Mechanisms of Curcumin in Metabolic Syndrome via Oxidative Stress Regulation: A Review

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ABSTRACT

Metabolic syndrome (MetS) is a group of related problems in our body, centering on insulin resistance. This is a great challenge to the health of the whole world. Oxidative stress, which occurs when there are too many reactive oxygen species (ROS) and our antioxidant defense can't work normally, is a very important point to promote MetS. Curcumin (Cur), a lipophilic polyphenol from the root stem of long ginger, is very interesting because of its properties, which act on several targets. This review explains how curcumin regulates oxidative stress to improve MetS. This paper mainly discusses the signal pathway of antioxidant axis Keap1/Nrf2/ARE, SIRT1, and the communication between antioxidant and anti-inflammatory networks. Curcumin activates Nrf2 by changing the important cysteine residue on Keap1. This leads to more phase II detoxification enzymes, such as superoxide dismutase (SOD), catalase (CAT) and heme oxygenase -1(HO-1). Together, these enzymes help restore the redox balance in cells. In addition, other mechanisms have been found, such as SIRT1 signal axis, post-transcriptional regulation of microRNAs (miRNAs) and mitochondrial bioenergy. These findings indicate that curcumin has many therapeutic potentials. This review accurately explains how curcumin regulates oxidative stress. This provides a solid theoretical basis for developing curcumin as a dietary intervention strategy and a supplementary therapeutic agent for MetS.

Keywords: Curcumin; Oxidative Stress; Nrf2; Metabolic Syndrome; SIRT1; Mechanism of Action.

1. INTRODUCTION

Globally, metabolic syndrome (MetS), a clinical constellation centered on insulin resistance, has emerged as a major public health challenge contributing to the development of cardiovascular disease and type 2 diabetes mellitus. MetS is characterized by central obesity, insulin resistance, dysregulated glucose and lipid metabolism, and hypertension. With the change of lifestyle, its prevalence rate is getting higher and higher, affecting younger and younger people. It affects many organs, such as the liver and heart, and is usually accompanied by chronic low-intensity systemic inflammation [1]. Recent research shows that oxidative stress is a very important link in promoting MetS: when there are too many reactive oxygen species (ROS) and the system that protects us from them cannot work normally, it will destroy the redox balance in cells. This will lead to a series of problems: signal defect, lipid metabolism dysfunction and chronic low-intensity inflammation. In the development of MetS, oxidative stress occupies a central position in the pathophysiological network; This means that when cells are injured, the production of ROS becomes stronger than the body's ability to eliminate them, thus making the redox body unbalanced. Under the conditions of hyperglycemic and hyperlipidemic, the overload of mitochondria triggers ROS outbreak. These explosions directly damaged protein and DNA. At the same time, they act as molecules that send signals. These signals interfere with the phosphorylation of IRS-1, leading to insulin resistance [13]. In addition, oxidative stress has a positive feedback loop with pro-inflammatory pathways such as NF-κ B. This aggravates the chronic metabolic damage of multiple organs.

At present, there are not many comprehensive strategies aimed at multiple problems in clinic, so it is very important to find natural bioactive compounds that can regulate multiple goals. Curcumin (Cur) is a lipophilic polyphenol extracted from the rhizome of Curcuma longue. The hydroxyl and methoxy groups in its chemical structure enable it to directly neutralize free radicals; More importantly, it can activate internal signal pathways, such as Nrf2/ARE, to produce antioxidant enzymes, such as SOD, CAT and GSH-Px. This enhances the defense from the source [4-5,13] cells. Tian et al. pointed out in their comments that curcumin has the mechanism of anti-oxidation and anti-inflammation, which has been widely recognized by regulating several pathways related to inflammation, such as Nrf2/ARE [12]. Although curcumin has poor solubility in water and low bioavailability, the progress of nano-delivery and complex technology (such as GAA/SSPS) is helpful to gradually overcome these problems in clinical use [2]. However, the exact mechanism of curcumin changing

oxidative stress network to improve glucose and lipid problems at the same time remains to be understood. Therefore, this review brings together the latest experimental evidence to analyze how curcumin acts on important pathways such as Nrf2/ARE, SIRT1 and NF-κ B. This shows how curcumin can restore the redox balance to repair several pathological aspects of MetS, thus providing a good theoretical basis for creating a natural strategy that works on multiple goals.

2. THEORETICAL BASIS OF CURCUMIN AND OXIDATIVE STRESS REGULATION

2.1 Pathological Association Between Metabolic Syndrome and Oxidative Stress

Metabolic syndrome (MetS) is not a single disease, but a systemic disease caused by obesity, insulin resistance and lipid metabolism. In this process, the imbalance of oxidative stress is considered as the main driving force to start and accelerate the progress of the disease.

Insulin resistance is the core pathological part of MetS. Under normal physiological conditions, insulin activates PI3K/Akt pathway to regulate blood sugar. However, because blood contains too much sugar and fat, excessive ROS produced by active mitochondria such as JNK and IKKβ will lead to abnormal phosphorylation of serum on IRS-1. This signal blocking not only prevents peripheral tissues from absorbing glucose, but also causes compensatory hyperinsulinemia, which in turn stimulates the production of ROS, thus forming a vicious circle of "oxidative stress-insulin resistance".

Zhao et al. showed that curcumin can significantly reduce the level of ROS in hepatocytes, increase the phosphorylation of IRS-1 by inducing nuclear translocation of Nrf2, and effectively reverse insulin resistance induced by oxidative stress [13]. The destructive power of oxidative stress lies in its "nonspecific" cascade effect, which shows strong specificity according to MetS tissues and subtypes. Oxidative stress leads to endothelial nitric oxide synthase (eNOS) detachment, which leads to vascular hypertension and stiffness. In adipocytes, oxidative stress reduces the secretion of adiponectin and promotes the release of pro-inflammatory cytokines such as TNF-α and IL-6, thus creating a chronic inflammatory environment in the whole body. The sudden and sharp increase of ROS in hyperglycemia leads to the depolarization of mitochondrial membrane potential in myocardial cells, which leads to the opening of mitochondrial permeability transition hole and finally triggers myocardial cells [4,11]. Xie and Al. In their review, it is pointed out that SIRT1, an important intracellular regulatory protein, plays a vital role in reducing oxidative stress in myocardial cells, maintaining mitochondrial function and inhibiting inflammatory response, and is a potential therapeutic target for diabetic cardiomyopathy [10].

2.2 Chemical Structure and Antioxidant Mechanisms of Curcumin

Curcumin molecule has two n-methylphenol rings, which are connected by β -diketone group. This unique chemical structure gives it dual antioxidant capacity: on the one hand, phenolic hydroxyl groups can generate hydrogen atoms to neutralize superoxide radicals and hydroxyl radicals; On the other hand, the hydrogen atom on the active methyl group can also be removed, thus preventing the chain reaction of lipid peroxidation. Unlike synthetic antioxidants, curcumin has the advantage that it can "wake up" and repair our body's system. It is not only a chemical trap, but also a good "signal modulator". Curcumin can change the important cysteine residue on protein Keap1, thus releasing Nrf2, thus activating endogenous antioxidant reactive element (ARE). This process leads to several second-stage detoxification enzymes, such as catalase (CAT), superoxide dismutase (SOD) and heme oxygenase -1 (HO-1), resulting in [4-5,8,13]. Tian and al. In their review, they pointed out that the Nrf2/HO-1 axis is the main way for curcumin to exert its antioxidant effect, and the strong expression of HO-1 can not only eliminate free radicals, but also provide various protections, such as anti-inflammatory and antibacterial activities [12]. In addition, curcumin shows the ability to act on a variety of metabolic related pathways; For example, by activating AMPK pathway to promote the oxidation of fatty acids and inhibiting NF-κB pathway to prevent the inflammatory chain reaction, it is helpful to restore the metabolic balance of the whole body.

2.3 Structural Advantages of Curcumin and Its Derivatives

In addition to curcumin itself, its natural analogues, such as demethoxycurcumin (DMC) and tetrahydrocurcumin (THC), also show unique advantages in regulating metabolism. Studies have shown that DMC has a very strong affinity for molecular docking and targeting protein Keap1, and its effect of protecting liver and reducing oxidative stress in some models may even be as good as the best antioxidant drugs used in clinic [3]. Li et al. found that tetrahydrocurcumin greatly reduced the oxidative stress and fibrosis caused by high glucose level, and improved the cardiac function of diabetic cardiomyopathy rats by activating SIRT1 pathway [11].

3. MECHANISMS OF CURCUMIN IN MODULATING OXIDATIVE STRESS AGAINST METABOLIC SYNDROME

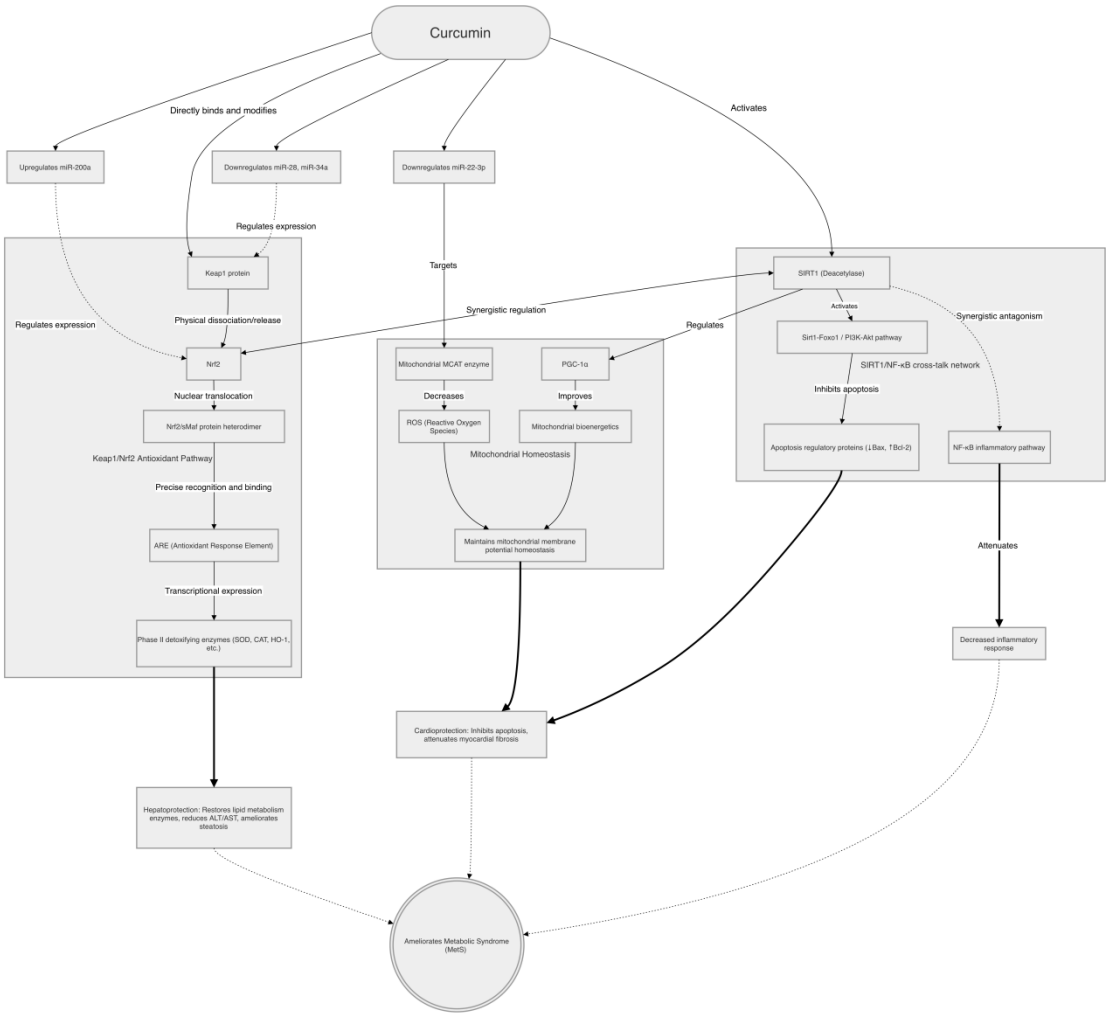

Figure 1. Mechanisms of Curcumin in Modulating Oxidative Stress Against Metabolic Syndrome.

3.1 Potential Activation of the Keap1/Nrf2 Pathway and Hepatoprotection

The core of curcumin intervention in MetS lies in strengthening endogenous redox buffering capacity. Curcumin and its derivatives (e.g., DMC), by virtue of their specific reactive functional groups, can directly bind to and modify key sites on the Keap1 protein, inducing conformational changes that promote the physical dissociation of Keap1 from Nrf2, thereby releasing Nrf2 from its cytoplasmic sequestration. The liberated Nrf2 accumulates and translocates into the nucleus, where it forms a heterodimer with small Maf proteins to recognize ARE sequences and initiate the transcription of phase II detoxifying enzymes including SOD, CAT, and HO-1 [4,13]. Regarding hepatoprotection, Gu et al. demonstrated that curcumin can significantly activate the Nrf2 pathway in the livers of high-fat diet-fed mice, restore lipid metabolism enzyme activity, markedly reduce serum ALT and AST levels, and ameliorate hepatic steatosis [5]. Wang et al. further confirmed that DMC significantly attenuated drug-induced liver injury by targeting the Keap1/Nrf2 interaction; given the high conservation of the Keap1/Nrf2 pathway in hepatic lipotoxic stress, this mechanism also provides a rational theoretical basis for explaining curcumin's intervention in MetS-associated fatty liver pathology [3]. Nano-carrier-encapsulated curcumin also significantly upregulated Nrf2 expression in liver injury models, demonstrating the efficient Nrf2 pathway activation capacity of novel delivery systems and providing a highly promising drug delivery strategy reference for the targeted treatment of MetS-related chronic low-grade hepatic injury [8].

3.2 SIRT1 Signaling Axis and Cardioprotective Mechanisms

Beyond the Nrf2 pathway, the SIRT1 signaling axis represents another core pathway through which curcumin regulates oxidative stress. In their review, Xie et al. systematically pointed out that SIRT1, as an important deacetylase, plays an important role in reducing oxidative stress of myocardial cells, maintaining mitochondrial balance, reducing endoplasmic reticulum stress and inhibiting the activation of renal-angiotensin-aldosterone system. They indicate that this is a potential therapeutic target for diabetic cardiomyopathy [10]. Ren et al. found that curcumin can significantly reduce oxidative stress, and inhibit myocardial cell death in diabetic cardiomyopathy through Sirt1-Foxo1 and PI3K-Akt signaling pathways. This is manifested in reducing Bax, increasing Bcl-2 expression and maintaining the stability of mitochondrial membrane potential [9]. Li et al. confirmed that tetrahydrocurcumin, as the main metabolite of curcumin, significantly reduced oxidative stress and myocardial fibrosis induced by high glucose by activating SIRT1 pathway. This reduces the levels of ROS and MDA in heart tissue and increases the activities of SOD and GSH-Px [11]. Kim and Al. It was found that lissartan and isoproxypyr can effectively reduce oxidative stress and inflammatory reaction in diabetic cardiomyopathy rats by coordinating and regulating SIRT1/Nrf2/NF-κB signaling pathway. This discovery shows that there is a close communication between SIRT1 and Nrf2, which provides a mechanical reference for the cardioprotective effect of turmeric through similar channels. The above research provides convincing evidence that turmeric protects METS from cardiac complications through SIRT1 signal axis with multilevel mechanical support.

3.3 miRNA-Mediated Post-Transcriptional Fine-Tuning and Mitochondrial Homeostasis

Curcumin can change the expression profile of microRNA in liver. It modifies the expression level of Nrf2/Keap1, reduces Mir space station 28 and Mir space station 34a, and increases Mir space station 200a. This breaks the oxidation imbalance [5]. Wang et al. found that curcumin targeted mitochondrial MCAT enzyme by reducing miR-22-3p to reduce ROS level and improve bioenergy metabolism without changing membrane potential. This happens in MetS at the subcellular level [7]. This discovery reveals a new mechanism: curcumin regulates oxidative stress through miRNA network. This provides an important molecular basis for understanding the role of curcumin in protecting mitochondrial function. It is worth noting that Xie et al. pointed out in their review that SIRT1 is closely related to mitochondrial function, and the activation of SIRT1 can improve mitochondrial biological energy by regulating PGC-1α. This provides a theoretical connection for explaining how curcumin regulates SIRT1 and mitochondrial balance [10].

3.4 Multi-Pathway Synergistic Regulation and Antioxidant-Anti-Inflammatory Cross-Talk

Curcumin has the advantage of “horizontal” regulation. When it activates Nrf2, curcumin will antagonize AhR/NF-κB/NLRP3 signal axis, thus preventing the release of pro-inflammatory cytokines such as TNF-α and IL-6. Chen et al. showed that curcumin could significantly block the AhR/NF-κB/NLRP3 signaling pathway in rats with acute periodontitis, thus reducing inflammatory tissue infiltration and bone absorption [6]. Wang et al. also confirmed that curcumin can effectively block the IL-6/JAK/STAT3 signaling pathway, thus greatly reducing the inflammatory response in acute stroke rats. Curcumin helps to activate Nrf2 and restore the balance of glucose and lipid metabolism by acting on PI3K/Akt and AMPK pathways. At the same time, curcumin can promote the oxidation of fatty acids by activating AMPK pathway, which is helpful to further improve glucose and lipid metabolism. Zhao et al. further confirmed that curcumin can effectively improve insulin resistance by inducing nuclear translocation of Nrf2, reducing MDA level and increasing GSH content in cells [13]. In addition, Jin et al. They found that all-isartan isoproxil can effectively reduce the oxidative stress and inflammatory reaction in diabetic cardiomyopathy rats. This is achieved through the coordinated adjustment of SIRT1/Nrf2/NF-κB signal paths. This indicates that the synergistic effect of antioxidant and anti-inflammatory of this pathway may be one of the important mechanisms of curcumin exerting multi-objective effects in the complex pathological network of MetS [14].

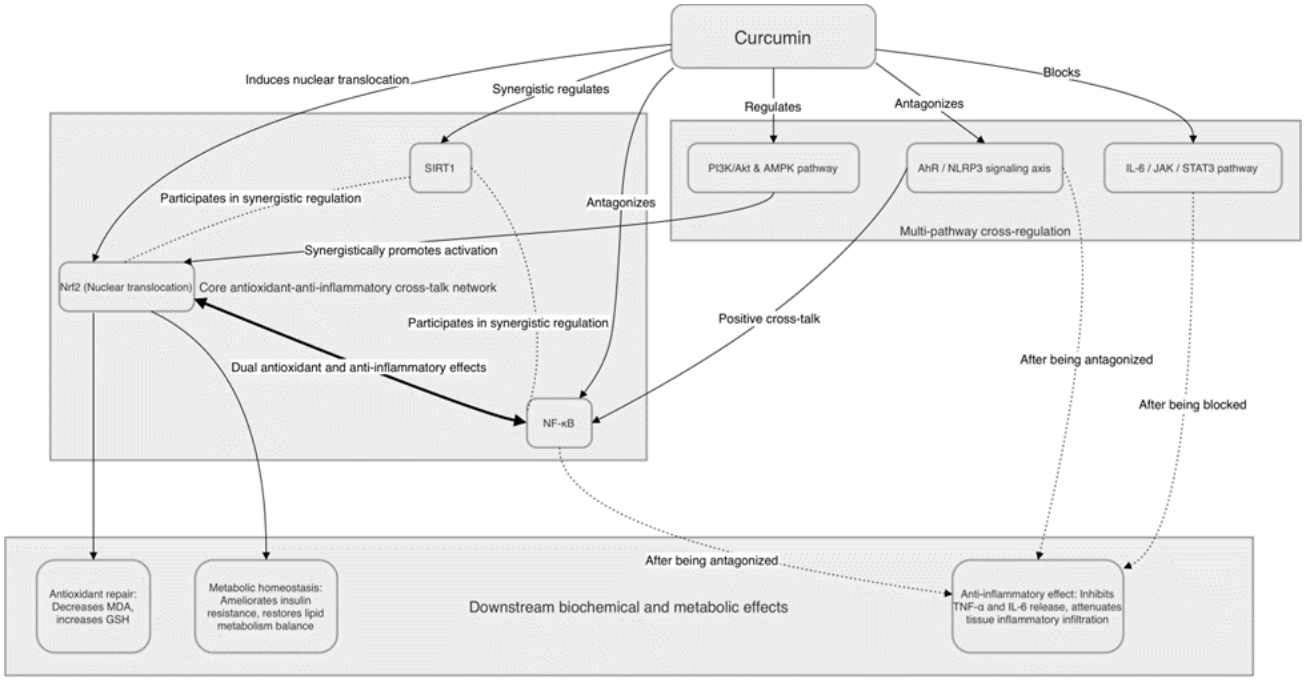

Figure 2. Multi-Pathway Synergistic Regulation and Antioxidant-Anti-Inflammatory Cross- Talk.

3.5 Bidirectional Redox Balance and Metabolic Homeostasis Reconstruction

Curcumin has created a system of "increasing income and reducing expenses": it realizes "increasing income" by increasing the activity of antioxidant enzymes and GSH level [5,7,12]; By greatly reducing the levels of damage markers such as MDA and 8-OHdG, the attack of ROS on lipids and DNA is completely prevented, thus "reducing the cost". For the regulation of glucose and lipid, curcumin promotes the phosphorylation and activation of AMPK, thus preventing the regulatory element binding protein -1c(SREBP-1c) and promoting the gene expression of fatty acid oxidation (PPARα, CPT1α) [5]; At the same time, it reconstructs glucose homeostasis through the bidirectional regulation of PPARγ. The evaluation of its effectiveness forms a complete cycle, from "molecular target (AMPK/Nrf2)-to histopathology (less lipid vacuoles on H&E staining)-to macroscopic manifestations (decrease of FBG, HOMA-IR and TG)" [4-5].

4. DIETARY INTERVENTION STRATEGIES AND COMBINATION THERAPIES

4.1 Dietary Intervention Strategies and Combination Regimens

Curcumin is a very safe natural compound, so it is beneficial for early intervention in MetS. Studies have shown that it can reduce blood sugar, improve insulin resistance [4], improve lipid spectrum, reduce fat in the liver [5], and control chronic low-level inflammation by helping the body maintain a good balance [1,6]. If you do "curcumin plus" treatment, it is even better: combining drugs such as dimethyl diester can have synergistic effect and reduce the side effects of conventional drugs; Combined with exercise therapy can help eliminate free radicals, protect mitochondria, and strengthen the impact on metabolism.

In dietary intervention, the way of giving curcumin is very important. Wang et al. showed that GAA/SSPS complex can greatly improve the stability and in vitro digestion and release characteristics of curcumin, which provides an important reference for the development of dietary supplements [2].

As for drug combination intervention, the synergistic effect of curcumin and dimethylbisphenol has been confirmed by many studies. Metformin, as the first treatment of MetS, mainly improves insulin sensitivity by activating AMPK pathway. Curcumin not only synergistically activates AMPK, but also enhances antioxidant defense through Nrf2 pathway, so their combination allows the dual intervention of "metabolic regulation-redox balance". In addition, the combination of curcumin and statins can improve the efficiency of reducing blood lipid and reduce the side effects of statins, such as myopathy. Tian et al. pointed out in their review that curcumin plays an antioxidant and anti-inflammatory role by

regulating several inflammation-related pathways, and its combination with conventional drugs has broad clinical prospects [12].

5. IN VITRO AND IN VIVO EXPERIMENTAL MODELS AND COMPREHENSIVE EVALUATION SYSTEMS

5.1 Model Validation of Targeted Intervention in Core Organs

Existing studies have extensively employed organ-specific models to objectively verify the intervention mechanisms of curcumin. In the liver, in vitro studies commonly utilize LO2/AML12 cells to induce ROS bursts [3,7], while in vivo models predominantly employ high-fat diet (HFD) to establish fatty liver models [5]; results confirmed that curcumin can significantly activate the Nrf2 pathway and restore lipid metabolism enzyme activity [3,5]. In the heart and pancreas, in vitro studies employ high glucose-induced H9c2 cells to observe mitochondrial membrane potential changes [4], while in vivo models predominantly use HFD combined with STZ to induce diabetic models; data indicate that curcumin can inhibit Caspase-3-mediated apoptosis and maintain myocardial and pancreatic islet function [4,10,12]. Regarding peripheral and systemic inflammation, studies based on inflammatory rat models confirmed that curcumin can downregulate pro-inflammatory pathways such as IL-6/JAK/STAT3 and significantly attenuate tissue inflammatory infiltration [1,6]. Furthermore, since MetS is frequently accompanied by systemic low-grade inflammation, the robust inhibitory evidence from the above extreme local inflammation rat models suggests that curcumin can not only intervene in systemic trunk inflammation but also holds potential protective value against common peripheral inflammatory complications of MetS [1,6].

5.2 Comprehensive Evaluation Indices and Objective Assessment

The research framework has established multi-dimensional quantitative criteria encompassing pharmacodynamics, metabolism, and molecular pathology to verify therapeutic efficacy. In vivo studies commonly employ dose gradients of 50–200 mg/kg to establish dose-response relationships [1] and verify multi-target advantages through comparison with positive control drugs (e.g., NAC, metformin) [3]. Following intervention, ROS, MDA, and 8-OHdG levels significantly decreased, while SOD and GSH-Px activities and the GSH/GSSG ratio markedly increased [5,7,12]; concurrently, blood glucose (FBG, HOMA-IR), lipid profiles, and liver function enzymes (ALT, AST, ALP) levels showed substantial improvement [3,5]. Significant decreases in serum inflammatory cytokines (e.g., IL-6) confirmed its safety profile [1,6]; H&E staining visually demonstrated the resolution of steatosis and inflammation [1,5]; Western blotting and RT-qPCR confirmed the precise regulation of core pathways including Nrf2, SIRT1, and NF-κB at the molecular level. All the above therapeutic effects achieved statistical significance ($P<0.05$) [1,6].

5.3 Bioavailability and Tissue-Specific Evaluation

Traditional curcumin suspensions suffer from low bioavailability, necessitating high-dose administration. In contrast, GAA/SSPS complexes or PDA nanoparticles can achieve high-fold activation of the Nrf2 pathway at low concentrations [2,8]. Wang et al. demonstrated that curcumin complexes can significantly improve the stability and in vitro digestive release characteristics of curcumin, providing a novel strategy for enhancing its bioavailability [2]. Evaluation criteria should incorporate “bioavailability-normalized” indices to assess the regulatory slopes of different drug delivery systems. In terms of tissue-specific evaluation, the liver should focus on the association between redox enzyme systems and lipid transport proteins [3,5]; the myocardium should focus on SIRT1 activity, mitochondrial membrane potential ($\Delta\Psi m$), and the Bcl-2/Bax ratio [4,10-12]; bone/periodontal tissues should focus on the RANKL/OPG ratio and osteoclast activity correlation, in order to comprehensively evaluate curcumin’s capacity to block the systemic metabolic-inflammatory network [6].

6. CONCLUSIONS

To sum up, existing studies have objectively indicated that curcumin may have “antioxidant and anti-inflammatory” effects. It does this at the molecular level by targeting the antioxidant axis Keap1-Nrf2/ARE, activating SIRT1 signaling pathway, and blocking pro-inflammatory pathways such as NF-κB and JAK/STAT3. This is helpful to reverse the problem of glucose and lipid regulation in metabolic syndrome (MetS) and protect important organs. In order to solve the problems of low water solubility and low bioavailability that hinder its clinical application, future research must focus on developing new targeted delivery systems, such as GAA/SSPS complex [2] or dopamine nanoparticles (PDA) [8], to overcome the absorption barrier. At the same time, more research is needed to understand the deeper mechanisms, such as miRNA [7], SIRT1/Nrf2 [11] interaction network and post-transcriptional adjustment of “good microbial-metabolic axis”. We also need

to accelerate the transition to high-quality randomized controlled trials (RCTs), and finally achieve accurate and personalized prevention and treatment of METS.

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