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Nutritional Intervention Strategies for People with Metabolic Syndrome Based on the Theory of Medicinal-Food Homology

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Abstract. Metabolic syndrome (MetS) is a multifactorial metabolic disease characterized by central obesity, glucose and lipid metabolism disorders, and hypertension. Its prevalence continues to rise, posing a serious threat to public health. While traditional drug treatments have some efficacy, long-term use is prone to adverse reactions and has low adherence. Nutritional interventions based on the concept of "medicine and food homology" have gradually become a research hotspot due to their naturalness and multi-target regulatory advantages. This article systematically reviews the mechanisms of action and application progress of substances derived from both medicine and food homology in MetS intervention, focusing on analyzing their synergistic regulatory mechanisms through four main pathways: improving insulin sensitivity, regulating lipid metabolism, inhibiting chronic inflammatory responses, and reshaping the gut microbiota structure. Studies have shown that multiple active ingredients form a synergistic effect across multiple pathways, significantly improving blood glucose, blood lipid levels, and inflammatory states, and demonstrating superior intervention effects compared to single-component interventions in animal experiments and some clinical studies. Nevertheless, current research still faces challenges such as a lack of standardized compound treatments and insufficient long-term clinical evidence. Future research should focus on strengthening high-quality clinical studies and mechanism exploration, promoting the precision development of interventions based on the homology of medicine and food, and providing stronger theoretical support for non-drug interventions for MetS.

Keywords: Metabolic syndrome, medicinal-food homology, nutritional intervention, insulin resistance, gut microbiota.

1. Introduction

Metabolic syndrome (MetS) is a clinical syndrome characterized by central obesity, abnormal blood glucose, abnormal blood lipids and hypertension. It is also a potential pathogenic factor for many diseases such as cardiovascular and cerebrovascular diseases, hyperuricemia and type 2 diabetes (T2DM) [1]. With the change of lifestyle in China, the prevalence of MetS has been rising continuously, with an adult prevalence of 31.1%, posing a serious threat to China's public health

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security [2]. At present, drug intervention is still the main treatment for MetS. Metformin and statins are commonly used in clinical practice to regulate blood glucose and blood lipids, respectively. However, long-term use can easily cause adverse reactions such as gastrointestinal discomfort and liver and kidney damage. In addition, strict regular medication is required, which conflicts with patients' daily diet and lifestyle, resulting in generally low treatment compliance and difficulty in meeting the clinical needs of long-term management of MetS [3].

For example, metformin's main side effects are gastrointestinal intolerance symptoms such as abdominal pain, diarrhea, bloating and loss of appetite. A randomized controlled trial of elderly patients with T2DM showed that after 361 patients received different doses of metformin, 62 of them chose to discontinue treatment within the first 4 weeks due to intolerance to adverse reactions [3]. In recent years, nutritional intervention based on the Traditional Chinese medicine concept of "medicine and food sharing the same origin" has become a research hotspot for non-drug intervention of MetS. This intervention method regulates the body's metabolism through daily diet, thereby reducing the risk of MetS from the root cause. It is a more gentle and sustainable intervention plan [4]. "Medicine and food sharing the same origin" refers to natural substances that have both medicinal and edible properties. The natural active ingredients of plants such as kudzu root, yam, and hawthorn can regulate insulin sensitivity and improve lipid metabolism while supplementing the body's nutrition, which is highly in line with the intervention needs of the MetS patient population [5]. However, existing studies mostly focus on the therapeutic effects of single "medicine and food sharing the same origin" substances, and the analysis of their combined intervention in clinical practice is still relatively insufficient. Therefore, this review focuses on the combined effects of "medicine and food sharing the same origin" substances, aiming to provide a more clinically relevant theoretical reference for non-drug intervention of MetS.

2. Current status of research on the intervention of MetS by food and drug homologous substances

The pathological mechanism of MetS is centered on insulin resistance (IR), forming a vicious cycle of multi-stage metabolic disorders. IR can directly cause the imbalance of glucose metabolism homeostasis in the body, leading to a decrease in the ability to regulate blood glucose. At the same time, it can cause abnormal synthesis and deposition of lipids in the liver and adipose tissue by regulating key pathways of lipid metabolism, resulting in hyperlipidemia and central obesity. Obesity, especially the abnormal increase in abdominal fat content, can further aggravate IR by secreting adipokines, and induce damage to vascular endothelial function, eventually leading to symptoms such as hypertension, forming a pathological feature of mutual promotion of glucose and lipid metabolism disorders, obesity, and hypertension [6]. Multiple large-sample epidemiological studies have confirmed that there is a clear causal relationship between the occurrence and development of MetS and residents' daily unhealthy lifestyle habits.

Excessive intake of high-fat, high-sugar diets and refined carbohydrates can significantly increase postprandial blood glucose and blood lipid levels, increase the metabolic burden on the pancreas and liver, and directly induce IR in the long term. An 8-year cohort study of 12,867 European adults showed that the incidence of MetS was 2.1 times higher in people who consumed ≥500 mL of sugary drinks per day than in people who did not consume them, and the risk of abnormal glucose and lipid metabolism was significantly increased [7]. At the same time, insufficient physical activity and sleep deprivation can also exacerbate the risk of MetS. People who exercise moderately for less than 150 minutes per week have a visceral fat accumulation rate 1.8 times that of people who exercise regularly. Long-term sleep deprivation can further regulate the expression of fat synthesis-

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related genes and accelerate the occurrence of obesity and metabolic disorders [8]. At the molecular level, excessive intake of long-chain saturated fatty acids can induce mitochondrial dysfunction by activating Toll-like receptor 4, regulating peroxisome proliferator-activated receptor-γ, sphingolipid remodeling and protein kinase C activation, which is also a key mechanism for the formation of IR [7].

In addition, genetic factors can influence the occurrence of typical MetS symptoms such as central obesity and abnormal blood sugar by regulating the insulin signaling pathway and the expression of lipid metabolism-related genes, further increasing the complexity of the pathological mechanism [8]; while the active ingredients in food and medicine homologous substances can reverse metabolic disorders by targeting these signaling pathways [9]. Existing studies have confirmed that food and medicine homologous substances such as kudzu root, hawthorn, yam, and Pu-erh tea can regulate MetS-related metabolic indicators through single application or synergistic use of multiple substances. Moreover, the intervention effect of synergistic use of multiple substances is significantly better than that of single substance intervention, and they have shown good application potential in blood glucose and blood lipid regulation [10].

In terms of single application, yam porridge can effectively reduce fasting blood glucose levels in patients. A clinical trial involving 90 patients with T2DM showed that after 8 weeks of intervention, there was no statistically significant difference in blood glucose levels between the experimental group treated with yam porridge and the control group treated with conventional intervention ($P>0.05$); while after 12 weeks of intervention, the fasting blood glucose in the experimental group was significantly lower than that in the control group. PP analysis showed that the fasting blood glucose in the experimental group decreased to 7.092 mmol/L, while that in the control group was 8.013 mmol/L ($t=-2.256$, $P=0.027$), and ITT analysis showed that the fasting blood glucose in the experimental group decreased to 7.154 mmol/L, while that in the control group was 8.235 mmol/L ($t=-2.737$, $P=0.007$) [11]. This result suggests that the fasting hypoglycemic effect of yam porridge alone is significant, but it has obvious time dependence, and its potential synergistic hypoglycemic effect when used in combination with other food and medicine homologous substances still needs further research.

In terms of compound application, the compound combination of kudzu root, hawthorn and Pu'er tea showed a significant synergistic effect in lipid metabolism regulation and had good potential to assist in lowering blood lipids. Related animal experiments compounded kudzu root and hawthorn at a mass ratio of 2:1, and then combined them with Pu'er tea extract, crude tea polysaccharide extract and crude tea polyphenol extract to prepare three complexes. A 4-week intervention experiment was carried out on rats with high blood lipids induced by a high-fat diet. The results showed that the tea polysaccharide compound group had the best lipid regulation effect, which could reduce the total cholesterol (TC) in rat serum from 5.18 mmol/L to 3.29 mmol/L and the triglyceride (TG) from 1.77 mmol/L to 1.08 mmol/L, while significantly inhibiting the abnormal increase of low-density lipoprotein cholesterol (LDL-C) [10]. Compared with the single application of food and medicine homologous substances, the compound application formula of kudzu root, hawthorn and Pu'er tea showed a more comprehensive targeted regulatory effect, and the regulatory effect did not show obvious time dependence, suggesting that the synergistic combination of multiple substances has an enhancing effect on the intervention.

At present, the application of food-medicine homology substances in MetS nutritional intervention has gradually extended from basic research to clinical practice and functional food development, and has shown good practical effects in multiple application scenarios [8]. However, overall, the application of food-medicine homology substances is still in the initial exploration stage,

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and a standardized application plan has not yet been formed. The intervention effects in different scenarios also vary to some extent, and further optimization and verification are still needed.

3. Mechanism of action of food-medicine homologous substances in the intervention of MetS

The intervention of food-medicine homologous substances on MetS works through four major pathways: improving insulin sensitivity, regulating lipid metabolism, inhibiting chronic inflammatory response, and regulating gut microbiota structure. The active ingredients of different food-medicine homologous substances can combine through common and specific mechanisms to achieve synergistic regulation of MetS.

3.1. Improve insulin sensitivity

The active ingredients such as polysaccharides, flavonoids, and amino acids in food-medicine homologous substances can improve insulin sensitivity, which is the core mechanism by which food-medicine homologous substances intervene in MetS glucose metabolism disorders.

Dioscorea polysaccharide in yam and puerarin in kudzu root can directly protect pancreatic β cells, reduce their apoptosis and promote insulin synthesis and secretion, and increase the effective concentration of peripheral blood insulin; amino acid combination can specifically promote the secretion of glucagon-like peptide-1 (GLP-1) by mouse intestinal STC-1 cells, delay gastric emptying and enhance the blood glucose regulation effect of insulin; the upregulation of specific miRNAs such as miR-5588-5p and miR-125b-2-3p and the downregulation of miR-496 are specific molecular characteristics of T2DM, which can provide a genetic basis for targeted intervention of MetS glucose metabolism; mitochondrial AAA protease Lon (Lonp1) is closely related to mitochondrial dysfunction in MetS, and the active ingredients of food and medicine can target and regulate Lonp1 to repair mitochondrial function, while mitochondrial dysfunction is a key cause of reduced responsiveness of insulin target organs such as skeletal muscle and liver [12,13].

After 12 weeks of intervention with yam porridge, PP analysis showed that the fasting blood glucose level in the experimental group decreased to 7.092 mmol/L and ITT analysis showed that it decreased to 7.154 mmol/L, which was significantly lower than that in the control group, confirming that the food-medicine homology substance can achieve the hypoglycemic effect by improving insulin sensitivity [11]. Subsequent analysis showed that the hypoglycemic effect of yam porridge was significantly time-dependent, and there was no significant effect after 8 weeks of intervention, suggesting that the improvement of insulin sensitivity by food-medicine homology substance is a slow and continuous process. Its active ingredients need to accumulate in the body for a long time to gradually repair the damaged insulin regulatory pathway, which is significantly different from the rapid hypoglycemic effect of chemical drugs. In the future, mechanism verification experiments can be carried out on these molecular targets to clarify the specific molecular pathways for improving insulin sensitivity, and provide a more direct theoretical basis for precise clinical intervention.

3.2. Regulation of lipid metabolism

Food and medicine homologous substances can regulate lipid metabolism by modulating key signaling pathways, achieving comprehensive regulation through "inhibiting lipid synthesis, promoting lipid decomposition, and accelerating lipid clearance," thereby reducing abnormal lipid deposition in the body and improving MetS lipid metabolism disorders and obesity characteristics.

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Volatile oils, flavonoids, triterpenic acids, and tea polysaccharides in cinnamon bark, kudzu root, hawthorn, and Pu-erh tea are core substances for regulating lipid metabolism.

Cinnamon bark (CC) can exert its effects through a multi-target mechanism. Its components such as CA, CAL, and polyphenols can activate the PPARγ and AKT pathways to promote insulin secretion. It can also activate PPARδ, PPARγ, and RXR through Procyanidin C1 and CAL, and enhance GLUT4 translocation to improve insulin sensitivity. At the same time, it can protect pancreatic β cells, activate the Nrf2/HO-1 pathway to reduce ROS and MDA for antioxidant purposes, regulate Bax and Caspase-3 to resist apoptosis, activate the AMPK/mTOR/ULK1 pathway to promote autophagy, and improve glucose metabolism disorders [12].

Among them, the volatile oil CAL extracted from cinnamon bark can stimulate the expression of fibroblast growth factor 21 (FGF21), upregulate the levels of peroxisome proliferator-activated receptor-γ coactivator-1α (PGC-1α) and UCP1 in brown adipose tissue (BAT), and increase the release of heat energy to improve obesity; at the same time, it inhibits the expression of sterol regulatory element binding protein-1 (SREBP-1) and mammalian target of rapamycin 1 (mTORC1), and reduces de novo fatty acid synthesis and fat accumulation [12]. In addition, puerarin and total triterpenic acids from hawthorn can jointly inhibit HMGCR activity, block key steps in cholesterol synthesis, and activate the LDL receptor pathway to promote the clearance of low-density lipoprotein cholesterol; tea polysaccharides in Pu-erh tea can reduce the activity of enzymes related to liver triglyceride synthesis, reduce liver fat accumulation, and alleviate fatty liver formation.

A complex of kudzu root and hawthorn in a 2:1 ratio, combined with tea polysaccharides from Pu-erh tea, was used to treat hyperlipidemic rats induced by a high-fat diet for 4 weeks. The rats' serum total cholesterol decreased from 5.18 mmol/L to 3.29 mmol/L and triglycerides decreased from 1.77 mmol/L to 1.08 mmol/L. At the same time, it significantly inhibited the abnormal increase in low-density lipoprotein cholesterol (LDL-C) levels [10]. This animal experiment confirmed the synergistic lipid-regulating effect of multiple food-medicine homologous substances, which was significantly better than single-substance intervention. The core reason is that the active ingredients of different substances can target different links in lipid metabolism and form a regulatory closed loop. However, this experiment only carried out a short-term intervention of 4 weeks, which could not verify the sustainability of its lipid-regulating effect. The intervention period could be increased and the mechanism verified in the future to provide more sufficient evidence for clinical application.

3.3. Anti-inflammatory effects

Inflammation is considered a key factor in metabolic abnormalities and plays an important role in the development and progression of metabolic syndrome. Many studies have confirmed that the levels of inflammatory markers such as C-reactive protein (CRP), tumor necrosis factor α (TNF-α), and interleukin-6 (IL-6) are significantly elevated in patients with MetS. These markers are produced by adipocytes, hepatocytes, and immune cells, and their overexpression is closely related to insulin resistance and oxidative stress [14]. Food-medicine homologous substances can block MetS-related chronic low-grade inflammatory responses, downregulate the expression of pro-inflammatory factors, and alleviate the interference of inflammatory factors on metabolic regulation, laying an important foundation for improving insulin resistance and regulating lipid metabolism. This is a key link in their multi-target intervention.

Flavonoids, polysaccharides, and polyphenols in food-medicine homologous substances are the core substances that exert anti-inflammatory effects. Puerarin in kudzu root and total flavonoids in hawthorn can inhibit the nuclear translocation of the NF-κB signaling pathway, block the transcription and release of pro-inflammatory factors from the source, significantly reduce the serum

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levels of TNF-α, IL-6, and IL-1β, and alleviate the inflammatory infiltration of adipose tissue and liver. Tea polysaccharides in Pu-erh tea can inhibit the production of the inflammatory mediator NO, reduce the recruitment of macrophages in adipose tissue, reduce the inflammatory damage to insulin target organs, and indirectly improve insulin sensitivity. Polygonatum polysaccharides and Polygonatum polysaccharides in Xiaoke Yin compound can synergistically downregulate the TNF/IL-6 pathway and at the same time alleviate the damage of inflammation to pancreatic β cells, achieving the dual effects of anti-inflammatory and insulin secretion protection [5]. In addition, the synthesis of pro-inflammatory molecules such as monocyte chemotactic protein I and IL-1 induced by uric acid can be significantly inhibited by the anti-inflammatory effects of food-medicine homologous substances, further blocking the vicious cycle of inflammation and metabolic disorders.

Based on network pharmacology and molecular docking studies, it has been confirmed that the Xiaoke Yin compound, composed of nine medicinal and edible herbs such as Polygonatum sibiricum, Dioscorea opposita, and Polygonatum odoratum, can significantly inhibit the body's inflammatory response by downregulating the TNF/IL-6 pathway, and at the same time achieve the dual effects of anti-inflammatory and cholesterol metabolism regulation [5]. The anti-inflammatory effect of the Xiaoke Yin compound reflects the advantage of the synergistic anti-inflammatory effect of multiple medicinal and edible ingredients. Its anti-inflammatory effect is coupled with its metabolic regulation effect, directly confirms the intrinsic relationship between "anti-inflammatory and metabolic improvement", and also explains why medicinal and edible substances can simultaneously intervene in multiple pathological features of MetS.

3.4. Regulation of gut microbiota structure

Food and medicine homologous substances can indirectly improve the glucose and lipid metabolism disorder and chronic inflammatory response of MetS by reshaping the gut microecological balance and repairing the intestinal mucosal barrier, and by regulating host metabolism through the "gut-liver axis" and "gut-fat axis". This is an important indirect mechanism for their intervention in MetS. Glucose and lipid metabolism disorder and oxidative stress can induce gut microbiota dysbiosis, and gut microbiota imbalance will accelerate the progression of MetS, forming a vicious cycle [15]. Gut microbiota dysbiosis is related to the pathogenesis and development of a variety of chronic low-grade inflammatory diseases. After the typical butyrate-producing gut microbiota is altered, its function of protecting the gut from inflammation and promoting gut integrity by producing short-chain fatty acid (SCFA) butyrate will be affected [15].

Polysaccharides and pectin components in food-medicine homologous substances cannot be digested and absorbed by the human body, but can serve as prebiotics for beneficial intestinal bacteria, regulating the abundance and diversity of the gut microbiota. Yam polysaccharides, Pu-erh tea polyphenols, and hawthorn pectin can significantly increase the relative abundance of beneficial bacteria such as Bifidobacteria, Lactobacillus, Akkermansia, and B. dorei in the gut, reduce the Firmicutes/Bacteroidetes ratio, and decrease the number of harmful bacteria such as Enterobacteriaceae [15,16]. Increased abundance of beneficial bacteria can promote the synthesis of short-chain fatty acids (SCFAs). SCFAs can activate GPR43/GPR41 receptors, regulate hepatic lipid metabolism and adipose tissue thermogenesis, and at the same time promote the repair of the intestinal mucosal barrier, prevent endotoxins from entering the bloodstream caused by intestinal leakage, and avoid endotoxin-induced systemic chronic inflammatory response. In addition, berberine, a substance that is both food and medicine, can improve metabolic abnormalities, enhance bioavailability by regulating the gut microbiota, strengthen the intestinal barrier function, and alleviate metabolic endotoxemia. Its mechanism of action includes inducing the death of harmful

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intestinal bacterial cells, increasing the number of SCFA-producing bacteria and BA-degrading bacteria, and reducing the abundance of LPS-producing bacteria [16].

Intervention experiments of Pu-erh tea extract on rats on a high-fat diet have shown that it can increase the relative abundance of Bifidobacteria in the rat gut by more than 30%, reduce the Firmicutes/Bacteroidetes ratio by 25%, and at the same time, the blood glucose and blood lipid levels of rats show a significant decrease along with the improvement of the gut microbiota structure [8]. Another study showed that B. dorei can reconstruct the gut microbiome composition of MetS model animals by enhancing early interferon expression and regulating the balance of pro-inflammatory or anti-inflammatory cytokines [15]. The above experiments directly confirmed the mechanism of action of food-medicine homologous substances in "regulating gut microbiota-improving metabolism", suggesting that gut microbiota is an important target of food-medicine homologous substances in intervention of MetS, and also provides a supplementary explanation for its "multi-target, multi-pathway" intervention characteristics. However, most of the relevant studies are currently focused on animal experiments, and there are few clinical studies on MetS patients. Furthermore, the specificity of different food-medicine homologous substances in regulating gut microbiota has not been clarified, such as whether there are differences in the target of yam polysaccharide and Pu-erh tea polyphenols in regulating beneficial bacteria. Further clinical studies can be conducted to explore the effects of food-medicine homologous substances on the gut microbiota of MetS patients, and to determine the optimal dosage and intervention period for regulating the microbiota. In addition, the regulatory mechanisms of microbiota metabolites (such as SCFA) on host metabolism can be studied in depth by combining the "gut-liver axis" and "gut-fat axis", so as to provide a more complete theoretical system for the intervention of food-medicine homologous substances on MetS through the gut microbiota.

4. Clinical advantages and existing issues of nutritional intervention based on food and medicine homologous to dietary products

Compared with traditional chemical drug interventions such as metformin and statins, interventions using food and medicine homologous to dietary products have significant advantages in terms of safety and long-term applicability. A 12-week yam porridge intervention trial successfully improved blood glucose levels in patients with type 2 diabetes without any adverse event reports.

Furthermore, food and medicine homologous to dietary products possess both edible and medicinal properties; their active ingredients, while exerting their interventional effects, can also supplement the body's nutritional needs. For example, dioscorea polysaccharides in yam are not only the core active ingredient for improving insulin sensitivity but also a necessary polysaccharide nutrient; tea polysaccharides in Pu-erh tea, while effectively regulating lipid metabolism and reducing serum TC, TG, and LDL-C levels, can also serve as a source of dietary carbohydrates.

The pathological mechanism of MetS is centered on insulin resistance, forming a vicious cycle of mutually reinforcing glucose and lipid metabolism disorders, obesity, chronic inflammation, and gut microbiota imbalance. Food and medicine homologous substances can exert multi-target synergistic effects through four major pathways: improving insulin sensitivity, regulating lipid metabolism, inhibiting chronic inflammation, and regulating gut microbiota. This can simultaneously improve various symptoms of metabolic syndrome, which is consistent with the pathological characteristics of multi-system and multi-target metabolic disorders in this disease.

However, the mechanisms of action of most food-medicine homologous substances remain unclear, and related research mostly focuses on the macroscopic effects of single substances, lacking molecular-level studies. For example, in the study of the combined application of yam

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polysaccharide and Pu-erh tea, the molecular pathways by which they improve MetS-related metabolic indicators are still unclear, failing to explain the core mechanism behind the apparent superiority of combined intervention over single intervention. Furthermore, the theoretical research on food-medicine homologous intervention lacks integration with clinical practice, making it difficult to translate research results into clinical applications. Existing clinical studies often suffer from small sample sizes and short intervention periods, further hindering the development and application of food-medicine homologous substances in MetS intervention.

5. Conclusion

MetS, as a major public health challenge, urgently requires safe and sustainable intervention strategies for its long-term management. Diet-based nutritional intervention, leveraging its naturalness, safety, and multi-target synergistic advantages, has demonstrated significant potential in regulating glucose and lipid metabolism, improving IR, inhibiting inflammatory responses, and restoring gut microbiota balance in patients with MetS. Extensive experimental and clinical evidence has been accumulated for both single substances and compound formulations. However, this field still faces core issues such as the lack of standards for compound interventions, insufficient long-term data, a scarcity of personalized solutions, and delayed clinical translation, which limit its large-scale clinical application.

In the future, by establishing a standardized composite intervention system, conducting high-quality long-term clinical research, developing stratified personalized plans, and deepening mechanism research and multidisciplinary translational cooperation, it is expected to promote the development of dietary and medicinal homology nutritional intervention towards precision and standardization. With the continuous deepening of research, the concept of dietary and medicinal homology will be further integrated into the prevention and treatment system of MetS, providing stronger theoretical support and more practical solutions for non-pharmacological intervention of MetS, helping to improve long-term patient outcomes and reduce the burden on public health.

Author contribution

All the authors contributed equally and their names were listed in alphabetical order.

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