Journal of Clinical and Nursing Research, 2026, Volume 10, Issue 5
http://ojs.bbwpublisher.com/index.php/JCNR
Online ISSN: 2208-3693
Print ISSN: 2208-3685

Research Progress on Improving Chronic Inflammation and Endothelial Function of Coronary Atherosclerotic Heart Disease with Integrated Traditional Chinese and Western Medicine

Zheng Huang[1], Qiao Zeng[2], Jianhui Gu[2], Liping Lu[1], Yongxi Zhou[1]*

[1]Fusui County Hospital of Traditional Chinese Medicine, Chongzuo 532100, Guangxi, China
[2]Guangxi University of Chinese Medicine, Nanning 530000, Guangxi, China

Author to whom correspondence should be addressed.

Abstract: The progression of coronary atherosclerotic heart disease (CAD) is closely associated with the persistent activation of chronic low-grade inflammation and vascular endothelial dysfunction. Although percutaneous coronary intervention (PCI) can effectively improve myocardial ischemia, patients often exhibit elevated inflammatory burden, insufficient endothelial repair, and impaired microcirculatory perfusion postoperatively, thereby contributing to a sustained residual cardiovascular risk. In recent years, integrated traditional Chinese (TCM) and Western medicine therapy have demonstrated advantages in multi-targeted, systematic, and comprehensive regulation to interrupt the vicious cycle of inflammatory responses and endothelial damage. This article reviews the modern pathological mechanisms of chronic inflammation and endothelial dysfunction in coronary atherosclerotic heart disease and explores the underlying pathogenesis from a TCM perspective. Within this framework, this article summarizes the foundational and clinical research progress on anti-inflammatory and endothelial protective effects of TCM through regulating inflammatory signaling pathways, protecting the endothelial glycocalyx, improving blood flow shear stress, and modulating immune metabolism. Finally, this article analyzes the limitations of current research in terms of the quality of evidence, the objectification of syndromes, and the elucidation of the material basis for efficacy of Chinese herbal medicine, and outlines future directions for translational medicine research, with the aim of providing a basis for optimizing integrated TCM and Western medicine prevention and treatment strategies for CHD.

Keywords: Coronary atherosclerotic heart disease; Integrated traditional Chinese and Western medicine; Chronic inflammation; Endothelial dysfunction; Residual risk; Microcirculation

Online publication: May 31, 2026

1. Introduction

Over the past two decades, significant advances have been achieved in the diagnosis and treatment of coronary atherosclerotic heart disease (CAD). Strategies including percutaneous coronary intervention (PCI), intensive lipid-lowering therapy, and antiplatelet therapy have effectively reduced the incidence of acute cardiovascular events, with marked improvements in revascularization rates and plaque stability [1]. However, revascularization does not mean the termination of the pathological process. A considerable proportion of patients still suffer from recurrent chest pain, reduced exercise tolerance, persistently elevated inflammatory markers, and impaired flow-mediated vasodilation postoperatively. This suggests that conventional treatments are insufficient to fully regulate the underlying pathological mechanisms of disease progression, and residual cardiovascular risk remains a key factor affecting long-term prognosis [2].

Recent studies have shown that the persistent activation of chronic low-grade inflammation and endothelial dysfunction mutually reinforce each other, jointly driving a series of pathological processes such as plaque formation, impaired microcirculatory perfusion, and reperfusion injury [3]. Among these, abnormalities such as NOD-like receptor protein 3 (NLRP3) inflammasome-mediated pro-inflammatory cytokine release, cascade amplification of the nuclear factor-kappa B (NF-κB) signaling pathway, disruption of the endothelial glycocalyx, and reduced nitric oxide (NO) bioavailability form an irreversible "inflammation–endothelial" dual-axis pathological framework [4].

Traditional Chinese medicine (TCM) recognizes the pathogenesis of chest impediment and cardiac pain as involving "toxin damaging the heart collaterals", "Qi deficiency and blood stasis", and "phlegm turbidity obstruction", which share some similarities with the chronic inflammation, energy metabolism disorders, and microcirculatory damage revealed by modern pathophysiology [5]. With the advancement of technologies such as metabolomics and single-cell sequencing, the molecular and biochemical bases of TCM syndromes have become increasingly elucidated, providing verifiable biological support for the holistic regulatory strategy emphasized in syndrome differentiation and treatment [6].

In recent years, TCM has demonstrated comprehensive, multi-targeted, and multi-level advantages in regulating inflammatory signaling, improving endothelial responsiveness, and stabilizing the cardiovascular microenvironment. Certain active components of Chinese materia medica can interfere with inflammasome assembly, inhibit oxidative stress, and modulate immune polarization; studies have also confirmed that they can upregulate the PI3K/Akt/eNOS signaling pathway, protect the endothelial glycocalyx structure, and improve microcirculatory perfusion [7]. These findings provide new therapeutic insights for regulating the "inflammation–endothelial" pathological axis and lay a theoretical foundation for repositioning TCM in the management of CAD.

2. Modern mechanisms of chronic inflammation and endothelial dysfunction in CAD

Coronary atherosclerosis is not merely a process of lipid deposition, but rather a persistent pathological event driven by immune activation and repeated vascular wall injury against a backdrop of lipid imbalance. Extensive research indicates that chronic low-grade inflammation and vascular endothelial dysfunction run throughout the entire course of atherosclerosis; together, they form a mutually reinforcing pathological axis that serves as the core driver of CAD progression [8].

2.1. Chronic inflammation: The sustained driver of atherosclerosis

Dyslipidemia marks the onset of atherosclerosis, yet the long-term immune response to intimal lipid deposits and cellular debris is what truly governs plaque evolution and vulnerability [9]. Key mechanisms include inflammasome activation and pro-inflammatory cytokine release, cascade amplification of inflammatory signals, and dysregulated macrophage polarization. The NLRP3 inflammasome serves as a critical danger signal recognition mechanism in atherosclerosis. Stimuli such as cholesterol crystals and oxidized low-density lipoprotein (LDL) can induce its assembly and activate caspase-1, thereby promoting the maturation and release of (interleukin-1 beta) IL-1β and interleukin-18 (IL-18) [10]. These factors not only amplify local inflammation but also drive smooth muscle cell migration, expansion of the necrotic core, and thinning of the fibrous cap, thereby increasing plaque vulnerability. The NF-κB signaling pathway is highly active in endothelial and immune cells, upregulating adhesion molecules such as vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1). This promotes monocyte adhesion and infiltration, forming a positive feedback loop of "infiltration–activation–re-infiltration" that underpins persistent plaque progression [11]. A high-lip microenvironment and pro-inflammatory stimuli drive macrophages migration toward the M1 phenotype, resulting in the excessive production of matrix metalloproteinases (MMPs) and the destruction of the fibrous cap structure, while the reparative M2 phenotype decreases, making it difficult to restore plaque homeostasis [12]. Landmark trials such as CANTOS have demonstrated that IL-1β blockade significantly reduces recurrent cardiovascular events, further highlighting the central role of inflammation in residual risk management [13]. In summary, the inflammatory response is not merely a concomitant phenomenon, but a critical basis driving plaque instability and clinical events.

2.2. Endothelial dysfunction: From structural damage to microcirculatory failure

The vascular endothelium is a highly active metabolic and signaling structure involved in regulating vasodilation, coagulation, inflammation, and permeability. Its functional decline plays a pivotal role in the progression of CAD [14]. Key processes include decreased NO bioavailability and endothelial nitric oxide synthase (eNOS) uncoupling, disruption of the endothelial glycocalyx and altered permeability, as well as a procoagulant state and increased susceptibility to microthrombosis. Under enhanced oxidative stress, eNOS shifts from a NO-producing coupled state to a superoxide-producing uncoupled state, resulting in reduced NO levels and elevated free radicals levels. This causes an imbalance in the NO/endothelin 1 (ET-1) ratio, resulting in decreased flow-mediated dilation (FMD), increased coronary microvascular resistance, and impaired vascular compliance [15]. The glycocalyx serves as a critical protective barrier on the endothelial surface, participating in shear stress sensing, anti-inflammation, and the regulation of permeability. Upon its disruption, endothelial adhesion molecules are exposed and leukocyte adhesion increases, thereby accelerating inflammatory cell the infiltration and forming a secondary feedback loop of "endothelial injury–inflammatory infiltration" [16]. Endothelial injury can promote increased expression of von Willebrand factor (vWF) and tissue factor, leading to a local hypercoagulable state. This not only aggravates microcirculatory dysfunction but also increases the risk of microembolism and reperfusion injury following percutaneous coronary intervention (PCI) [17]. Overall, endothelial dysfunction reflects impaired vascular regulatory capacity and serves as an important pathological basis for microcirculatory abnormalities and the persistence of symptoms.

3. Modern biological basis of TCM pathogenesis in CAD

Although traditional medical literature does not use modern terms such as "endothelial dysfunction" or "inflammasome", its understanding of the pathogenesis of conditions such as chest impediment and cardiac pain, embodied in TCM theories of "deficiency at the root and excess at the branch", "combined deficiency and excess" and "Qi-blood disharmony", aligns with modern understanding of the pathological processes of CAD at multiple levels. With the development of multi-omics technologies, systems biology, and network pharmacology, the biological basis of TCM pathogenesis has gradually been clarified, providing pattern differentiation with quantifiable and verifiable modern interpretations [18].

3.1. Qi deficiency pattern: Insufficient energy metabolism and mitochondrial dysfunction

TCM holds that "Qi is the commander of blood". Qi deficiency manifests as fatigue, shortness of breath, and palpitations, reflecting a diminished capacity to to propel blood circulation and generate essence and Qi. In animal models of Qi deficiency and some patients with CHD, reduced ATP synthase activity and decreased membrane potential have been observed, leading to low energy of cardiomyocytes [19]. Insufficient activity of the AMP-activated protein kinase (AMPK)/PGC-1α signaling axis leads to impaired regulation of mitochondrial biogenesis and autophagy [20]. Patients with Qi deficiency often present with reduced heart rate variability (HRV), which is associated with impaired myocardial tolerance to ischemia and oxidative stress [21]. Therefore, "Qi deficiency" can be regarded as a state of systemic downregulation of the myocardial energy metabolism network, making the heart vulnerable to injury and laying the foundation for subsequent inflammation and microcirculatory dysfunction.

3.2. Blood stasis pattern: A comprehensive manifestation of endothelial activation, blood viscosity, and coagulation-fibrinolysis imbalance

"Blood stasis" is one of the concepts in TCM pathogenesis that most closely resembles the pathological essence of atherosclerosis, manifesting as stabbing chest pain, purplish tongue, and hesitant pulse. Decreased eNOS activity and an imbalanced NO/ET-1 ratio predispose blood vessels to vasoconstriction [22]. Patients with blood stasis often show elevated ICAM-1 and VCAM-1 levels, which facilitate leukocyte and platelet adhesion and thereby establish an inflammatory microcirculatory environment [23]. Enhanced platelet activation and elevated levels of P-selectin and platelet activating factor (PAF) contribute to a hyperviscous and hypercoagulable state [24]. These changes collectively result in "vessel obstruction", which closely aligns with clinical characteristics of CAD, such as inadequate microcirculatory perfusion and worsening chest pain after exercise. These findings also provide a modern biological basis for elucidating the mechanism by which blood-activating and stasis-resolving medicinal improve endothelial function and reduce blood viscosity.

3.3. Phlegm turbidity pattern: Lipid metabolism disorders, lipotoxicity, and immune-metabolic imbalance

In TCM, "phlegm turbidity" refers to body fluid metabolism disorders and viscous obstruction; its modern biological basis centers on lipid metabolism abnormalities and metabolic inflammation. Patients with phlegm turbidity pattern often present with elevated small dense low-density lipoprotein (sdLDL) and triglycerides levels and are prone to macrophage lipid uptake via receptors including CD36, thereby promoting foam cell formation [25]. Obesity-related adipokine imbalance (e.g., elevated leptin and decreased adiponectin) can trigger systemic low-grade inflammation [26]. Abnormalities in metabolic pathways such as mammalian

target of rapamycin (mTOR) and peroxisome-proliferator-activated receptor (PPAR) make the cardiovascular system more susceptible to oxidative stress and lipotoxicity [27]. Consequently, "phlegm-turbidity" can be understood as a state of systemic metabolic stress and lipid accumulation, serving as a key contributing factor to the development and progression of atherosclerosis.

4. Molecular mechanisms of TCM in improving chronic inflammation and endothelial function

TCM is characterized by holistic regulation and multi-target intervention in the treatment of CAD. With advancements in molecular biology, systems pharmacology, and imaging technologies, the mechanisms by which TCM modulates inflammatory responses and maintains endothelial function are gradually shifting from empirical understanding to verifiable biological frameworks. Extensive research shows that active components of Chinese materia medica and classical formulas can act on multiple critical nodes, including inflammasomes, oxidative stress regulation, glycocalyx repair, NO synthesis, and immune-metabolic pathways, thereby intervening in the "inflammation–endothelial" pathological axis.

4.1. Precise regulation of inflammatory responses: Weakening the inflammatory amplification chain at its source

Inflammation is a key driver of the pathological progression of CAD. The core of TCM therapeutic approaches such as "clearing heat and removing toxins" and "activating blood circulation and unblocking collaterals" lies in suppressing abnormal inflammatory responses and excessive immune activation. Modern research reveals that various types of Chinese materia medica can exert inhibitory effects on the upstream stages of the inflammatory process. Inhibiting the overactivation of the NLRP3 inflammasome, an essential initiating node in the inflammatory amplification cascade of atherosclerosis, is particularly crucial, as its abnormal activation induces the sustained release of IL-1β and IL-18 [28]. Studies have shown that tanshinone IIA can reduce mitochondrial reactive oxygen species (ROS) production, thereby inhibiting NLRP3 assembly at its source; baicalin can downregulate the expression of caspase-1 and apoptosis-associated speck-like protein with CARD domain (ASC) proteins and reduce the level of inflammasome activation [29]. These effects are highly consistent with modern research and development strategies for identifying NLRP3-targeted inhibitors, suggesting that certain Chinese materia medica components have potential anti-inflammasome activity. Blocking the nuclear translocation and signal amplification of the NF-κB pathway: NF-κB serves as the central hub of the pro-inflammatory transcriptional network; its activation enhances the expression of adhesion molecules and promotes immune cell infiltration. Studies have shown that ligustrazine can inhibit IκBα phosphorylation, thereby blocking NF-κB/p65 nuclear translocation; *Panax notoginseng* saponins (PNS) can inhibit tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) expression, forming a "negative feedback" regulation of the inflammatory response. Regulating macrophage polarization to restore immune homeostasis: Macrophage phenotypic transformation within plaques determines the inflammation progression. Certain compound formulations, such as Danhong Injection, have been confirmed to promote M1-to-M2 polarization, reduce the release of MMPs and pro-inflammatory cytokines; and enhance plaque stability [30]. The TCM therapeutic principle of "regulating without suppression" in maintaining immune homeostasis aligns with the current direction of modern immunomodulatory drug development.

4.2. Multilevel restoration of endothelial homeostasis: Reconstructing the vascular "defensive barrier"

The vascular endothelium is central to maintaining vascular vasodilation, anticoagulation, and inflammatory balance. TCM therapeutic approaches such as promoting blood circulation, tonifying Qi, and nourishing yin have demonstrated definite endothelial protective effects in modern research.

(1) Activating the PI3K/Akt/eNOS pathway and enhancing NO bioavailability. NO is a key factor in maintaining vasodilation and inhibiting platelet adhesion. Astragaloside IV can promote Akt phosphorylation, increase eNOS activity, improve eNOS uncoupling, and enhance NO production, which is consistent with clinical findings of improved fibromuscular dysplasia (FMD) and increased exercise tolerance.

(2) Activating the nuclear factor-erythroid 2-related factor 2 (Nrf2) antioxidant pathway and mitigating oxidative stress damage. Oxidative stress is a major contributor to endothelial injury and inflammation. Ginsenoside Rg1 can enhance Nrf2 nuclear translocation and upregulate antioxidant enzymes such as heme oxygenase-1 (HO-1) and quinone oxidoreductase 1 (NQO1) [31]. Quercetin can remove excess ROS and alleviate reperfusion injury. This mechanism provides a molecular basis for the application of TCM in endothelial repair following percutaneous coronary intervention.

(3) Inhibiting the expression of adhesion molecules and reducing the aggregation of inflammatory cells. Blood-activating and stasis-resolving medicinal, such as salvianolate, can downregulate the expression of ICAM-1 and VCAM-1, thereby reducing leukocyte adhesion and trans-endothelial migration; this effect is of significant importance for reversing the inflammatory burden in the microcirculation [32].

5. Conclusion

The development and progression of CAD are driven by a dual mechanism involving chronic low-grade inflammation and vascular endothelial dysfunction. This "inflammation–endothelial" pathological axis not only determines plaque formation, evolution, and vulnerability but also profoundly impacts the quality of microcirculatory perfusion and long-term prognosis following revascularization [33]. Although current standard treatments have achieved remarkable success in improving coronary patency, stabilizing plaques, and controlling risk factors, there remains an inadequate intervention targeting the mutually reinforcing processes between inflammation and endothelial function, resulting in residual cardiovascular risk that is difficult to eliminate.

Against this backdrop, TCM, characterized by holistic regulation and multi-pathway, multi-target interventions, can exert compensatory advantages in suppressing inflammatory responses, protecting endothelial structure, improving microcirculatory perfusion, and regulating metabolic homeostasis. In recent years, basic and clinical studies have continuously revealed the key molecular mechanisms underlying these effects, providing a biologically based explanation for TCM concepts such as "toxin damaging the heart collaterals" and "stasis obstructing the vessels". This also highlights the value of integrated TCM and Western medicine in the management of CAH.

Funding

Chongzuo Municipal Science and Technology Program (Project No.: Chongke FA2020027); National Project

for the Establishment of Inheritance Studios for Renowned Senior Traditional Chinese Medicine Experts at the Grassroots Level: Li Dafen, Fusui County Hospital of Traditional Chinese Medicine, Guangxi Zhuang Autonomous Region, National Administration of Traditional Chinese Medicine, Personnel and Education Circular [2024] (Project No.: 256)

Disclosure statement

The authors declare no conflict of interest.

References

[1] Levine G, Bates E, Blankenship J, et al., 2011, 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention. Journal of the American College of Cardiology, 58(24): e44–e122.

[2] Serruys P, 2021, Coronary Syndromes After PCI. Lancet, 398(10294): 1233–1246.

[3] Libby P, 2021, Inflammation in Atherosclerosis. Nature, 592: 524–533.

[4] Toldo S, Abbate A, 2018, The NLRP3 Inflammasome in Acute Myocardial Infarction. Nature Reviews Cardiology, 15(4): 203–214.

[5] Hu D, 2020, Expert Consensus on the Diagnosis and Treatment of Coronary Atherosclerotic Heart Disease with Integrated Traditional Chinese and Western Medicine (2020). Chinese Journal of Internal Medicine, 59(4): 261–270.

[6] Zhang A, 2018, Metabolomics in TCM Syndrome Research. Journal of Ethnopharmacology, 213: 310–319.

[7] Li C, 2021, Herbal Compounds Targeting NLRP3 Inflammasome in Cardiovascular Diseases. Phytomedicine, 92: 153737.

[8] Moore K, Tabas I, 2011, Macrophages in the Pathogenesis of Atherosclerosis. Cell, 145: 341–355.

[9] Duewell P, Kono H, Rayner K, et al., 2010, NLRP3 Inflammasomes Are Required for Atherogenesis and Activated by Cholesterol Crystals. Nature, 464(7293): 1357–1361.

[10] Baker R, Hayden M, Ghosh S, 2011, NF-κB, Inflammation, and Metabolic Disease. Cell Metabolism, 13: 11–22.

[11] Murray P, 2017, Macrophage Polarization. Annual Review of Physiology, 79: 541–566.

[12] Ridker P, 2017, CANTOS Trial. New England Journal of Medicine, 377: 1119–1131.

[13] Deanfield J, 2007, Endothelial Function and Dysfunction. Circulation, 115: 1285–1295.

[14] Förstermann U, Sessa W, 2012, Nitric Oxide Synthases. European Heart Journal, 33: 829–837.

[15] Tarbell J, Cancel L, 2016, The Glycocalyx and Vascular Permeability. Journal of Internal Medicine, 280: 97–113.

[16] Choi B, 2010, Endothelial Dysfunction and Microvascular Obstruction. Journal of the American College of Cardiology, 55: 538–546.

[17] Zhang J, 2019, Mitochondrial Dysfunction in CHD Models with Qi Deficiency. Biomedicine & Pharmacotherapy, 118: 109282.

[18] Hardie D, 2016, AMPK: A Central Regulator of Metabolism. Nature Reviews Molecular Cell Biology, 17: 801–819.

[19] Shaffer F, 2017, HRV and Cardiac Function. Frontiers in Public Health, 5: 258.

[20] Weber C, Noels H, 2011, Cell Adhesion Molecules in Atherosclerosis. Nature Medicine, 17: 1112–1121.

[21] Ruggeri Z, 2002, Platelets in Atherothrombosis. New England Journal of Medicine, 347: 1130–1132.

[22] Moore K, Tabas I, 2011, Macrophages and Foam Cell Formation. Cell, 145: 341–355.

[23] Ouchi N, Parker J, Lugus J, et al., 2011, Adipokines in Inflammation and Metabolic Disease. Nature Reviews Immunology, 11(2): 85–97.

[24] Saxton R, Sabatini D, 2017, mTOR Signaling in Metabolism. Cell, 168: 960–976.

[25] Zhou Z, 2021, Tanshinone IIA Attenuates Atherosclerosis via Inhibiting ROS-Mediated NLRP3 Inflammasome Activation. Phytomedicine, 91: 153693.

[26] Li J, 2020, Baicalin Inhibits NLRP3 Inflammasome Activation. International Immunopharmacology, 81: 106195.

[27] Liu X, 2018, Ligustrazine Suppresses NF-κB Activation. Biomedicine & Pharmacotherapy, 103: 1415–1421.

[28] Fan Y, 2012, Panax Notoginseng Saponins Inhibit Inflammation via NF-κB Modulation. Journal of Ethnopharmacology, 141: 222–230.

[29] Hu X, 2020, Danhong Injection Modulates Macrophage Polarization. Frontiers in Pharmacology, 11: 582234.

[30] Zhang W, 2016, Astragaloside IV Improves Endothelial Dysfunction via PI3K/Akt/eNOS. Atherosclerosis, 247: 88–96.

[31] Yang L, 2016, Ginsenoside Rg1 Protects Endothelial Cells via Nrf2/HO-1. Free Radical Biology and Medicine, 97: 229–240.

[32] Boots A, 2008, Quercetin and Oxidative Stress. Mutation Research, 659: 47–52.

[33] Chen Y, 2019, Salvianolic Acids Protect Endothelium and Reduce Adhesion Molecule Expression. Phytomedicine, 62: 152940.