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Regulatory Mechanisms of Inflammatory Signaling Pathways in Atherosclerosis by Traditional Chinese Medicine

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Abstract

Atherosclerosis is the main pathological basis of cardiovascular disease. According to the Report on Cardiovascular Health and Diseases in China 2023, there are about 290 million patients in China, and related deaths account for more than 40% of total mortality. Inflammation plays a central role in the onset and progression of atherosclerosis by impairing endothelial function, promoting foam cell formation, and destabilizing plaques, thus forming a persistent "inflammation–injury" cycle. Current Western medical treatments, such as lipid-lowering drugs, antiplatelet therapy, and vascular interventions, remain limited by adverse drug reactions and restenosis. Guided by the holistic perspective and pattern-based treatment of Traditional Chinese Medicine (TCM), therapeutic strategies emphasize the regulation of yin–yang balance and pathological factors such as phlegm, stasis, and toxins, showing multi-component and multi-target advantages. Recent studies have demonstrated that single compounds, herb pairs, and formulae can modulate inflammatory pathways including NF-κB, TLRs, MAPK, and JAK/STAT, thereby suppressing pro-inflammatory cytokines, improving endothelial function, regulating lipid metabolism, and slowing plaque progression. This article reviews research advances over the past five years on TCM interventions in atherosclerosis, aiming to provide theoretical support for mechanistic studies and clinical applications.

Keywords

Atherosclerosis; Inflammation; Signaling Pathways; Traditional Chinese Medicine; NF-κB

1. Introduction

According to the China Cardiovascular Health and Disease Report 2023, approximately 290 million people in China suffer from atherosclerotic cardiovascular diseases (ASCVD), with deaths caused by these conditions accounting for over 40% of total mortality [1]. Atherosclerosis (AS) is the core pathological basis of these diseases, characterized by abnormal lipid accumulation in the arterial intima, excessive smooth muscle cell proliferation, and fibrous tissue deposition [2]. Studies indicate that inflammatory responses are a central mechanism driving the initiation and progression of AS. Inflammation affects endothelial function, promotes macrophage transformation into foam cells, and influences plaque stability, forming a sustained "inflammation–injury" cycle [3]. Therefore, controlling inflammation is critical for AS treatment.

Currently, Western medicine primarily treats AS through lipid-lowering drugs, antiplatelet therapy, and vascular interventional procedures. However, long-term medication may lead to hepatic or renal dysfunction or drug resistance, while interventional therapy can result in restenosis or in-stent thrombosis [4]. Traditional Chinese Medicine (TCM) emphasizes the balance of the internal environment, considering "yin–yang imbalance" as the root cause of

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disease. The inflammatory responses associated with AS can be seen as a manifestation of this imbalance in the vascular system [5].

In recent years, guided by TCM's holistic concept, researchers have applied systems biology and network pharmacology to explore the mechanisms of TCM in treating AS. Studies have found that TCM monomers, herbal pairs, and compound formulas can regulate multiple inflammatory signaling pathways—including NF-κB, TLRs, MAPK, and JAK/STAT—thereby inhibiting proinflammatory factor production (Fig. 1).

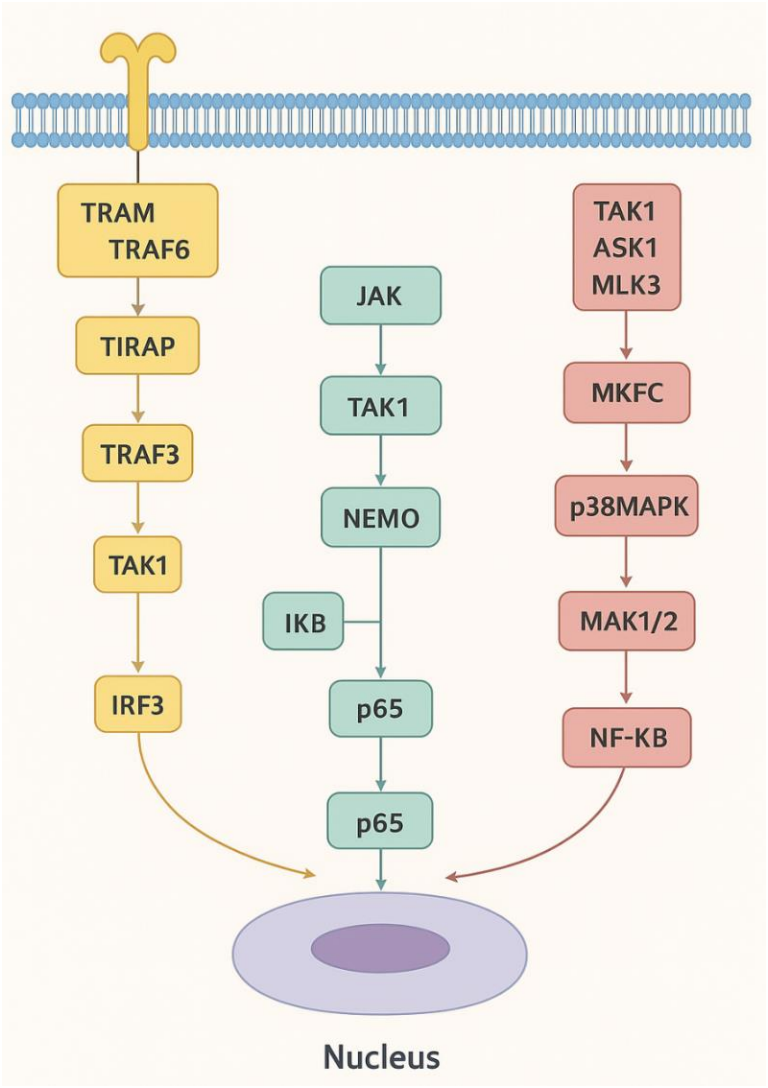

Fig.1 Mechanism of inflammatory signaling pathways in atherosclerosis

Based on this background, this review summarizes research from the past five years on TCM interventions against AS through modulation of signaling pathways, aiming to provide a reference for mechanism studies and clinical applications.

1. TCM Understanding of Atherosclerosis

Although TCM does not explicitly record "atherosclerosis," based on pathological features and clinical manifestations, it can be categorized under "Mai Bi" [6]. The Su Wen · Bi Lun states: "When bi affects the vessels, blood congeals and does not flow," indicating that the disease is primarily caused by obstruction in the blood vessels. The condition is characterized by deficiency in the root and excess in the branch and is closely associated with dysfunction of the heart, liver, spleen, and kidneys.

The Jin Gui Yao Lue notes "Yang is deficient, Yin is tense," suggesting that insufficient heart qi leads to poor blood circulation; heart yang deficiency causes blood stasis; liver dysfunction

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causes qi stagnation, which over time transforms into fire and damages yin fluids, forming phlegm; excessive liver constraint impairs the spleen's transport function, leading to accumulation of turbid lipids; kidney yang deficiency disrupts qi transformation, causing water metabolism disorders; kidney yin depletion results in poor vessel nourishment. Yin–yang imbalance and the interaction of phlegm and blood stasis deposit in the vessels, thickening and hardening vessel walls and forming pathological "vessel blockages."

Although TCM does not have the modern concept of "inflammatory response," theories such as "heat toxin," "phlegm-fire," and "stasis-heat" share similarities. In TCM, inflammation is seen as a process of "struggle between the upright and pathogenic factors," associated with qi stagnation, phlegm–stasis transforming into heat, and accumulation of pathogenic toxins. Chronic accumulation of phlegm and blood stasis can generate heat as "phlegm-fire" or "stasis-heat," further damaging vessels and causing redness, exudation, and tissue injury. The Huangdi Neijing states, "All pains, itching, and sores belong to the heart," indicating that external or internally generated toxins can disrupt qi and blood balance, leading to disease. Modern research shows that "heat toxin" is closely related to excessive release of inflammatory factors and oxidative stress, while "phlegm-stasis" corresponds to lipid metabolism disorders and immune cell infiltration [7].

TCM prevention and treatment of AS emphasize the "holistic concept" and "syndrome differentiation and treatment," focusing on "supporting the upright and dispelling pathogenic factors, harmonizing qi and blood." Multi-level interventions target phlegm, stasis, heat toxins, and deficiency. Common therapeutic approaches include clearing heat and activating blood, resolving phlegm and reducing turbidity, tonifying qi and supporting the upright, along with promoting blood circulation, removing toxins, and dispersing masses. Through multi-target effects, TCM can regulate the inflammatory microenvironment, inhibit inflammatory factor release, reduce endothelial injury, modulate lipid metabolism, and suppress macrophage infiltration within plaques.

Table 1. Regulatory effects of monomeric components of Traditional Chinese Medicine on AS-related signal pathways

Monomeric component	Pathway regulated	Molecular targets
Ginsenoside Rd	NF-κB↓	MDA, iNOS, VCAM-1↓
Verbascoside	HMGB1/RAGE/NF-κB↓	HMGB1, RAGE, p-NF-κB p65↓, SOD, GSH-Px↑
Muscone	NF-κB/p65↓	SOD, VCAM-1, p65, TNF-α↓
Arctium lappa ethanol extract	PI3K/Akt/NF-κB↓	p-PI3K/PI3K, p-Akt/Akt, p-NF-κB p65↓
Artesunate	NF-κB/NLRP3↓	IL-1β, IL-6, IL-18, TNF-α, NLRP3↓
Schisandrin A	NF-κB/MAPK↓	TNF-α, IL-6, p-NF-κB p65↓
Baicalein	NF-κB↓	IL-6, TNF-α, ICAM-1, VCAM-1 mRNA↓
Hyperoside	TLR4/NF-κB↓	IL-6, TNF-α, IL-8, TLR4, NF-κB↓, T cells↑
Gypenosides	TLR4/MyD88/NF-κB↓	TNF-α, TLR4, MyD88, NF-κB↓
Flavonoids from Polygonum hydropiper	TLR4/NF-κB↓	TC, TG, LDL-C, TNF-α, IL-1β, IL-6↓, HDL-C↑
Flavonoids from Chenopodium album	TLR/NF-κB↓	TG, TC, LDL-C↓, HDL-C↑

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Phytolaccoside B	TLR4/NF-κB↓	TLR4, MyD88, NF-κB p65↓
Allicin	TLR4/MyD88/NF-κB↓	TC, TG, LDL-C, non-HDL-C↓, IL-6, IL-1β, TNF-α↓
Honokiol	MAPK/NF-κB/NLRP3↓	Th17 cells↓, MMP-2/9 mRNA↓
Caesalpinia sappan extract	p38MAPK/NF-κB↓	IL-1β, TNF-α, ICAM-1, VCAM-1↓
Salvianolic acid B	MAPKs/NF-κB↓	VCAM, iNOS, P38, ERK1/2, JNK↓
Polydatin	JAK2/STAT3↓	IL-6, TNF-α, JAK2, STAT3↓
Dracocephalum tanguticum flavonoids	JAK/STAT↓	TNF-α, IL-6, JAK, STAT↓
BYHWT glycosides	JAK/STAT↓	TC, TG, LDL-C↓, HDL-C↑
Paeonol	CD40/CD40L↓	TNF-α, IL-6, IL-1↓, miR-145↑
Polydatin	TGF-β/Smad↓	TC, TG, LDL-C↓, TNF-α, IL-6↓

Table 2. Regulatory effects of TCM formulas on AS-related signal pathways

Formula	Pathway regulated	Molecular targets
Yishen Huoxue Huatan Decoction	RhoA/ROCK/NF-κB↓	IL-1β, RhoA, ROCK↓
Huayu Qutan Decoction	G6PD/NF-κB↓	IL-6, IL-1β, TNF-α↓
Dahuang Zhechong Pill	NF-κB↓	TG, TC, LDL-C↓, HDL-C↑
Gualou Xiebai Banxia Decoction	NF-κB↓	TNF-α/IKKβ/NF-κB↓
Scutellaria-Coptis Pair	TLR4/MyD88/NF-κB↓	TNF-α, IL-6, MMP-2, MMP-9↓
Tianxiangdan	TLR4/NF-κB↓	TAG, TC, LDL-C↓, HDL-C↑
Xiaoji Pill	TNF-α/IKKβ/NF-κB↓	TC, TG, LDL-C↓
Yiqi Tongmai Decoction	TLR4/NF-κB↓	TNF-α, MCP-1↓
BYHWT with Astragalus-Angelica	TLR4/MyD88/NF-κB↓	VCAM-1, iNOS↓
Ruanmai Jian	YWHAZ/p38MAPK/CASP3↓	YWHAZ, p38MAPK, CASP3↓
Guanxinkang	MAPKs/NF-κB↓	TC, TG, ALT, AST, ERK1/2, P38, JNK↓
Zexie Drink	MAPK/NF-κB↓	IL-1α, IL-1β, IL-6↓, IL-10↑
Guanxinping	MAPK/NF-κB↓	TNF-α, ICAM-1, VCAM-1↓
Astragalus-Coptis Pair	STAT5↑	TG, TC, MMP-9↓, IL-10↑
Yiqi Huoxue Qingre Jiedu Decoction	JAK/STAT↓	IL-6, JAK1, STAT3↓
Huoxue Qingre Jiedu Decoction	JAK2/STAT3↓	IL-6, IL-1β, TNF-α↓
Zhuyu Pill	ROS/TXNIP/NLRP3↓	NLRP3, Caspase-1↓
Xuefu Zhuyu Decoction	cAMP/PKA/PPARγ↑	TC, TG, LDL-C↓, HDL-C↑
Jiedu Huoxue Decoction	PI3K/Akt/mTOR↓	MCP-1, IL-6, PI3K, Akt, mTOR↓

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2. Role of Inflammation-Related Signaling Pathways in Atherosclerosis and TCM Regulatory Mechanisms

2.1 NF-κB Signaling Pathway

NF-κB is a central transcription factor in the inflammatory response of atherosclerosis, composed of five subunits: p50, p52, RelA/p65, RelB, and c-Rel [8]. Under normal conditions, the p65/p50 dimer of NF-κB binds to the inhibitory protein IκBα, remaining inactive in the cytoplasm [9]. Upon cellular stimulation, the upstream IKK complex is activated, leading to phosphorylation and degradation of IκBα. This allows NF-κB to translocate into the nucleus and initiate the expression of proinflammatory factors such as TNF-α and IL-1β, aggravating vascular inflammation and promoting atherosclerosis progression [10].

Many active compounds from TCM can inhibit the NF-κB signaling pathway. For example, ginsenoside Rd reduces blood lipids and proinflammatory factor levels in atherosclerotic mice, associated with NF-κB inhibition [11]. Mahonia glycosides decrease HMGB1, RAGE, and p-NF-κB p65 protein expression while enhancing antioxidant enzyme activity, alleviating endothelial injury [12]. Musk ketone reduces NF-κB/p65 and TNF-α expression, effectively decreasing plaque area [13]. Ethanol extracts of burdock leaves may regulate the PI3K/Akt/NF-κB pathway to inhibit smooth muscle cell proliferation and inflammatory factor release [14]. Artesunate downregulates NLRP3 inflammasome and related factors, likely through NF-κB/NLRP3 pathway inhibition [15]. Schisandrin A reduces p-NF-κB p65 phosphorylation and oxidative stress. Baicalin inhibits NF-κB p65 phosphorylation and IκB-α degradation, decreasing vascular adhesion molecule expression and mitigating arterial inflammation.

TCM formulas also exert effects via NF-κB modulation. The Kidney-Tonifying and Blood-Activating Decoction reduces RhoA, ROCK, and p-NF-κB p65 levels, decreasing plaque area. The Stasis-Resolving and Phlegm-Removing Decoction improves intimal thickening and plaque stability via the G6PD/NF-κB pathway. Da Huang Zhe Chong Pill regulates lipids and inhibits NF-κB p65 and IκBα phosphorylation, reducing proinflammatory factor release. β-Sitosterol in Gualou Xiebai Banxia Decoction blocks the IKKβ/NF-κB axis, inhibiting NF-κB nuclear translocation and improving lipid metabolism and inflammation.

2.2 TLRs Signaling Pathway

Toll-like receptors (TLRs) are key pattern recognition receptors, among which TLR4 plays a prominent role in atherosclerosis. Upon activation, TLR4 transmits signals via MyD88-dependent and -independent pathways. The MyD88-dependent pathway activates IKK, subsequently activating NF-κB and inducing early inflammatory factor production. The MyD88-independent pathway activates IRF3 and late-phase NF-κB through TRIF and TRAM. Both pathways ultimately promote inflammatory factor expression, exacerbating vascular injury.

Various TCM components can modulate TLRs signaling. Hypericin reduces TLR4 and NF-κB protein expression in a dose-dependent manner. Total saponins from Gynostemma inhibit TLR4, MyD88, and NF-κB activation at the mRNA level. Total flavonoids from Syzygium aromaticum downregulate TLR4, NF-κB, and p-P65 protein expression. Total flavonoids from Portulaca oleracea reduce lipids and lower TLR3 and NF-κB p62 levels. Saponin A from Phytolacca blocks TLR4/NF-κB, decreasing aortic plaque area. Allicin downregulates TLR4/MyD88/NF-κB signaling, improving lipid metabolism and inflammation.

TCM formulas also act via this pathway. Huangqin-Huanglian Decoction reduces macrophage infiltration in plaques and suppresses TNF-α, IL-6 release, likely by modulating TLR4/MyD88/NF-κB. Tianxiang Dan may protect endothelial cells by inhibiting TLR4/NF-κB. Xiaojimaru downregulates TNF-α/IKKβ/NF-κB protein expression. Yi Qi Tong Mai Decoction

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targets TLR4/MyD88, reducing macrophage infiltration and lipid accumulation. Bu Yang Huan Wu Tang and Astragalus-Angelica combinations also modulate this pathway, improving vascular inflammatory microenvironment.

2.3 MAPK Signaling Pathway

The MAPK pathway includes ERK, JNK, and p38 subfamilies, with JNK and p38 particularly important in inflammation. External stimuli activate upstream kinases, which phosphorylate MKK4/7 or MKK3/6, ultimately activating JNK or p38, which translocate to the nucleus to regulate gene transcription and exacerbate inflammation. Honokiol may improve atherosclerotic lesions via antagonizing MAPK/NF-κB/NLRP3. Sumac extracts reduce p38 MAPK and NF-κB expression. Danshensu B inhibits phosphorylation of p38, ERK1/2, and JNK, acting via MAPKs/NF-κB. The Ruanmai Decoction may modulate lipid metabolism by inhibiting YWHAZ/p38 MAPK/CASP3. Guanxin Kang reduces inflammatory factor release via MAPKs/NF-κB. Ze Xie Yin downregulates p38, ERK, and NF-κB, alleviating inflammation. Guanxin Ping inhibits MAPK/NF-κB, improving intimal thickening.

2.4 Multi-Target Network Regulation of TCM: Other Signaling Mechanisms

The JAK/STAT pathway is a critical intracellular signaling cascade; cytokine-receptor binding activates Janus kinases, which phosphorylate STATs, enabling nuclear translocation and gene regulation in inflammation. Resveratrol reduces JAK2 and STAT3 phosphorylation, mitigating endothelial inflammation. Total flavonoids from Kaempferia elegans downregulate JAK and STAT protein and mRNA expression, slowing inflammation. Bu Yang Huan Wu Tang glycosides regulate this pathway to improve lipid deposition and inhibit smooth muscle proliferation.

Formulas such as Huangqi-Huanglian may restore immune balance via STAT5 activation. Yi Qi Huo Xue Qing Re Jie Du Decoction decreases IL-6, JAK1, and STAT3 mRNA levels. Huo Xue Qing Re Jie Du Decoction may act via JAK2/STAT3 inhibition. Beyond JAK/STAT, the CD40/CD40L pathway participates in AS inflammation, recruiting TRAF6, activating NF-κB, and promoting TNF-α and IL-1β release; paeonol may reduce foam cell damage and enhance plaque stability via this pathway. Excess ROS damages endothelium, promotes smooth muscle proliferation, and accelerates lipoprotein oxidation; NADPH oxidase-derived ROS exacerbates inflammation via the NOX/ROS-NF-κB axis, which Zhuyu Pill may attenuate via ROS/TXNIP/NLRP3 inhibition.

Additionally, Xuefu Zhuyu Decoction may regulate lipid metabolism and inflammation via cAMP/PKA/PPARγ; Jiedu Huoxue Decoction may promote autophagy and stabilize plaques via PI3K/Akt/mTOR inhibition; resveratrol can also modulate TGF-β/Smad to improve endothelial function.

3. Conclusion

The development of atherosclerosis is closely linked to inflammatory responses, with inflammation-related signaling pathways playing a central role in disease progression. In recent years, guided by the traditional principles of “holistic perspective” and “pattern-based treatment,” Traditional Chinese Medicine (TCM), in combination with modern molecular biology, has gradually revealed its unique advantages in regulating inflammatory pathways. Numerous studies have confirmed that single compounds, herb pairs, and formulae can, through multi-target and multi-level synergistic actions, modulate key pathways such as NF-κB, TLRs, MAPK, and JAK/STAT. These interventions inhibit the release of pro-inflammatory cytokines, improve endothelial function, regulate lipid metabolism, and, to some extent, prevent plaque progression and instability. Such multi-component and multi-target effects align well with the multifactorial pathological mechanisms of atherosclerosis.

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Therapeutic approaches in TCM—such as "clearing heat and detoxifying, activating blood circulation to remove stasis, resolving phlegm and eliminating turbidity, and reinforcing qi to strengthen the body"—are increasingly being reinterpreted in modern studies as corresponding anti-inflammatory, antioxidant, lipid-regulating, and immunomodulatory mechanisms. This not only enriches the modernization of traditional theories but also provides directions for innovative drug development. It is noteworthy, however, that most existing research remains at the level of animal experiments and in vitro mechanisms, with insufficient clinical evidence from evidence-based medicine and a lack of standardized evaluation systems. Therefore, future research should strengthen high-quality clinical trials and integrate multi-omics approaches to explore both the systemic effects and specific mechanisms of TCM interventions in atherosclerosis, aiming to advance the standardization and internationalization of TCM in cardiovascular disease prevention and treatment.

Bridging traditional theory and modern science not only helps to uncover the complexity of inflammatory mechanisms in atherosclerosis but also provides new perspectives and practical foundations for integrative therapies that combine TCM and Western medicine.

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