down gastric emptying, limiting total energy intake. GLP-1 agonists also improve glucose metabolism by increasing glucose-dependent insulin secretion, inhibiting glucagon secretion, and improving tissue sensitivity to insulin. In addition, they have a beneficial effect on the liver, reducing the degree of steatosis and enhancing anti-inflammatory effects. The presence of GLP-1 receptors in immune system cells also plays a significant role. Their activation initiates many signaling pathways, including PKA, PI3K/Akt, MAPK, and NF-κB, which may explain the observed anti-inflammatory effects of these drugs in the liver and other organs [36].

Surgical treatment

The basis for obesity treatment remains conservative therapy, including lifestyle changes, increased physical activity, treatment of comorbidities, and, in justified cases, pharmacotherapy. However, if these measures do not produce lasting results and severe or morbid obesity with metabolic complications persists, bariatric surgery may be an effective therapeutic alternative [37]. In most patients, bariatric surgery leads to significant, long-term weight loss and improvement or remission of comorbidities. The most commonly performed procedures include sleeve gastrectomy and Roux-en-Y gastric bypass [38]. Sleeve gastrectomy is preferred in adolescents because it does not disrupt the continuity of the gastrointestinal tract and causes a

lower risk of malabsorption and intestinal complications. Regardless of the technique used, both procedures lead to a decrease in ghrelin concentration and an increase in the secretion of appetite-suppressing incretins, which promotes improved glycemic control and insulin sensitivity [15]. However, it should be emphasized that surgical treatment of obesity is associated with the risk of long-term nutritional complications. Stomach volume reduction and accelerated intestinal transit significantly modify eating habits, and procedures involving bypassing part of the intestine further increase the risk of malabsorption. As a result, many patients develop food intolerances manifested by nausea, vomiting, a feeling of food retention, bloating, or diarrhea, which may persist for a long time after surgery. Intolerances to products that are the main source of protein, vitamins, and minerals are a particular problem. Eliminating meat makes it difficult to meet the demand for protein, iron, and vitamin B12, avoiding dairy products promotes calcium deficiency, and limiting vegetable consumption increases the risk of insufficient fiber and vitamin intake [38]. As a result, malnutrition and micronutrient deficiencies may develop, and paradoxically, weight loss may be inhibited or weight may increase again if poorly tolerated products are replaced with high-energy, nutrient-poor foods [39].

Conclusions

Obesity in childhood and adolescence is one of the most serious challenges facing modern medicine and public health, leading to numerous health consequences that often persist into adulthood and significantly reduce quality of life. Due to the growing scale of this problem and its long-term health, social, and economic effects, it is essential to take coordinated action at the global, social, and individual levels to curb the further increase in the prevalence of this disease.

The development of obesity in children and adolescents is influenced by many interrelated factors, including environmental conditions, genetic predisposition, hormonal disorders, and social determinants. Lifestyle, eating habits, level of physical activity, and the attitudes and behaviors of caregivers, which significantly shape a child's daily functioning, also play an important role. For this reason, effective treatment of obesity requires a comprehensive approach tailored to the individual needs of the patient, based on close cooperation between specialists from various fields who jointly plan, implement, and monitor the therapeutic process.

Early diagnosis of complications associated with excess weight is particularly important, as rapid and appropriately targeted intervention can significantly reduce the risk of developing serious diseases in the future, such as metabolic disorders, cardiovascular diseases, and psychosocial problems. Although obesity prevention from an early age plays a key role in reducing the scale of the phenomenon, the methods currently available are still insufficient and need further improvement. Therefore, only joint, long-term actions involving the healthcare system, education, family, and the social environment can effectively reduce the negative impact of this global epidemic on the health of children and adolescents.

Disclosure

Author's contribution

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