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The Correlation, Prevention, and Treatment of Obesity and Type 2 Diabetes

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Abstract: ***In today's world, obesity and type 2 diabetes have become a dual crisis threatening global health. According to data from the World Health Organization, the global population of obese individuals has tripled over the past four decades [1], while the International Diabetes Federation's report in 2021 indicates that approximately 537 million adults worldwide suffer from diabetes [2]. The situation in China is equally severe, with persistently high rates of overweight and obesity among adult residents [3], over 90% of which are type 2 diabetes. What is even more striking is the close association between these two diseases - research shows that approximately 80-85% of patients with type 2 diabetes also suffer from overweight or obesity. This is not an accidental coincidence, but rather a deep-seated physiological and pathological connection. This article aims to delve into the complex relationship between obesity and type 2 diabetes, analyze their common pathogenesis, and explore integrated prevention and treatment strategies.***

Keywords: Obesity, Type 2 diabetes, Physiological pathology, Prevention and control.

1. The Epidemiology of Obesity and Type 2 Diabetes are Intertwined

1.1 Epidemiological Characteristics of Obesity

Global Trend: Obesity has emerged as a global public health issue. According to data from the World Health Organization, over 13% of adults worldwide are obese, and the obesity rate continues to rise [1]. Since 1975, the global obesity prevalence has nearly doubled. It is estimated that by 2030, the number of obese adults globally will increase to 1.02 billion, accounting for approximately 18% of the adult population.

China's situation: According to the report on the nutrition and chronic disease status of Chinese residents, the overweight rate among Chinese adults (aged ≥ 18) is 34.3%, and the obesity rate is 16.4%. The overweight and obesity rates among adolescents and children are also on the rise, with the overweight rate among children aged 6-17 being 11. 1% and the obesity rate being 7.9% [3].

Gender and age differences: The overweight rate and prevalence of obesity in males are generally higher than those in females, but the metabolic risks associated with obesity may be higher in females. The prevalence of obesity increases with age, with higher rates among middle-aged and elderly populations. However, in recent years, the problem of obesity among adolescents has also become increasingly prominent [1,3].

1.2 Epidemiological Characteristics of Type 2 Diabetes

Global Trend: Type 2 diabetes is the most common type of diabetes globally, accounting for over 90% of all diabetes cases. According to the International Diabetes Federation, the number of type 2 diabetes patients worldwide continues to rise, and it is estimated that by 2045, the global number of diabetes patients will exceed 780 million [2].

China's situation: China is one of the countries with the largest number of diabetes patients. According to data from 2018, the prevalence rate of diabetes among adults aged 18 and above in China was 12.4%, with type 2 diabetes accounting for more than 90% [3]. The prevalence rate of diabetes tends to be higher in the eastern region and lower in the western region, correlating with the level of economic development.

Age and gender differences: The age of onset of type 2 diabetes is showing a trend towards younger age groups, with the incidence rate gradually increasing among young and middle-aged populations. The prevalence rate of type 2 diabetes in males is usually higher than that in females, but female patients may be diagnosed at higher BMI levels and face higher risks of cardiovascular disease and death.

1.3 The Association Between Obesity and Type 2 Diabetes

Obesity is a significant risk factor for type 2 diabetes [4]: Numerous studies have shown that obese individuals have a significantly increased risk of developing type 2 diabetes. The higher the body mass index (BMI), the higher the risk of developing type 2 diabetes. For example, the prevalence of type 2 diabetes in individuals with a BMI of ≥30kg/m² is significantly higher than that in individuals with a BMI of <25kg/m². Obesity leads to insulin resistance [5]: Obesity, especially abdominal obesity, can cause adipose tissue to secrete excessive adipokines and inflammatory factors, interfering with insulin signaling and leading to insulin resistance, which in turn increases the risk of developing type 2 diabetes. Weight loss can improve metabolic indicators [8]: For patients with obesity and type 2 diabetes, weight loss is an important treatment strategy. A weight loss of 3%-7% can significantly improve metabolic indicators such as blood glucose, blood lipids, and blood pressure, and some patients may even achieve diabetes remission or reversal. In summary, obesity and type 2 diabetes have a close epidemiological relationship, with obesity being a significant risk factor for type 2 diabetes, and both showing a global upward trend. Strengthening obesity prevention and control and promoting healthy lifestyles are of great significance for the prevention and control of type 2 diabetes.

2. Physiological and Pathological Mechanisms of Obesity and Type 2 Diabetes

2.1 Insulin Resistance

2.1.1 Impact of adipose tissue

In obesity, especially visceral fat accumulation, adipocytes secrete a large amount of free fatty acids and inflammatory factors (such as tumor necrosis factor-α, interleukin-6, etc.). These substances interfere with the insulin signaling pathway, reducing the sensitivity of muscles, liver, and adipocytes to insulin, leading to insulin resistance [5]. For example, free fatty acids can inhibit the insulin-stimulated transport of glucose transporter 4 (GLUT4) to the cell membrane, reducing the cell's glucose uptake and utilization.

2.1.2 The role of inflammatory response

In a state of obesity, adipose tissue experiences chronic low-grade inflammation, with immune cells such as macrophages infiltrating the adipose tissue and releasing inflammatory mediators, further exacerbating insulin resistance [6]. Activation of inflammatory signaling pathways can inhibit the phosphorylation of insulin receptor substrates (IRS), hindering insulin signaling transmission.

2.2 Islet β-cell Dysfunction

Compensatory secretion increase: In the early stage of insulin resistance, pancreatic β cells will compensatorily secrete more insulin to maintain blood glucose stability. However, long-term hyperinsulinemia and metabolic stress will gradually impair β cell function [5]. Lipotoxicity and gluotoxicity: During obesity, the levels of free fatty acids and triglycerides in the blood increase. Fatty acids are metabolized within β cells to produce lipid intermediates (such as diacylglycerols and ceramides), which can inhibit insulin gene expression, secretion, and β cell proliferation, inducing β cell apoptosis [5]. Meanwhile, long-term hyperglycemia also exerts toxic effects on β cells, affecting their function and survival.

2.3 Liver Glucose and Lipid Metabolism Disorder

Increased gluconeogenesis: Obesity-induced insulin resistance reduces the sensitivity of the liver to insulin, weakening the inhibitory effect of insulin on gluconeogenesis. This increases glucose output from the liver, leading to elevated fasting blood glucose. Simultaneously, the release of free fatty acids from adipose tissue increases, providing substrates for gluconeogenesis in the liver and further exacerbating hyperglycemia. Abnormal fat metabolism: Obese patients experience increased triglyceride deposition in the liver, making them prone to non-alcoholic fatty liver disease (NAFLD). Hepatic steatosis can affect insulin signaling, exacerbate insulin resistance, and promote the synthesis and secretion of very low-density lipoprotein (VLDL), leading to hypertriglyceridemia [4].

2.4 Abnormal Skeletal Muscle Metabolism

Reduced glucose uptake: Skeletal muscle is the main tissue for insulin-stimulated glucose uptake and utilization. In obesity, intracellular lipid deposition in skeletal muscle cells increases, and free fatty acids and lipid intermediates can inhibit the insulin signaling pathway, reducing the transport of GLUT4 to the cell membrane, leading to decreased glucose uptake and oxidation capacity [5]. Mitochondrial dysfunction: Obesity-related metabolic disorders can lead to abnormal mitochondrial function in skeletal muscle, affecting energy metabolism and fatty acid oxidation, further aggravating insulin resistance and metabolic disorders.

2.5 Gut Microbiota

In recent years, the gut microbiota has garnered widespread attention as a potential mediator between obesity and type 2 diabetes [7]. The human gut harbors trillions of microorganisms, and their collective genome (microbiome) encodes more than 100 times the number of genes found in the human genome, profoundly influencing the host's metabolic health. The gut microbiota composition of obese individuals significantly differs from that of lean individuals, characterized by reduced microbial diversity and altered proportions of specific bacterial groups (such as an increased Firmicutes/Bacteroidetes ratio) [7, 15]. This "ecological imbalance" may promote metabolic disorders through multiple mechanisms: firstly, the dysregulated microbiota extracts energy more efficiently from the diet, promoting weight gain [15] ; secondly, they alter bile acid metabolism, affecting fat digestion and the regulation of glycolipid metabolism; thirdly, they disrupt the gut barrier function, leading to the entry of endotoxins (such as lipopolysaccharides) into the circulation, triggering metabolic endotoxemia and systemic inflammation [6]; finally, they produce various microbial metabolites (such as short-chain fatty acids, branched-chain amino acids, trimethylamine-N-oxide, etc.), which can directly or indirectly affect insulin sensitivity and β-cell function. The gut microbiota may partially explain the complex relationship between diet, obesity, and type 2 diabetes. A high-fat, high-sugar Western diet can rapidly alter the gut microbiota composition, promoting the growth of metabolically harmful bacterial groups. Conversely, a diet rich in dietary fiber can promote the proliferation of beneficial bacteria (such as those producing short-chain fatty acids), which not only provide energy for colonic cells but also enhance the gut barrier function, reduce inflammation, and improve insulin sensitivity [7].

Obesity increases the risk of developing type 2 diabetes by inducing insulin resistance, dysfunction of pancreatic β-cells, metabolic disorders in the liver and skeletal muscle, and alterations in the gut microbiota. These factors interact with each other, forming a vicious cycle that collectively promotes the occurrence and progression of metabolic diseases.

3. Prevention and Treatment of Obesity and Type 2 Diabetes

Given the close association between obesity and type 2 diabetes, modern clinical management emphasizes the integration of treatment strategies, targeting both diseases simultaneously and breaking the vicious cycle of mutual promotion.

3.1 Lifestyle Intervention - the Cornerstone of Prevention and Treatment

This is the most economical, effective, and side-effect-free fundamental measure, which permeates the entire process of disease prevention, treatment, and management.

3.1.1 Medical nutritional therapy

It is not simply "dieting", but rather a personalized and sustainable adjustment of dietary patterns. Highly recommended: calorie-restricted balanced diet: reduce daily calorie intake by 500-750 kcal while ensuring nutritional balance; Mediterranean diet pattern: rich in olive oil, nuts, fish, whole grains, vegetables, and fruits, with significant anti-inflammatory effects [14]; low-carbohydrate or low-glycemic index diet: helps to rapidly reduce weight, improve blood glucose and insulin sensitivity. The core goal is to achieve and maintain a weight loss of 5%-10%, which is sufficient to significantly improve insulin resistance, reduce glycosylated hemoglobin, and decrease cardiovascular risk [8].

3.1.2 Regular physical activity

Exercise is an independent insulin sensitizer. It is recommended to engage in at least 150 minutes of moderate-intensity aerobic exercise (such as brisk walking, cycling) per week, combined with 2-3 sessions of resistance training (such as weightlifting, elastic band exercises) per week. Exercise not only burns energy and reduces fat, but also directly increases muscle glucose uptake, improves mitochondrial function, and reduces inflammation levels. Even without significant weight loss, regular exercise can bring significant metabolic benefits [8].

3.1.3 Behavioral and cognitive intervention: Through cognitive behavioral therapy, motivational interviewing, etc., help patients identify unhealthy eating behavior patterns, manage stress, improve sleep quality, overcome laziness, and establish long-term healthy habits.

3.2 Pharmacotherapy - a Weapon of Precise Targeting

When lifestyle intervention proves insufficient, pharmacotherapy should be initiated promptly. The modern pharmacotherapy concept has shifted from solely "lowering blood glucose" to "improving metabolism and cardiorenal outcomes" [12].

3.2.1 Drugs for obesity

GLP-1 receptor agonists (such as semaglutide and liraglutide): These drugs mimic the action of the gut hormone GLP-1, which not only effectively lowers blood glucose but also strongly suppresses appetite and delays gastric emptying, resulting in significant weight loss (an average of 10%-15%). They also have a clear cardiovascular protective effect, perfectly meeting the treatment needs of "glycemic obesity" [9].

3.2.2 Other weight-loss drugs

Such as the central nervous system-acting phenformin/topiramate combination and the intestinal-acting orlistat, which can be used for specific populations under the guidance of a doctor [12].

3.2.3 Drugs for type 2 diabetes

When selecting drugs, both hypoglycemic effects and metabolic benefits should be considered. Metformin: a first-line basic medication that can improve insulin resistance, achieve mild weight loss, and is inexpensive [8, 12]; SGLT2 inhibitors (such as empagliflozin and dapagliflozin): they lower blood glucose by promoting urinary glucose excretion, while bringing multiple benefits such as weight loss, blood pressure reduction, reduced risk of hospitalization for heart failure, and kidney protection [10]; DPP-4 inhibitors can be used as options for combination therapy. The key to treatment strategy lies in early combination therapy, with multi-target intervention targeting different pathological defects (insulin resistance, β-cell dysfunction, obesity, etc.), to rapidly and durably control metabolic indicators and delay complications.

3.3 Metabolic Surgery - a Powerful Means to Alter the Disease Progression

For patients with type 2 diabetes who are severely obese (BMI ≥ 37.5 kg/m², or BMI ≥ 32.5 kg/m² with severe complications), metabolic surgery (such as sleeve gastrectomy and gastric bypass surgery) is currently the most effective long-term treatment. Its efficacy far exceeds that of any medication: significant weight loss (typically reducing 60%-80% of excess body weight); a high rate of diabetes remission (approximately 75% of patients achieve normal blood glucose levels without medication one year post-surgery) [11]; its mechanism is extremely complex, far beyond the mere "restriction of intake and absorption", involving significant changes in the secretion of gut hormones (GLP-1, PYY, etc.), remodeling of bile acid metabolism, adjustment of gut microbiota, and changes in central appetite regulation, fundamentally altering the body's metabolic "set point".

3.4 Public Health and Social Environmental Intervention - Creating a Healthy "macro-climate"

Individual efforts require a supportive environment. Governments and society should shoulder their responsibilities and make healthy choices the easy choice through policies and environmental transformation [1, 12].

3.4.1 Policy level

Implementing sugar taxes or imposing taxes on unhealthy foods; mandatory and concise food labeling (such as "traffic light" labeling); strictly restricting junk food advertising targeted at children.

3.4.2 Environmental level

Urban planning should incorporate more green spaces, pedestrian walkways, and bicycle lanes; encourage enterprises to establish a system for exercising during work breaks; and schools should ensure sufficient physical education classes and provide healthy school meals.

3.4.3 Education level

Carry out universal health education to enhance public awareness of the hazards and prevention methods of obesity and diabetes, and eliminate discrimination against obese individuals [1,3].

In summary, obesity and type 2 diabetes are interrelated, and their prevention and treatment require comprehensive lifestyle intervention, pharmacotherapy, and surgical intervention when necessary. Through rational diet, moderate exercise, and weight management, these two diseases can be effectively prevented and controlled, reducing the risk of complications and improving quality of life. In addition, personalized medicine is increasingly important in the management of obesity and type 2 diabetes. Based on patients' clinical characteristics (such as age, disease duration, and complications), biochemical indicators (such as C-peptide levels and insulin resistance), genetic background, and gut microbiota composition, developing individualized treatment goals and plans has become a trend in modern clinical practice. The application of digital health technologies (such as continuous glucose monitoring and mobile health applications) provides new tools for patient self-management and remote monitoring by healthcare teams, helping to improve treatment adherence and effectiveness [12]. Preventing obesity and type 2 diabetes requires going beyond the individual level and adopting a socio-ecological approach, intervening simultaneously at multiple levels. At the individual level, emphasizing early screening and risk identification is crucial. Identifying individuals with prediabetes (impaired fasting glucose or impaired glucose tolerance) and providing intensive lifestyle intervention has been proven to be an economical and effective prevention strategy [13]. The application of genetic risk scores and multi-omics markers is expected to further improve the accuracy of risk prediction and achieve precision prevention [12]. At the societal level, creating an environment that promotes healthy choices is key. This includes implementing policies such as taxing sugary beverages, improving food labeling systems, restricting the marketing of unhealthy foods to children, and planning community spaces that facilitate physical activity. Workplace health promotion programs and school nutrition education are also important components.

Looking ahead, emerging technologies are poised to revolutionize prevention strategies. Artificial intelligence can be harnessed to analyze complex data and identify high-risk individuals; mobile health applications can offer personalized behavioral interventions; and wearable devices make continuous health monitoring feasible. However, these technologies must be integrated with social policies that address health inequalities to ensure that all populations benefit.

4. Conclusion

The close relationship between obesity and type 2 diabetes goes far beyond superficial epidemiological associations. It is rooted in shared pathophysiological mechanisms, involving complex interactions at multiple levels such as adipose tissue dysfunction, insulin resistance, β-cell failure, and intestinal microbiota imbalance. These mechanisms are intertwined, forming a dynamic metabolic network that drives the occurrence and progression of the disease.

Understanding these deep connections not only holds scientific significance but also has profound implications for clinical practice and public health policy. It prompts us to re-examine the traditional treatment model centered on a single disease and shift to a holistic management strategy with metabolic health as the core. At the same time, it emphasizes the importance of prevention, especially intervening early in life, which may break the intergenerational transmission of metabolic risks [13].

Facing the challenges posed by the global epidemic of obesity and diabetes, we need to integrate multidisciplinary perspectives from biomedicine, public health, and social sciences to develop comprehensive solutions ranging from molecular mechanisms to social policies. Only through such comprehensive efforts can we curb the spread of these twin epidemics, alleviate their heavy burden on individual health and socio-economic systems, and move towards a future where metabolic health is more prevalent.

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