

Exploration on the Correlation between Mitophagy Dysfunction and Atherosclerosis Progression Based on the Phlegm-Blood Stasis Interaction Theory

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Abstract: Atherosclerosis (AS) is the common pathological basis of various cardiovascular diseases. As a selective form of autophagy, mitophagy specifically eliminates damaged mitochondria and acts as a pivotal regulatory mechanism during the initiation and progression of AS. Moderate mitophagy clears dysfunctional mitochondria, maintains mitochondrial quality stability and cellular homeostasis, and protects against vascular pathological damage. In contrast, defective mitophagy results in the accumulation of impaired mitochondria, aggravating intracellular oxidative stress, vascular inflammation and excessive cellular apoptosis, and ultimately accelerating AS progression. From the perspective of traditional Chinese medicine (TCM), intermingled phlegm and blood stasis represents the core pathogenesis of AS. Mechanistically, the vascular-protective effect of mitophagy is highly consistent with the TCM therapeutic principle of eliminating phlegm and resolving blood stasis. Based on the TCM pathogenesis theory of phlegm-stasis intermingling, this review elaborates the intrinsic association between mitophagy dysfunction and AS progression, and systematically summarizes the core signaling pathways and molecular mechanisms by which TCM intervenes in AS via regulating mitophagy, so as to provide novel theoretical evidence and potential therapeutic strategies for the prevention and clinical treatment of atherosclerosis with TCM.

1. Introduction

Atherosclerosis (AS) is a fundamental pathological lesion underlying most cardiovascular diseases, and its progression is driven by multiple key pathological processes, including abnormal lipid deposition, vascular endothelial dysfunction, and persistent inflammatory cell infiltration [1]. Although modern clinical interventions, represented by statin lipid-lowering therapy and antiplatelet treatment, have effectively reduced the incidence of severe adverse cardiovascular events and achieved remarkable progress in AS prevention and treatment [2], residual clinical risks remain unresolved. Specifically, residual inflammatory injury and unstable atherosclerotic plaque rupture are still the leading causes of refractory cardiovascular events after standardized conventional treatment

[3]. Mitophagy is a subtype of selective autophagy that exclusively targets and degrades damaged or functionally impaired mitochondria. As a core component of mitochondrial quality control systems, mitophagy eliminates dysfunctional mitochondria and toxic metabolic byproducts, stabilizes intracellular physiological homeostasis, and preserves mitochondrial structural and functional integrity [4]. Accumulating preclinical and clinical evidence has demonstrated that mitophagy exerts a bidirectional regulatory effect on the entire course of AS. By modulating lipid metabolism disorders, oxidative stress injury, and vascular inflammatory responses, mitophagy acts as a crucial molecular regulator of vascular lesion progression, providing innovative mechanistic insights and potential therapeutic targets for the precise prevention and treatment of AS [5]. Modern TCM pharmacological studies have verified that the classical TCM pathogenesis of phlegm-stasis intermingling in AS is closely correlated with lipid metabolic disorders and mitochondrial dysfunction induced by insufficient mitophagy [6]. Impaired mitophagy triggers persistent mitochondrial damage and excessive reactive oxygen species (ROS) generation, which induces abnormal apoptosis of vascular cells, accelerates vascular wall injury and atherosclerotic plaque hyperplasia, and constitutes the core pathological basis of AS with phlegm-stasis intermingling syndrome [7]. In view of the above, this review systematically discusses the regulatory role of mitophagy in the occurrence and progression of AS under the guidance of the TCM phlegm-stasis intermingling theory, aiming to provide innovative theoretical references and research directions for the integrated Chinese and Western medical prevention and treatment of AS.

2. Intermingled Phlegm and Blood Stasis Theory and Atherosclerosis

The term “atherosclerosis” is not explicitly recorded in classical TCM literature. However, its clinical manifestations, etiological characteristics and complications correspond to TCM disease categories such as Chest Obstruction (Xiong Bi), True Heart Pain (Zhen Xin Tong), Stroke (Zhong Feng), and Phlegm Fluid Retention (Tan Yin). The TCM core pathogenesis of AS is summarized as “root deficiency and branch excess”, in which spleen qi deficiency and kidney essence depletion constitute the fundamental root cause, while turbid phlegm accumulation and blood stasis intermingling serve as the superficial pathological manifestation. As early as in Synopsis of Golden Chamber (Jin Gui Yao Lue), Zhongjing Zhang put forward that “yang-weak and yin-string” is the core pathogenesis of this disease, manifested as inhibited chest-yang and obstruction by yin pathogens, and formulated the classic prescription Gualou Xiebai Baijiu Decoction. Modern studies have demonstrated that lipid deposition and inflammatory response in atherosclerotic lesions are consistent with pathological products such as turbid phlegm and blood stasis in TCM theory, which provides modern pathological evidence for the core pathogenesis of phlegm-stasis intermingling in AS [8]. In the pathological process of AS, turbid phlegm and blood stasis follow a dynamic evolutionary law: phlegm generating stasis, phlegm-stasis intermingling, and lingering combination inducing disease. Among them, turbid phlegm acts as the initial pathogenic factor, which is mostly caused by dysfunction of zang-fu organs in transportation and transformation, leading to endogenous lipid-turbidity that stagnates blood vessels. This corresponds to the initial vascular injury induced by lipid infiltration and fatty-streak formation in early-stage AS. Long-term retention of turbid phlegm in vessels hinders the circulation of qi and blood, resulting in sluggish blood flow and obstructed collaterals. As an intermediate link in disease progression, blood stasis continuously aggravates the condition and is closely related to the pathological changes of vascular wall during fibrous plaque formation. The long-term combination of turbid phlegm and blood stasis exacerbates vascular injury and plaque progression, which serves as the core pathological mechanism underlying the occurrence and development of AS [8-10]. Eventually, turbid phlegm and blood stasis interweave and stagnate in collaterals to cause vascular injury. Over time, phlegm-stasis intermingling becomes the dominant

pathogenesis of AS.

3. Relationship Between the Theory of Intermingled Phlegm and Blood Stasis and Mitophagy

Mitophagy is a conserved biological process that selectively recognizes, encapsulates, and degrades damaged mitochondria via autophagic mechanisms, acting as the core guarantee of intracellular mitochondrial homeostasis and functional stability [11]. In the progression of AS, functional mitophagy exerts significant vascular protective effects. By clearing damaged mitochondria that produce excessive reactive oxygen species (ROS), mitophagy inhibits ROS overaccumulation, alleviates oxidative stress injury, and maintains mitochondrial structural integrity and normal cellular energy metabolism. The dynamic progression of phlegm-stasis intermingling lesions in AS can be effectively explained by the imbalance between mitophagy and mitochondrial apoptosis. The homeostatic disequilibrium of mitophagy and apoptosis is a critical molecular mechanism driving AS progression. Under pathological conditions, insufficient mitophagy and abnormally activated mitochondrial apoptosis induce excessive apoptosis of vascular smooth muscle cells (VSMCs) and macrophages [12]. This pathological change accelerates lipid deposition in the vascular wall, reduces atherosclerotic plaque stability, and further triggers plaque rupture and secondary thrombosis [13]. Conversely, secondary thrombosis caused by plaque rupture further exacerbates vascular obstruction induced by phlegm-stasis accumulation, which inhibits protective mitophagy activity and aggravates vascular cell apoptotic injury, ultimately forming a vicious cycle of progressive vascular damage [14]. Modern pharmacological studies have demonstrated that TCM therapies with the effects of eliminating phlegm and resolving blood stasis can improve mitochondrial functional activity. These interventions can upregulate mitophagy levels to clear damaged mitochondria, reduce excessive ROS accumulation and oxidative stress injury, and exert anti-atherosclerotic vascular protective effects [15]. Collectively, phlegm-stasis intermingling is closely correlated with mitophagy dysfunction throughout the occurrence and development of AS. The pathological progression of AS is essentially regulated by mitophagy activity, and the anti-atherosclerotic mechanism of TCM phlegm-resolving and stasis-removing therapies is highly consistent with the molecular pathway of mitophagy-mediated damaged mitochondrial clearance [16].

4. Regulatory Mechanism of Mitophagy in Atherosclerosis Progression

Mitophagy is an essential part of the mitochondrial quality control system, which removes impaired mitochondria through selective autophagy to maintain intracellular mitochondrial homeostasis and prevent metabolic disorder [17]. Under physiological conditions, basal mitophagy clears spontaneously damaged mitochondria, inhibits reactive oxygen species accumulation, and reduces oxidative stress-mediated cellular damage, maintaining vascular physiological stability [18]. Under pathological conditions, defective mitophagy fails to eliminate dysfunctional mitochondria in a timely manner, leading to persistent accumulation of damaged mitochondria and excessive ROS production, which triggers a series of vascular pathological injuries [19]. Mitophagy participates in the whole progression of AS, and regulates multiple core pathological links including vascular endothelial injury, foam cell formation, and plaque stability, thus playing a decisive regulatory role in AS development. Mitophagy mediates the recognition, translocation and lysosomal degradation of damaged mitochondria through multiple conserved signaling pathways, among which the PINK1/Parkin ubiquitin-dependent pathway and BNIP3/NIX receptor-mediated pathway are the most classic and well-studied regulatory mechanisms [20]. The PINK1/Parkin signaling pathway is currently the most clearly defined core pathway regulating mitophagy in mammalian vascular cells [21]. Mitochondrial membrane potential depolarization stabilizes PINK1 expression on the mitochondrial outer membrane, and PINK1-mediated ubiquitin phosphorylation further activates and

recruits Parkin, ultimately triggering complete mitophagy activation [22]. PINK1/Parkin-dependent mitophagy facilitates the clearance of impaired mitochondria, inhibits excessive apoptosis of vascular smooth muscle cells under lipid stress, and improves vascular microenvironment [23].

Multiple studies have confirmed the protective effect of PINK1/Parkin-mediated mitophagy on AS. Ma et al. reported that melatonin activates Sirt3/FoxO3a-Parkin-dependent mitophagy, which reduces mitochondrial ROS generation, inhibits NLRP3 inflammasome activation, and alleviates atherosclerotic lesion severity [24]. Notably, the PINK1/Parkin pathway exerts a bidirectional regulatory effect on AS progression, showing a dual role of protection and pathogenicity depending on activation intensity and pathological context. He et al. found that excessive activation of AMPK-mediated PINK1/Parkin-dependent mitophagy induced by endogenous APJ ligand promotes abnormal proliferation of vascular smooth muscle cells, thereby aggravating atherosclerotic lesions in ApoE mice [25]. In summary, mitophagy dysfunction is a key driver of AS progression. Insufficient mitophagy triggers oxidative stress and inflammatory cascade responses to initiate early atherosclerotic lesions, while excessive mitophagy induces abnormal proliferation and phenotypic transformation of vascular cells to deteriorate advanced plaques. Moderate mitophagy maintains vascular cell survival and plaque stability, while mitophagy imbalance (insufficiency or overactivation) accelerates lipid deposition, vascular remodeling and plaque instability. Therefore, mitophagy serves as a pivotal dynamic regulator of the initiation, progression and homeostasis maintenance of atherosclerosis, and its functional imbalance is highly consistent with the progressive deterioration characteristics of TCM phlegm-stasis intermingling lesions.

5. Advances in the Treatment of Atherosclerosis by Traditional Chinese Medicine Through the Regulation of Mitophagy

Traditional Chinese medicine (TCM) exerts unique therapeutic superiority against atherosclerosis (AS) through multi-target and multi-pathway synergistic modulation. The restoration of mitophagy homeostasis effectively ameliorates lipid metabolic disorders, oxidative stress injury and chronic vascular inflammation, representing one of the core mechanisms of TCM intervention for AS. The pathological evolution of AS dominated by phlegm-stasis intermingling is highly consistent with the occurrence and deterioration of mitophagy dysfunction. Mechanistically, the classic TCM therapeutic principle of resolving phlegm and removing blood stasis can produce anti-atherosclerotic effects by enhancing mitophagic flux, promoting the clearance of damaged mitochondria, restoring mitochondrial functional homeostasis, and ultimately halting the continuous progression of AS lesions.

5.1. Progress in the Intervention of Individual Chinese Medicine Components

A variety of active compounds isolated from Chinese materia medica have been shown to exert anti-atherosclerotic effects by activating mitophagy and modulating autophagy-related signaling pathways. Nobiletin, an active constituent derived from citrus peel, has been reported to upregulate the expression of LC3-II, Beclin-1, PINK1, and Parkin in ApoE^{-/-} mice, thereby activating the mitophagy pathway, inhibiting NLRP3 inflammasome activation, and effectively suppressing macrophage foam cell formation [26]. Li Yuting [27] revealed that apigenin promoted mitophagy via activation of the PINK1/Parkin pathway, improved mitochondrial function, alleviated vascular oxidative stress and inflammatory responses, and consequently retarded the progression of AS. Zhu Junfeng et al. demonstrated that Panax notoginseng saponins (PNS), the principal bioactive extract of Panax notoginseng, increased the protein levels of LC3-II, Beclin-1, PINK1 and Parkin in ApoE^{-/-} mice, enhanced vascular mitophagy activity and thereby delayed the progression of atherosclerotic plaques [28].

5.2. Progress in the Intervention of Chinese Herbal Formulas

TCM formulas are characterized by holistic regulation and syndrome differentiation and treatment, which exert unique advantages in the prevention and treatment of chronic vascular diseases. In recent years, accumulating pharmacological studies have demonstrated that TCM formulas can alleviate atherosclerotic lesions and delay disease progression by modulating mitophagy homeostasis. As a classic TCM prescription with the effects of tonifying qi, strengthening the spleen, eliminating phlegm and removing blood stasis, the Huayu Qutan Formula is precisely formulated to target the core pathological mechanism of “phlegm-stasis intertwining” underlying atherosclerosis. Chen et al. [15] demonstrated that Huayu Qutan Recipe significantly upregulated the protein expression of PINK1, Parkin, and LC3-II in hepatic tissues of atherosclerotic rabbits. By activating PINK1/Parkin-dependent mitophagy, this formula reduced excessive hepatic lipid deposition, improved lipid metabolic disorders, and ultimately delayed atherosclerotic pathological progression. Cai et al. [29] conducted animal experiments and found that Tan-Yu Tongzhi Formula could regulate the expression of autophagy-related proteins to achieve bidirectional modulation of angiogenesis. Specifically, it suppressed pathological neovascularization within atherosclerotic plaques, thereby stabilizing vulnerable plaques and inhibiting atherosclerotic progression. Multiple *in vivo* and *in vitro* studies have further confirmed the therapeutic effects of TCM formulas against atherosclerosis. As validated by experimental evidence, Xinmaikang Formula activates macrophage mitophagy via the PINK1/Parkin signaling pathway, reduces plaque area and vascular lipid deposition in ApoE^{-/-} mice, and exerts prominent anti-atherosclerotic effects [30]. Soufeng Qutan Huayu Formula, which has the functions of dispelling wind, resolving phlegm, strengthening the spleen, and removing blood stasis, was found to activate the PINK1/Parkin signaling pathway and upregulate the expression of mitophagy-related proteins, including LC3B-II, Beclin-1, and PINK1, thereby delaying the progression of AS [31]. In summary, TCM formulas with the therapeutic actions of resolving phlegm, removing blood stasis, and dispelling wind are developed based on the core pathogenesis of phlegm–stasis intertwining in atherosclerosis. Through multi-target and multi-pathway synergistic regulation of mitophagy and restoration of mitochondrial homeostasis, these formulas provide solid experimental evidence supporting the prevention and treatment of AS by TCM.

6. Conclusion

Mitophagy constitutes a vital mechanism for maintaining mitochondrial quality control, preserving cellular energy metabolic homeostasis, and inhibiting inflammatory responses, and it plays a critical role in the pathogenesis of atherosclerosis (AS). In recent years, mitophagy has gradually emerged as a hot research topic for exploring the pathogenesis and therapeutic interventions of AS. Traditional Chinese medicine (TCM) confers unique advantages in the prevention and treatment of AS owing to its multi-pathway, multi-target and holistic regulatory properties. Accumulating evidence demonstrates that numerous Chinese herbal monomers and formulas can target mitophagy-related signaling pathways, including PINK1/Parkin and BNIP3L/NIX, improve mitochondrial function, alleviate oxidative stress, inflammatory injury and lipid metabolic disorders, and thereby effectively delay AS progression. From the TCM pathogenesis perspective, “intermingled phlegm and blood stasis” is recognized as the core pathological mechanism of atherosclerosis, which shows high consistency with mitophagy-dysfunction-related pathological changes in modern medicine. This indicates that modulation of mitophagy by resolving-phlegm-and-removing-blood-stasis therapies may serve as one of the key mechanisms underlying TCM intervention against AS. Nevertheless, several limitations remain in current studies. Most available evidence is derived from animal and cellular experiments, whereas systematic clinical investigations are scarce. Furthermore, the majority of studies focus on single-mechanism exploration, and our understanding of the multi-component,

multi-target and multi-pathway synergistic regulation of mitophagy exerted by TCM formulas remains inadequate. The complicated regulatory network by which multi-component formulas modulate mitophagy has not been fully elucidated. Future research should carry out randomized controlled trials and real-world studies, combined with multi-omics strategies, network pharmacology and artificial-intelligence-based technologies, to systematically clarify the core mechanisms and pharmacological material basis of TCM-mediated mitophagy regulation against AS, as well as to optimize therapeutic efficacy evaluation systems. Deep integration of traditional Chinese and Western medicine may provide novel theoretical evidence and research avenues for the precise prevention and individualized treatment of atherosclerosis.

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