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Recent Advances in the Therapeutic Mechanisms and Potential of Baicalin in Atherosclerosis

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Abstract: ***Atherosclerosis is a chronic and progressive vascular disease underlying most cardiovascular and cerebrovascular events. Its pathogenesis involves a complex interplay of inflammation, oxidative stress, lipid metabolism disorders, and endothelial dysfunction. In recent years, increasing attention has been paid to natural compounds with multi-target therapeutic potential. Baicalin, a major flavonoid component derived from Scutellaria baicalensis, exhibits a wide range of pharmacological activities, including anti-inflammatory, antioxidant, lipid-regulating, and anti-platelet effects. Emerging evidence from in vitro and in vivo studies suggests that baicalin may exert protective effects against atherosclerosis through multiple signaling pathways, by modulating endothelial function, inhibiting plaque formation, and attenuating vascular inflammation. However, its clinical translation is still limited by the lack of standardized preparations, optimized formulations, and high-quality clinical trials. This review summarizes recent progress on the mechanisms and therapeutic potential of baicalin in the prevention and treatment of atherosclerosis, aiming to provide a scientific basis for its future development in cardiovascular medicine.***

Keywords: Baicalin, Atherosclerosis, Anti-inflammation, Oxidative stress, Traditional Chinese medicine, Cardiovascular disease.

1. Introduction

Cardiovascular diseases (CVDs) remain the leading cause of mortality worldwide, accounting for approximately 19.2 million deaths annually [1]. In China, the mortality rate exceeding 45%, posing a critical threat to public health [2]. Atherosclerosis (AS) serves as the fundamental pathological substrate for a wide spectrum of cardiovascular and cerebrovascular events. The progression of AS is a highly complex, chronic inflammatory process characterized by lipid deposition, maladaptive immune responses, and profound vascular endothelial dysfunction [3]. Currently, the clinical management of AS predominantly relies on the aggressive control of established risk factors. Standard pharmacological regimens include statins for lipid modulation, antiplatelet agents for thrombosis prevention, and angiotensin-converting enzyme inhibitors for blood pressure control [4-8]. Recently, targeted strategies such as PCSK9 inhibitors and RNA interference technologies have expanded the therapeutic armamentarium [9]. However, these therapies primarily focus on mitigating the downstream consequences of disease progression and often lack precision in intervening during the critical initiating events of AS, particularly early-stage endothelial dysfunction. Therefore, exploring novel molecular targets and intervention strategies from the perspective of disease pathogenesis is of paramount importance.

Traditional Chinese Medicine has accumulated extensive clinical experience in the prevention and treatment of AS, offering profound insights for modern drug discovery. Baicalin, the predominant bioactive flavonoid extracted from the root of Scutellaria baicalensis Georgi, possesses a broad spectrum of pharmacological properties, including potent anti-inflammatory, antibacterial, and antioxidant activities [10]. It has demonstrated remarkable protective efficacy across cardiovascular, hepatic, and oncological diseases [11-13]. Accumulating evidence confirms that baicalin can significantly attenuate the progression of AS through the modulation of multiple signaling cascades. This review systematically summarizes the current research progress on baicalin in AS, focusing on its mechanistic roles in mitigating inflammatory responses, oxidative stress, lipid dysmetabolism, and endothelial dysfunction, while discussing its clinical translational value.

2. Anti-Inflammatory Mechanisms of Baicalin

2.1 Inhibition of the NF-κB Signaling Pathway

Nuclear factor kappa-B (NF-κB) functions as a master transcriptional regulator orchestrating vascular inflammatory responses. Upon exposure to atherogenic stimuli, endothelial cells initiate a signaling cascade via Toll-like receptors (TLRs), triggering the TLR4-MyD88 pathway. This leads to the degradation of IκB and the release of the NF-κB dimer, exposing its p65 subunit. The liberated NF-κB p65 translocates into the nucleus, inducing the robust expression of pro-inflammatory cytokines and driving the inflammatory cascade [14]. Extensive research has demonstrated that baicalin effectively suppresses the nuclear translocation of the NF-κB p65 subunit in vascular endothelial cells. By downregulating the expression of downstream pro-inflammatory mediators, baicalin significantly alleviates local vascular inflammation and retards the progression of atherosclerotic plaques [15].

2.2 Suppression of NLRP3 Inflammasome Activation

The aberrant activation of the NLRP3 inflammasome plays a pivotal role in determining the vulnerability of AS plaques. Its activation relies on a "two-signal" pathway: a priming signal via NF-κB to induce NLRP3 and precursor cytokines, and an activation signal triggered by potassium efflux, reactive oxygen species (ROS), or mitochondrial damage [16]. This assembly activates Caspase-1, which cleaves pro-IL-1β and pro-IL-18 into their mature forms [17, 18]. Furthermore, activated Caspase-1 cleaves Gasdermin D, inducing

pyroptosis—a highly inflammatory form of programmed cell death that causes cell lysis and the massive release of intracellular inflammatory contents [19]. Studies elucidate that baicalin effectively impedes the assembly of the NLRP3 inflammasome by inhibiting ROS overproduction and preventing potassium efflux. Consequently, baicalin diminishes the secretion of IL-18 and IL-1β, exerting a profound inhibitory effect on intravascular inflammation [20].

2.3 Promotion of M2 Macrophage Polarization

The phenotypic transition of macrophages dictates the trajectory of the AS inflammatory response. M1 macrophages, activated by stimuli like LPS or IFN-γ, secrete copious amounts of pro-inflammatory cytokines (e.g., IL-1β, TNF-α, IL-6), exacerbating endothelial injury and accelerating foam cell formation. Conversely, M2 macrophages, activated by IL-4 and IL-13, secrete anti-inflammatory mediators like IL-10 and TGF-β, which are crucial for resolving inflammation, promoting tissue repair, and enhancing plaque stability [21].

Recent investigations reveal that baicalin acts as a potent immunomodulator, effectively driving the phenotypic polarization of macrophages from the M1 to the M2 state. This transition significantly ameliorates the local immune microenvironment, contributing to the stabilization of atherosclerotic lesions [22].

3. Antioxidant Effects and Regulation of Lipid Metabolism

3.1 Activation of the Nrf2/HO-1 Antioxidant Pathway

The disruption of the balance between oxidative stress and endogenous antioxidant defense is a core driving factor for vascular endothelial injury. Nuclear factor erythroid 2-related factor 2 (Nrf2) is the central transcription factor governing the cellular adaptive response to oxidative stress. Under oxidative conditions, activated Nrf2 dissociates from Keap1, translocates into the nucleus, and binds to antioxidant response elements (ARE). This initiates the transcription of cytoprotective enzymes, including heme oxygenase-1 (HO-1) and superoxide dismutase [23]. Research indicates that baicalin facilitates Nrf2 nuclear translocation and enhances its binding affinity to ARE. This upregulates antioxidant enzyme expression, scavenges free radicals, and inhibits ROS accumulation, ultimately mitigating oxidative damage in the vascular wall [24].

3.2 Inhibition of Foam Cell Formation and Enhancement of Cholesterol Efflux

During the nascent stages of AS, circulating monocytes infiltrate the intima and differentiate into macrophages. These macrophages engulf oxidized LDL (ox-LDL) via surface scavenger receptors, leading to massive intracellular cholesterol accumulation and their transformation into foam cells [25, 26].

Under physiological conditions, macrophages internalize ox-LDL primarily via CD36 and expel excess cholesterol via efflux transporters like ABCA1, ABCG1, and SR-BI. Impairment of this reverse cholesterol transport (RCT) exacerbates plaque formation. Studies demonstrate that baicalin inhibits the expression of scavenger receptors such as CD36, reducing ox-LDL uptake. Simultaneously, it upregulates the expression of cholesterol efflux proteins. This dual mechanism facilitates RCT and significantly curtails foam cell formation and lipid burden within plaques [27].

3.3 Amelioration of Systemic Lipid Profiles

Dyslipidemia is a well-established independent risk factor driving AS progression. Beyond localized interventions against lipid deposition, baicalin exerts systemic regulatory effects on the overall lipid profile. In vivo experiments utilizing Apoe atherosclerotic mouse models have shown that chronic baicalin administration significantly reduces serum levels of total cholesterol, triglycerides, and low-density lipoprotein cholesterol, while concurrently elevating high-density lipoprotein cholesterol. This demonstrates robust lipid-lowering and anti-atherosclerotic efficacies [28].

4. Modulation of Immune Responses and Local Immune Homeostasis

The initiation and progression of AS are inextricably linked to immune dysregulation. Within the plaque, dendritic cells (DCs) activate the innate immune response by presenting ox-LDL-derived antigens. Baicalin significantly downregulates the expression of DC surface maturation markers (CD11c and CD83), thereby suppressing their antigen-presenting capabilities and mitigating AS lesions.

In adaptive immunity, the imbalance of CD4+ T cell subsets is a core mechanism precipitating plaque instability. Regulatory T cells (Tregs) confer atheroprotection by releasing anti-inflammatory cytokines (IL-10, TGF-β). Baicalin enhances the expression of Foxp3, promoting Treg differentiation within the plaque. Furthermore, baicalin inhibits the differentiation of pro-inflammatory Type 17 Helper T cells (Th17) by downregulating IL-6 and RORγt expression. This dual action effectively restores the Treg/Th17 balance, re-establishes local immune tolerance, and exerts significant anti-atherosclerotic effects [29].

5. Regulation of Intracellular Signaling Pathways

Baicalin intervenes in the pathological progression of AS through a multi-target, multi-pathway synergistic regulatory network.

The metabolic cascade formed by PPAR-γ and LXR-α is crucial for regulating macrophage reverse cholesterol transport. SR-BI mediates bidirectional cholesterol transport between cells and HDL. In ox-LDL-stimulated macrophages, baicalin inhibits lipid accumulation and enhances cholesterol efflux efficiency by upregulating the expression of SR-BI, PPAR-γ, LXR-α, ABCA1, and ABCG1. Thus, baicalin promotes cholesterol efflux via the PPAR-γ/LXR-α/ABCA1/ABCG1/SR-BI signaling axis [30].

This pathway influences AS progression by regulating

cellular proliferation and lipid homeostasis. Baicalin upregulates the expression of DKK1, an endogenous antagonist, thereby blocking the Wnt/β-catenin pathway, reducing pro-inflammatory cytokine release, and delaying AS progression [31, 32].

Angiotensin II (Ang II) activates Myosin Light Chain Kinase (MLCK), inducing pathological vascular smooth muscle cell (VSMC) contraction and proliferation. Baicalin downregulates the expression of MLCK, phosphorylated MLC, and calmodulin, thereby blocking the proliferative phenotypic switching of VSMCs and ameliorating endothelial dysfunction [33].

Excessive apoptosis and dysregulated autophagy exacerbate plaque vulnerability. Baicalin significantly reduces the expression of Cytochrome C (Cyt C), Apaf-1, and Caspase-9, effectively protecting myocardial cells by inhibiting the mitochondrial apoptotic pathway [34]; It also decreases the pro-apoptotic factor Bax and increases the anti-apoptotic factor Bcl-2 [35].

Furthermore, baicalin maintains cellular homeostasis by preventing excessive autophagy via the inhibition of the AMPK/mTOR signaling axis [36].

6. Summary of In Vitro and In Vivo Experimental Studies

A plethora of experimental models substantiate the multi-dimensional targeting capabilities of baicalin against AS. In an LPS-induced inflammatory model using RAW264.7 cells, baicalin upregulated miR-181b to inhibit HMGB1 expression, thereby blocking the HMGB1/TLR4/NF-κB signaling axis and alleviating inflammation [37]; In an LPS/ATP-induced HUVEC model, baicalin intervened in NEK7-mediated NLRP3 inflammasome assembly, significantly suppressing NLRP3 and IL-1β expression [38].

Furthermore, in a thrombin-induced VSMC inflammatory model, baicalin inhibited ERK1/2 phosphorylation and downregulated PAR-1 expression, suppressing intimal hyperplasia and inflammation [39].

These findings indicate that baicalin mitigates AS through diverse mechanisms, including miRNA regulation, inflammasome interference, and kinase phosphorylation modulation.

7. Clinical Applications, Limitations, and Future Perspectives

Currently, baicalin tablets are widely utilized in clinical practice. They significantly improve the prognosis of patients with cerebral infarction, demonstrating notable efficacy in neurological repair [40].

In cardiovascular cohorts, baicalin monomers effectively reduce serum lipids and high-sensitivity C-reactive protein (hs-CRP) in patients with coronary heart disease [41, 42], Classical TCM formulas containing baicalin, such as Gegen Qinlian, Huanglian Jiedu, and Dachaihu decoctions, are extensively applied in cardiovascular therapeutics. Clinical studies reveal that Gegen Qinlian Decoction is highly effective in treating carotid atherosclerosis [43]; When combined with atorvastatin, it significantly improves lipid metabolism and reduces adverse drug reactions [44]; Huanglian Jiedu Decoction exerts anti-inflammatory effects and promotes M2 macrophage polarization via the PPARγ/NF-κB pathway, showing potential in treating AS and diabetes when co-administered with Western medications [45, 46]. Additionally, Dachaihu Decoction lowers blood lipids and improves hemorheological parameters [47]

Despite the broad prospects of baicalin in AS prevention, the translation from basic research to clinical application faces challenges. Existing clinical data predominantly derive from small-sample observational studies or compound formulas, lacking large-scale, multi-center randomized controlled trials (RCTs) specifically evaluating baicalin monomers. Furthermore, baicalin exhibits poor aqueous solubility, resulting in limited oral bioavailability and difficulties in targeted in vivo delivery.

Future research must prioritize high-quality RCTs to elucidate the dose-effect relationship and safety profile of baicalin in AS therapy. Leveraging modern pharmaceutical technologies, such as nanotechnology, is crucial to overcome its physicochemical bottlenecks and achieve localized delivery to atherosclerotic plaques. Ultimately, in-depth exploration of the synergistic mechanisms between baicalin and contemporary cardiovascular drugs will provide more precise and effective integrative medicine strategies for cardiovascular disease management.

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