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Research Progress on Epidemiological Characteristics, Pathogenesis, and Prevention and Control of Multisystem Complications of Childhood Obesity

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Abstract: Childhood obesity has emerged as a significant global public health issue, with its epidemic trend and the complexity of its pathogenic mechanisms posing formidable challenges to prevention and control efforts. This article systematically reviews the epidemiological characteristics of childhood obesity, focusing on its global and national prevalence status and core risk factors. It delves into the collaborative pathogenic mechanisms involving genetic and epigenetic regulation, intestinal microbiota dysbiosis, and disruptions in the neuro-endocrine-metabolic network. Furthermore, it comprehensively elucidates the clinical features of multisystem complications. Finally, it summarizes research progress in prevention strategies, clinical interventions, and novel technologies, providing references for the standardized prevention and control of childhood obesity.

Keywords: Childhood obesity; Epidemiology; Pathogenesis; Multisystem complications; Prevention and control progress

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1. Introduction

Childhood obesity is a chronic metabolic disease caused by excessive accumulation of body fat, diagnosed based on a Body Mass Index (BMI) ≥ the 95th percentile for age and gender [1]. With dramatic changes in dietary patterns and lifestyles, childhood obesity has exhibited trends of "globalization, younger age of onset, and increasing severity," and has been identified by the World Health Organization as one of the most urgent public health challenges of the 21st century. Obesity in childhood not only directly hinders growth and development but also increases the risk of chronic diseases in adulthood through the "metabolic memory" effect, while triggering psychological and behavioral issues, resulting in lifelong health impairments [2]. This paper integrates core evidence from recent years to systematically review research progress in childhood obesity, providing a scientific basis for

clinical practice and public health interventions.

2. Epidemiological characteristics

2.1. Global and domestic prevalence

The incidence of childhood obesity worldwide has exploded over the past half-century, with particularly notable "catch-up growth" in low- and middle-income countries, which have become new engines driving the global obesity epidemic. Developed countries in Europe and North America are at a high plateau of prevalence, while Asia, despite having a lower baseline prevalence, is experiencing a growth rate far exceeding that of other continents.

The prevalence of childhood obesity in China is also severe. According to the "Chinese Guidelines for the Diagnosis and Treatment of Obesity (2024 Edition)" [3] released by the National Health Commission in 2024, the overweight rate among children and adolescents aged 6 to 17 reached 11.1%, and the obesity rate was 7.9%, with a combined total of nearly 20%. For children under 6 years old, the overweight and obesity rates were 6.8% and 3.6%, respectively, showing a clear trend towards younger ages. In terms of population distribution, the characteristics of "higher prevalence in the north than in the south, in urban areas than in rural areas, and among boys than among girls" remain, but the increase in rural areas has been significantly higher than that in urban areas, and the gap between urban and rural areas continues to narrow. Special groups such as left-behind children and only children face a significantly higher risk of obesity due to differences in lifestyle and care patterns, making them a key focus group.

2.2. Core risk factors

The occurrence of childhood obesity is not the result of a single factor but rather the interaction of genetic and environmental factors, with environmental factors being the primary driving force behind the current epidemic. In terms of diet, excessive intake of high-energy, high-sugar, and high-fat processed foods and sugary beverages, as well as insufficient intake of dietary fiber and high-quality protein, and irregular meal patterns and overeating, directly contribute to energy imbalance [4]. Among behavioral factors, the "triple threat" of insufficient outdoor activities, prolonged screen time, and sleep deprivation collectively reduces energy expenditure and disrupts metabolic rhythms. At the social environmental level, parental cognitive biases, the lack of health education in schools, insufficient community sports facilities, and misleading food marketing collectively create an "obesogenic environment" that is detrimental to health.

Genetic factors provide a predisposing background for the development of obesity, with the heritability of childhood obesity estimated to be around 40%—70%. Classic gene variants such as FTO and MC4R are closely associated with obesity susceptibility [5]. It is noteworthy that genetic risks can only fully manifest under the catalysis of environmental factors, and improving lifestyle can effectively reduce the incidence probability among genetically susceptible populations.

3. Pathogenesis

3.1. Genetic and epigenetic regulation

Genetic factors influence obesity susceptibility by regulating pathways related to energy metabolism and fat synthesis. Polymorphisms in genes such as PPARG and ADIPOQ can promote fat accumulation by affecting

adipocyte differentiation and insulin sensitivity, respectively [6]. More critically, epigenetic mechanisms play a "bridging role" in the interaction between genetics and the environment. Maternal obesity, malnutrition, or exposure to environmental pollutants during pregnancy can lead to abnormal epigenetic modifications such as methylation in the fetal genome, such as methylation changes in the promoter region of the LEP gene. This "metabolic imprinting" can persist into adulthood, significantly increasing the risk of obesity [7]. Additionally, microRNAs such as miR-143 and miR-221 can participate in the pathological process of obesity by targeting and regulating genes involved in adipocyte differentiation.

3.2. Intestinal dysbiosis

Intestinal microbiota imbalance is a significant pathogenic mechanism underlying childhood obesity [8]. Overweight/obese children exhibit significantly lower diversity in their intestinal microbiota compared to children of normal weight, along with characteristic alterations in microbial composition: an increase in the abundance of certain Firmicutes genera and a decrease in certain Bacteroidetes genera. This imbalance promotes obesity through three pathways: enhancing intestinal fat absorption efficiency; fermenting dietary fiber to produce short-chain fatty acids that activate intestinal receptors and stimulate appetite; and triggering low-grade inflammation that activates the TLR4/NF-κB pathway, leading to insulin resistance. Additionally, the intestinal microbiota can regulate central appetite centers via the "gut-brain axis," forming a closed regulatory loop of "microbiota-metabolism-nerve." Research has also found that feeding practices, dietary patterns, exercise habits, and sleep behaviors are modifiable factors influencing the intestinal microbiota, providing targets for obesity prevention and control through microbiota intervention [9].

3.3. Disruption of the neuro-endocrine-metabolic network

The regulatory network formed by the central nervous system and the endocrine system is central to maintaining energy balance. Dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis is extremely common in obese children, with elevated cortisol levels specifically promoting abdominal fat accumulation [10]. Disruption of the leptin-leptin receptor signaling pathway is a critical factor. Obese children often exhibit "leptin resistance," characterized by elevated leptin levels that fail to effectively suppress appetite or increase energy expenditure, leading to a vicious cycle of metabolic imbalance. Additionally, endocrine abnormalities such as decreased insulin sensitivity and insufficient growth hormone secretion can exacerbate obesity by promoting fat synthesis and reducing fat breakdown, respectively [11].

3.4. Mediating role of environmental factors

The aforementioned environmental risk factors do not directly cause obesity but rather facilitate its development by targeting the aforementioned mechanisms. High-sugar diets can activate the hypothalamic reward pathway, leading to appetite addiction, while also causing dramatic fluctuations in blood glucose levels and excessive insulin secretion [12]. A lack of physical activity not only reduces energy expenditure but also decreases muscle mass and basal metabolic rate. Sleep deprivation disrupts the function of the hypothalamic-pituitary-adrenal (HPA) axis, resulting in decreased leptin secretion and increased ghrelin levels, as well as reduced insulin sensitivity, forming a pathological chain of "sleep deprivation-metabolic disorder-obesity."

4. Multisystem complications

Childhood obesity is not merely a matter of excessive body weight; it can cause persistent damage to multiple organ systems, triggering a series of serious complications. Moreover, these damages are characterized by their insidious and progressive nature.

4.1. Metabolic system complications

Metabolic syndrome is the most common complication in obese children, characterized by central obesity, hypertension, hyperglycemia, and dyslipidemia as its core manifestations, significantly increasing the risk of long-term chronic diseases. The age of onset for type 2 diabetes mellitus (T2DM) continues to advance, with obese children facing a more than tenfold higher risk of developing the disease compared to children of normal weight. Moreover, T2DM often has an insidious onset, leading to delays in diagnosis and treatment. Non-alcoholic fatty liver disease is another highly prevalent complication, with approximately half of obese children exhibiting varying degrees of hepatic steatosis. Some cases may progress to hepatitis and fibrosis, becoming the primary cause of liver disease during childhood [13].

4.2. Cardiovascular system complications

Childhood obesity is an independent risk factor for cardiovascular diseases in adulthood and can directly cause cardiovascular damage during childhood. The prevalence of hypertension is significantly higher among obese children, often presenting insidiously and going unnoticed [14]. Dyslipidemia is characterized by elevated triglycerides and low-density lipoprotein cholesterol, along with reduced high-density lipoprotein cholesterol, accelerating the progression of atherosclerosis. Long-term obesity can also lead to structural and functional changes in the heart, such as left ventricular hypertrophy and myocardial strain, with severe cases potentially resulting in heart failure.

4.3. Complications of the respiratory system and musculoskeletal system

Obstructive sleep apnea syndrome is a characteristic complication in obese children, manifesting as nighttime snoring and apnea, daytime drowsiness, and lack of concentration. Chronic hypoxemia can adversely affect brain development and cardiovascular function [15]. Obese children also have a significantly higher prevalence of asthma, with more frequent recurrences and greater difficulty in control. In terms of the musculoskeletal system, excessive body weight can increase pressure on the lower limb bones, leading to premature epiphyseal closure, scoliosis, flat feet, and other deformities. It also accelerates the wear and tear of articular cartilage in the knee and hip joints, accompanied by insufficient muscle strength and decreased motor coordination, creating a vicious cycle of "obesity - reduced physical ability - worsening obesity" [16].

4.4 .Psychological behavior and social adaptation complications

Obese children often face ridicule and discrimination from their peers due to their body size, leading to a significantly higher incidence of psychological issues such as low self-esteem, anxiety, and depression compared to children of normal weight [17]. They also exhibit higher rates of inattention and decreased learning efficiency, with impaired social skills and confidence. Over the long term, this can result in decreased social adaptability, affecting career development and quality of life in adulthood.

5. Prevention and control strategies

The prevention and control of childhood obesity should adhere to the core principle of "prevention first and comprehensive intervention," establishing a multi-dimensional, full-cycle prevention and control system, while implementing differentiated intervention measures for children with different weight statuses.

5.1. Prevention strategies

The prevention and control of childhood obesity adhere to the core principle of "prevention first" and establish a comprehensive prevention and control model integrating "family-school-community-society." At the family level, parents should set a healthy example, optimize dietary structure, ensure children's daily intake of sufficient vegetables, fruits, and high-quality protein, supervise them to engage in at least one hour of moderate-intensity physical exercise, control screen time, and ensure adequate sleep. At the school level, nutrition and health education should be incorporated into the curriculum, the quality of cafeteria meals should be improved, and time for physical activities should be guaranteed. At the community level, sports facilities should be improved, and scientific popularization on obesity prevention and control should be carried out. At the societal level, food advertising should be strictly regulated, the production of healthy foods should be encouraged, and a unified national network for monitoring and intervening in childhood obesity should be established [18].

5.2. Clinical intervention measures

For children who have already developed obesity, a comprehensive treatment plan combining "dietary adjustment + exercise intervention + behavioral correction" should be adopted. Dietary adjustment focuses on "energy control and nutritional balance," reducing the intake of sugary beverages and processed snacks, maintaining regular meals, and avoiding extreme dieting [19]. Exercise interventions primarily consist of moderate-intensity aerobic exercises combined with strength training, with personalized plans tailored according to children's age and interests. Behavioral modification enhances intervention adherence by establishing healthy habits, implementing reward and punishment mechanisms, improving parent-child relationships, and providing psychological counseling.

5.3. Research on novel therapeutic technologies and medications

Currently, multiple breakthroughs have been achieved in the field of pediatric obesity treatment. Digital health interventions have emerged as a new hotspot. For instance, family-oriented online healthy lifestyle programs can significantly reduce BMIz scores, improve dietary quality and exercise habits, and enhance the quality of life in children, offering a feasible pathway for large-scale obesity interventions.

In terms of pharmacological treatment, GLP-1 receptor agonists such as semaglutide have demonstrated clear efficacy in obese children aged 12 and above [20]. For example, daily subcutaneous injection of liraglutide 3.0 mg combined with lifestyle interventions can significantly reduce BMI in obese children aged 6 to 12, with good safety profiles, providing a new option for pharmacological treatment in younger obese children [21]. Fecal microbiota transplantation remains in the clinical research stage, aiming to achieve weight reduction by improving gut microbiota composition, but its long-term efficacy and safety require further validation. Bariatric surgery is only applicable to a very small number of severely obese children with serious comorbidities, necessitating strict indication control and long-term postoperative management.

6. Conclusion

Childhood obesity is a chronic disease resulting from the combined effects of multiple factors, including genetics, environment, gut microbiota, and disturbances in the neuro-endocrine-metabolic network. Its prevalence is alarming and has emerged as a significant global public health issue. Childhood obesity not only affects growth and development but also triggers complications across multiple systems, such as metabolic, cardiovascular, and respiratory systems, while also causing psychological, behavioral, and social adaptation impairments. The breakthroughs in the 2024 updated clinical guidelines and multiple clinical studies have provided new evidence for prevention and control efforts. Current prevention and control strategies should adhere to a "prevention-first, comprehensive intervention" approach, creating a healthy environment through collaborative efforts from families, schools, communities, and society at large. For children already affected by obesity, individualized comprehensive interventions should be implemented, supplemented by novel drugs or technological treatments when necessary. In the future, with the advancement of precision medicine and digital health technologies, individualized and precise prevention and control will become the mainstream trend, offering stronger guarantees for the effective management of childhood obesity.

Disclosure statement

The authors declare no conflict of interest.

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