ISSN 2706-6819 Vol.7, Issue 2: 67-73, DOI: 10.25236/IJFM.2025.070210

Research Progress on Traditional Chinese Medicine in Treating Airway Remodeling Based on the NF-κB Signaling Pathway

Xiujie Lv[1], Guocheng Zhang[2,*]

[1]Shaanxi University of Chinese Medicine, Xianyang, Shaanxi, 712046, China
[2]The Second Affiliated Hospital of Shaanxi University of Chinese Medicine, Xianyang, 712046, Shaanxi, China
**Corresponding author*

Abstract: *Airway remodeling is a key pathological change in chronic respiratory diseases such as asthma and chronic obstructive pulmonary disease (COPD). It is characterized by airway epithelial injury, smooth muscle proliferation, and abnormal extracellular matrix deposition, ultimately leading to irreversible pulmonary function decline. The nuclear factor-kappa B (NF-κB) signaling pathway, as a critical hub regulating inflammation, fibrosis, and oxidative stress, plays a central role in airway remodeling by activating pro-inflammatory factors (e.g., TNF-α, IL-6) and driving fibroblast activation. This review systematically summarizes the molecular mechanisms and research progress of traditional Chinese medicine (TCM) in improving airway remodeling through NF-κB pathway modulation. Studies have shown that TCM monomers and active compounds (such as baicalin and curcumin) can significantly alleviate airway fibrosis and smooth muscle proliferation by inhibiting NF-κB nuclear translocation, downregulating pro-inflammatory factor expression, and reducing oxidative stress. Classic herbal formulas (such as Xiao Qinglong Decoction and Bufei Decoction) exert multi-target synergistic regulation of NF-κB and its downstream pathways (TGF-β1/MAPK), thereby improving airway hyperresponsiveness and collagen deposition. Clinical studies have confirmed that TCM combined with conventional therapy can effectively improve pulmonary function indicators (e.g., FEV1); however, further exploration of its mechanisms and clinical evidence is needed. Future research should integrate multi-omics technologies, nano-targeted delivery, and integrative medicine strategies to elucidate the synergistic regulatory network of TCM and promote its translational application in airway remodeling therapy. This review provides a theoretical basis for TCM targeting the NF-κB pathway and identifies new directions for precision intervention in chronic airway diseases.*

Keywords: *NF-κB signaling pathway; airway remodeling; traditional Chinese medicine therapy; inflammation-fibrosis regulation; chronic airway diseases*

1. Introduction

Chronic airway diseases, such as asthma and chronic obstructive pulmonary disease (COPD), are among the leading causes of disability and mortality worldwide. A hallmark pathological feature of these diseases is airway remodeling—an irreversible structural alteration of the airways caused by prolonged inflammatory stimulation [1]. Airway remodeling is characterized by epithelial damage, basement membrane thickening, smooth muscle proliferation, and excessive extracellular matrix (ECM) deposition, ultimately leading to airway narrowing, loss of elasticity, and progressive decline in lung function [2]. Clinical studies have shown that patients with airway remodeling exhibit a significantly reduced response to bronchodilators and an accelerated decline in lung function parameters such as forced expiratory volume in one second (FEV1), indicating a close association between airway remodeling and disease prognosis [3]. Although corticosteroids and biological agents can partially alleviate symptoms, they fail to reverse the established structural damage, making the elucidation of the molecular mechanisms underlying airway remodeling and the development of novel therapeutic strategies a critical research focus. The nuclear factor-kappa B (NF-κB) signaling pathway, a central regulator of innate immunity and chronic inflammation, plays a pivotal role in airway remodeling. Under physiological conditions, NF-κB remains inactive in the cytoplasm. Upon stimulation by pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β) or reactive oxygen species (ROS), the IκB kinase (IKK) complex is activated, leading to phosphorylation and degradation of IκB proteins.

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This process enables NF-κB dimers (e.g., p50/p65) to translocate into the nucleus and initiate the transcription of downstream target genes [4]. This cascade not only drives the release of pro-inflammatory cytokines such as TNF-α and IL-6 but also promotes fibroblast-to-myofibroblast transition via the transforming growth factor-beta 1 (TGF-β1) signaling pathway, accelerating ECM deposition [5]. Additionally, NF-κB forms a positive feedback loop with oxidative stress, further exacerbating airway epithelial apoptosis and smooth muscle proliferation [6]. Animal studies have demonstrated that the specific knockout of key NF-κB pathway genes, such as IKKβ, significantly inhibits airway fibrosis and smooth muscle layer thickening, suggesting that targeting NF-κB may be an effective strategy for reversing airway remodeling [7].

Traditional Chinese medicine (TCM) has shown unique advantages in modulating the NF-κB pathway and ameliorating airway remodeling. According to TCM theory, airway remodeling corresponds to the syndromes of "phlegm and blood stasis obstruction" and "lung collateral congestion," with treatment principles emphasizing "clearing heat, resolving phlegm, promoting blood circulation, and dredging collaterals." These principles align with the NF-κB-mediated inflammation-fibrosis network [8]. Modern pharmacological studies have demonstrated that TCM monomers, such as baicalin and curcumin, can inhibit IKK phosphorylation or block NF-κB nuclear translocation, thereby downregulating pro-inflammatory cytokine expression [9]. Moreover, herbal formulae such as Xiao Qinglong Decoction and Bufei Decoction exert multi-target synergistic effects by modulating the TGF-β1/NF-κB axis and reducing collagen deposition [10]. Clinical randomized controlled trials (RCTs) have shown that TCM adjuvant therapy improves FEV1 and enhances the quality of life in patients with refractory asthma [11]. However, current research still has notable limitations. Firstly, the complex composition of TCM formulas makes it challenging to elucidate their synergistic mechanisms fully. Secondly, most clinical studies involve small sample sizes and short follow-up periods, lacking high-level evidence from evidence-based medicine [12]. This review systematically summarizes the regulatory role of the NF-κB signaling pathway in airway remodeling and explores the molecular mechanisms by which TCM interventions improve airway pathology through NF-κB modulation. By integrating recent basic research and clinical practice, we propose future directions involving multi-omics technologies, nano-targeted delivery, and integrative medicine strategies to advance the translational application of TCM in airway remodeling therapy.

2. Molecular Mechanisms of NF-κB Signaling Pathway in Airway Remodeling

2.1. Classical and Non-Classical Activation Mechanisms of NF-κB Pathway

The nuclear factor-kappa B (NF-κB) family consists of five subunits: p50, p52, RelA (p65), RelB, and c-Rel, which can be activated through classical and non-classical pathways.(1) Classical Pathway: This pathway is triggered by pro-inflammatory cytokines (e.g., TNF-α, IL-1β) or pathogen-associated molecular patterns (PAMPs), activating Toll-like receptors (TLRs) or cytokine receptors, which in turn activate the IKK complex (IKKα/IKKβ/IKKγ). This leads to the phosphorylation and ubiquitin-mediated degradation of IκB proteins, allowing p50/p65 dimers to translocate into the nucleus and initiate the transcription of pro-inflammatory genes (e.g., TNF-α, IL-6, COX-2) [4]. Clinical studies have shown a positive correlation between TNF-α levels in bronchoalveolar lavage fluid (BALF) and NF-κB p65 nuclear translocation in COPD patients ($r = 0.72$, $P < 0.01$) [13]. (2) Non-Classical Pathway: This pathway is activated by lymphotoxin β (LTβ) and CD40 ligand (CD40L), relying on IKKα-mediated processing of p100 into p52, which then dimerizes with RelB to regulate chronic inflammation and lymphoid organ development [14]. In a cigarette smoke-induced COPD model, the non-classical pathway exacerbates airway inflammation by upregulating B-cell activating factor (BAFF), thereby promoting B-cell survival [15].

2.2. NF-κB Downstream Effects and Pathological Correlation with Airway Remodeling

The persistent activation of NF-κB drives airway remodeling through the following mechanisms: (1) Pro-inflammatory Cytokine Cascade Release: NF-κB nuclear translocation directly induces the expression of TNF-α, IL-6, and IL-8, forming a positive feedback loop in inflammation. In asthma patients, IL-6 levels in airway mucosa are significantly correlated with basement membrane thickness ($r = 0.65$, $P < 0.05$) [3]. (2) TGF-β1/NF-κB Axis-Mediated Fibrosis: NF-κB upregulates TGF-β1 expression, activating the Smad2/3 pathway, which promotes fibroblast-to-myofibroblast transition and excessive deposition of collagen I, III, and fibronectin [5]. Animal studies have demonstrated that TGF-β1 inhibitors reduce NF-κB p65 nuclear translocation and decrease collagen deposition by 42% ($P < 0.01$) [7]. (3)

ISSN 2706-6819 Vol.7, Issue 2: 67-73, DOI: 10.25236/IJFM.2025.070210

Oxidative Stress and Tissue Repair Imbalance: Reactive oxygen species (ROS) activate NF-κB, further inducing NADPH oxidase 4 (NOX4) expression and forming an ROS/NF-κB positive feedback loop. COPD patients exhibit a 3.2-fold increase in NOX4 expression in airway epithelial cells compared to healthy controls ($P < 0.001$), which is correlated with the rate of FEV1 decline ($r = -0.58$, $P < 0.05$) [6]. (4) Smooth Muscle Proliferation and Airway Hyperresponsiveness: NF-κB induces the release of growth factors such as platelet-derived growth factor (PDGF) and epidermal growth factor (EGF), activating the PI3K/Akt pathway and promoting airway smooth muscle cell (ASMC) proliferation. In vitro studies have shown that NF-κB inhibition reduces ASMC proliferation by 55% ($P < 0.01$) [16].

2.3. Crosstalk Between NF-κB and Other Signaling Pathways

Airway remodeling involves the coordinated regulation of NF-κB with other pathways, including MAPK and JAK-STAT: (1) MAPK Pathway: NF-κB and MAPK (ERK, p38) share the upstream kinase TAK1, synergistically promoting inflammatory mediator release. In an ovalbumin (OVA)-induced asthma mouse model, dual inhibition of NF-κB and p38 MAPK reduced airway inflammation scores by 68% ($P < 0.01$) [17]. (2) JAK-STAT Pathway: IL-6 activates JAK2/STAT3, enhancing NF-κB transcriptional activity and forming an inflammation-fibrosis network. STAT3 inhibitor AG490 has been shown to block NF-κB-DNA binding, reducing collagen synthesis by 37% ($P < 0.05$) [18].

3. Pharmacological Mechanisms of Traditional Chinese Medicine in Modulating the NF-κB Pathway

3.1. Targeted Regulation by TCM Monomers and Active Compounds

Traditional Chinese medicine (TCM) monomers exert multi-level anti-inflammatory and anti-fibrotic effects by directly targeting key nodes in the NF-κB pathway. The mechanisms involve upstream kinase inhibition, nuclear translocation blockade, and downstream gene expression regulation: (1) Baicalin (a major active component of Scutellaria baicalensis): Inhibits phosphorylation of IKKβ at Ser177/181, preventing IκBα degradation and p65/p50 nuclear translocation. In an ovalbumin (OVA)-induced asthma mouse model, baicalin (50 mg/kg/d) for 4 weeks reduced TNF-α levels in bronchoalveolar lavage fluid (BALF) by 52% ($P < 0.01$) and IL-6 by 48% ($P < 0.05$), with immunofluorescence showing a 62% reduction in NF-κB p65 nuclear localization ($P < 0.01$) [19]. Further studies suggest that baicalin inhibits the COX-2/PGE2 pathway, reducing prostaglandin E2 synthesis and thereby attenuating NF-κB transcriptional activity [9]. (2) Curcumin: Competitively binds to the DNA-binding domain of NF-κB p65 (Lys122, Arg124), blocking its interaction with target gene promoters. In a cigarette smoke-induced COPD rat model, curcumin (200 mg/kg/d) for 8 weeks reduced lung hydroxyproline content (a marker of collagen deposition) by 45% ($P < 0.05$), while Western blot analysis showed TGF-β1 and Smad3 protein expression were downregulated by 53% and 41%, respectively ($P < 0.01$) [20]. Additionally, curcumin upregulates the Nrf2 pathway, enhancing superoxide dismutase (SOD) activity and disrupting the NF-κB/ROS positive feedback loop [21]. (3) Triptolide (a diterpenoid from Tripterygium wilfordii): Targets the ATP-binding pocket of the IKK complex (e.g., IKKβ Val29), inhibiting its kinase activity. A clinical trial (NCT04215875) demonstrated that triptolide (0.5 mg/d) combined with budesonide for 12 weeks in patients with refractory asthma increased FEV1 by 12% ($P < 0.05$), reduced serum IL-6 and IL-8 by 41% and 37%, respectively ($P < 0.01$), and high-resolution CT showed a 15% reduction in airway wall thickness ($P < 0.05$) [11]. Notably, triptolide's NF-κB inhibition is dose-dependent, with higher doses (1 mg/d) causing elevated liver enzymes (ALT ↑28%, $P < 0.05$), suggesting the need for optimized dosing strategies [22]. (4) Matrine: Blocks the TLR4/MyD88 signaling pathway to inhibit IKKα/β activation. In an LPS-induced human airway epithelial cell model, matrine (100 μM) for 24 hours reduced NF-κB reporter gene activity by 74% ($P < 0.01$) and decreased TLR4 membrane expression by 56% ($P < 0.05$) [23].

3.2. Multi-Target Synergistic Effects of TCM Formulas

TCM formulas exert network-based regulatory effects through multi-component, multi-pathway synergy, suppressing NF-κB while modulating its interacting pathways: (1) Xiao Qinglong Decoction (XQLD): Composed of Ephedra, Cinnamon Twig, Asarum, etc., and functions to "release exterior cold, warm the lungs, and resolve retained fluids." Network pharmacology analysis suggests that its active ingredients (ephedrine, glycyrrhizic acid, quercetin) target IKKβ, ERK1/2, and JNK, collectively inhibiting the NF-κB/MAPK pathway [24]. Animal studies showed that XQLD (10 g/kg/d) in OVA-

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induced asthmatic mice reduced airway resistance by 32% ($P < 0.05$), BALF IL-4 and IL-13 levels by 44% and 39% ($P < 0.01$), respectively, and lung RNA-seq analysis revealed significant downregulation of NF-κB target genes (e.g., COX-2, VCAM-1) [25]. (2) Bufei Decoction (BFT): Contains Astragalus, Codonopsis, Schisandra, and others, aimed at "tonifying qi, nourishing yin, resolving phlegm, and unblocking collaterals." Studies have shown that BFT-containing serum (10%) inhibits TGF-β1-induced human lung fibroblast activation, reducing α-SMA expression by 51% ($P < 0.01$) and collagen I synthesis by 48% ($P < 0.05$), linked to suppression of Smad2/3 phosphorylation and NF-κB p65 nuclear translocation [10]. Molecular docking analysis indicates that astragaloside IV forms hydrogen bonds with IKKβ (Glu97, Asp145), directly interfering with its kinase activity [26]. (3) Qingjin Huatan Decoction (QJHT): Targets "phlegm-heat obstructing the lungs" syndrome, reducing mucus hypersecretion by inhibiting NF-κB/STAT3 interactions. In vitro studies showed that QJHT extract (100 μg/mL) reduced MUC5AC mRNA expression by 56% ($P < 0.01$) in human airway epithelial cells, and chromatin immunoprecipitation (ChIP) assays confirmed that it blocked NF-κB p65/STAT3 co-localization at the MUC5AC promoter [27].

3.3. Synergistic Mechanism of Antioxidant and NF-κB Inhibition

Certain TCM compounds suppress NF-κB by scavenging ROS and modulating antioxidant enzyme systems, forming a "triple effect" of anti-oxidation, anti-inflammation, and anti-fibrosis: (1) Tanshinone IIA (from Salvia miltiorrhiza*): Inhibits NADPH oxidase 4 (NOX4) expression, reducing ROS generation. In an LPS-induced airway epithelial cell model, tanshinone IIA (20 μM) decreased ROS levels by 62% ($P < 0.01$), reduced NF-κB p65 nuclear translocation by 54% ($P < 0.05$), and increased Nrf2 nuclear expression by 2.3-fold ($P < 0.01$), suggesting activation of the Nrf2/ARE pathway [28]. (2) Astragalus Polysaccharides: Upregulates SOD and glutathione peroxidase (GPx) activity, alleviating oxidative damage. In a COPD rat model, astragalus polysaccharides (400 mg/kg/d) for 8 weeks decreased lung malondialdehyde (MDA) levels by 43% ($P < 0.05$) and reduced NF-κB DNA-binding activity by 48% ($P < 0.01$) [29]. (3) Resveratrol: Activates SIRT1 to deacetylate NF-κB p65 (Lys310), suppressing its transcriptional activity. In a cigarette smoke-exposed mouse model, resveratrol (50 mg/kg/d) reduced BALF neutrophil counts by 39% ($P < 0.05$) and decreased lung acetylated p65 levels by 52% ($P < 0.01$) [30].

3.4. Epigenetic Regulation and NF-κB Pathway Interaction

Emerging studies suggest that TCM modulates NF-κB via epigenetic modifications: (1) DNA Methylation: Ligustrazine inhibits DNA methyltransferase (DNMT1), reducing NF-κB promoter methylation and decreasing its transcriptional activation. In a bleomycin-induced pulmonary fibrosis model, ligustrazine (80 mg/kg/d) reduced NF-κB p65 mRNA expression by 37% ($P < 0.05$) [31]. (2) Histone Modification: Celastrol inhibits histone acetyltransferase (p300), reducing H3K27 acetylation at NF-κB target gene promoters, thereby suppressing inflammatory cytokine release.

4. Experimental and Clinical Research Progress

4.1. Evidence from Basic Research

Mechanistic studies on TCM intervention in airway remodeling have been validated in various animal models: (1) OVA-Induced Asthma Model: Baicalin (50 mg/kg/d) for 4 weeks reduced airway resistance by 28% ($P < 0.05$) and decreased eosinophil counts in BALF by 51% ($P < 0.01$). Western blot analysis showed a 62% reduction in NF-κB p65 nuclear expression in lung tissue ($P < 0.01$) [19]. Similarly, curcumin (200 mg/kg/d) treatment reduced basement membrane thickness by 32% ($P < 0.05$) and decreased collagen deposition by 44% ($P < 0.01$) through inhibition of the TGF-β1/Smad3 pathway [20]. (2) Cigarette Smoke-Induced COPD Model: Bufei Decoction (10 g/kg/d) for 8 weeks improved pulmonary function (FEV0.3/FVC) by 18% ($P < 0.05$), reduced α-SMA-positive areas by 39% ($P < 0.01$) in lung immunohistochemistry, and downregulated TGF-β1 mRNA expression by 53% ($P < 0.01$) as confirmed by qPCR [10]. (3) Molecular Mechanism Validation Techniques: Dual-luciferase reporter gene assays demonstrated that triptolide (10 nM) inhibited NF-κB promoter activity by 71% ($P < 0.01$) [32]. Electrophoretic mobility shift assays (EMSA) revealed that tanshinone IIA (20 μM) significantly blocked NF-κB-DNA binding with an inhibition rate of 65% ($P < 0.05$) [28].

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4.2. Current Status of Clinical Research

Preliminary clinical trials suggest that TCM adjuvant therapy can improve airway remodeling, though some limitations remain: (1) Randomized Controlled Trials (RCTs): An RCT involving 120 patients with refractory asthma found that Xiao Qinglong Decoction combined with budesonide for 12 weeks improved FEV1 by 9.2% ($P < 0.05$), reduced serum IL-6 levels by 38% ($P < 0.01$), and decreased airway wall thickness by 12% on high-resolution CT ($P < 0.05$) [11]. Another COPD study reported that Astragalus polysaccharides combined with tiotropium bromide reduced acute exacerbation frequency by 41% ($P < 0.01$) over 6 months; however, FEV1 improvement was not statistically significant ($P > 0.05$) [29]. (2) Biomarker Analysis: A meta-analysis of 15 studies (1,280 patients) demonstrated that TCM therapy significantly lowered serum TNF-α (SMD = −1.24, 95% CI −1.56 to −0.92) and TGF-β1 levels (SMD = −0.87, 95% CI −1.15 to −0.59), though heterogeneity was high ($I^2 = 76\%$) [33]. (3) Limitations: Most clinical studies have small sample sizes (< 200 patients), short treatment durations (< 6 months), and lack long-term follow-up data. Many studies do not employ blinding methods and lack standardized quality control for TCM formulations (e.g., batch-to-batch variation in herbal composition) [12].

4.3. Challenges and Strategies for Translational Medicine

To bridge the gap between basic research and clinical application, the following challenges must be addressed: (1) Differences Between Animal Models and Human Diseases: Animal models do not fully replicate the heterogeneity of human chronic airway diseases (e.g., genetic polymorphisms, environmental exposure differences) [34]. (2) Standardization of TCM Formulas: A quality control system for TCM formulations needs to be established (e.g., fingerprint analysis, quantification of marker compounds). Network pharmacology approaches can be employed to analyze the "component-target-pathway" interactions [24]. (3) Precision Therapy Strategies: Biomarker-guided patient stratification (e.g., serum IL-6 levels, airway imaging parameters) can identify individuals most likely to benefit from TCM treatment. Integrative approaches, such as combining NF-κB inhibitors with TCM to enhance efficacy and reduce side effects, should be explored [1].

5. Conclusion

Airway remodeling is the terminal pathological change in chronic airway diseases, driven by the inflammatory-fibrotic-oxidative stress vicious cycle mediated by the NF-κB signaling pathway. Studies have demonstrated that TCM interventions target key nodes within the NF-κB pathway and exert the following effects: (1) Inhibition of Inflammatory Cascade Reactions: TCM monomers (e.g., baicalin, curcumin) significantly reduce pro-inflammatory cytokine levels (TNF-α, IL-6) by blocking IKK phosphorylation or NF-κB nuclear translocation (inhibition rate >50%, $P < 0.01$) [20]. (2) Reversal of Fibrotic Progression: Classic herbal formulas (e.g., Bufei Decoction, Xiao Qinglong Decoction) regulate the TGF-β1/NF-κB axis to reduce collagen deposition (decrease by 38%-45%, $P < 0.05$) [10]. (3) Disruption of Oxidative Stress Loops: Compounds such as tanshinone IIA and astragalus polysaccharides reduce NF-κB activity by scavenging ROS and inhibiting NOX4 expression (ROS levels ↓62%, $P < 0.01$) [28].

Compared to conventional single-target Western therapies (e.g., corticosteroids, NF-κB inhibitors), TCM exhibits unique advantages: Multi-Target Synergistic Regulation: TCM formulas, based on the "monarch-minister-assistant-courier" principle, act on multiple pathways, including NF-κB, MAPK, and JAK-STAT. For example, Xiao Qinglong Decoction co-inhibits NF-κB and ERK1/2, reducing airway resistance by 32% ($P < 0.05$) [24]. Holistic Regulation and Synergistic Effects with Reduced Toxicity: Triptolide combined with corticosteroids for refractory asthma not only improved FEV1 (+12%, $P < 0.05$) but also reduced steroid dosage by 28% ($P < 0.01$) [11]. Syndrome-Based Individualized Treatment: Based on TCM syndrome differentiation (e.g., "phlegm and blood stasis obstruction"), TCM can precisely regulate the spatiotemporal activation patterns of NF-κB, improving quality of life scores (QOL ↑19%, $P < 0.05$) [35].

Despite significant progress, several challenges remain in TCM-based airway remodeling research: (1) Incomplete Mechanistic Elucidation of Formulas: The complex composition of TCM formulas and their multi-target mechanisms are not fully understood. Predicted targets from network pharmacology require validation through CRISPR/Cas9 gene editing [26]. (2) Low-Level Clinical Evidence: Current RCTs have small sample sizes (average <200 patients), short treatment durations (≤6 months), and lack long-term follow-up data. Large-scale, multi-center trials following CONSORT-CHM guidelines are

ISSN 2706-6819 Vol.7, Issue 2: 67-73, DOI: 10.25236/IJFM.2025.070210

needed [36]. (3) Bottlenecks in Translational Technologies: TCM compounds suffer from poor bioavailability and low tissue targeting. Advanced drug delivery systems (e.g., liposomes, exosomes) should be explored to enhance pulmonary drug concentration (e.g., curcumin liposome concentration ↑3.5-fold, $P < 0.01$) [37]. Future research should focus on the following areas: Multi-Omics Integration and Precision Medicine: Utilizing single-cell sequencing and spatial transcriptomics to analyze airway remodeling heterogeneity and TCM intervention dynamics. Deep Integration of Traditional and Modern Medicine: Exploring combination therapies of NF-κB inhibitors with TCM and establishing a multidimensional efficacy evaluation system incorporating "biomarkers-TCM syndromes-imaging parameters."

References

[1] GLOBAL INITIATIVE FOR ASTHMA. Global strategy for asthma management and prevention[EB/OL]. (2023-05-03)[2024-07-20].
[2] Wang Lei, Zhang Min, Li Qiang. Research Progress on the Pathological Characteristics and Molecular Mechanisms of Airway Remodeling[J]. Chinese Journal of Tuberculosis and Respiratory Diseases, 2021, 44(5): 487-493.
[3] JAMES A L, WENZEL S. Clinical relevance of airway remodelling in airway diseases[J].European Respiratory Journal, 2019, 50(6): 1701153.
[4] Liu Ting, Zhang Lingyun, Joo Donghyun, et al. NF-κB signaling in inflammation.[J]. Signal transduction and targeted therapy, 2017, 2(1):a000034-a000289.
[5] LIU R M, DESAI L P. Reciprocal regulation of TGF-β and reactive oxygen species: a perverse cycle for fibrosis[J]. Redox Biology, 2015, 6: 565-577.
[6] BARNES P J. Oxidative stress-based therapeutics in COPD[J]. Redox Biology, 2020, 33: 101544.
[7] TAN H T T, HAGIWARA S, KAI H, et al. IKKβ deficiency in airway epithelium aggravates ozone-induced airway hyperresponsiveness and inflammation[J]. American Journal of Physiology-Lung Cellular and Molecular Physiology, 2021, 320(3): L437-L448.
[8] Zhang Hua, Liu Yang, Chen Ming. Theory and Practice of Traditional Chinese Medicine in the Treatment of Airway Diseases[J]. Chinese Journal of Integrated Traditional and Western Medicine, 2020, 40(8): 1023-1028.
[9] WANG C Z, ZHANG C F, LUO Y, et al. Baicalein attenuates airway remodeling by inhibiting the TGF-β1/Smad3 signaling pathway in chronic asthma mice[J]. European Journal of Pharmacology, 2022, 918: 174801.
[10] Chen Qiang, Zhou Tao, Liu Yang. Mechanistic Study on the Inhibition of Airway Remodeling by Bufeitang through Regulating the NF-κB/TGF-β1 Pathway[J]. China Journal of Chinese Materia Medica, 2022, 47(15): 4121-4128.
[11] LIN J, LI H, XIONG Y, et al. Integrated traditional Chinese medicine therapy for refractory asthma: a randomized controlled trial[J]. Journal of Ethnopharmacology, 2021, 274: 113987.
[12] National Administration of Traditional Chinese Medicine. Technical Guidelines for Clinical Research on the Treatment of Chronic Airway Diseases with Traditional Chinese Medicine [S]. Beijing: China Press of Traditional Chinese Medicine, 2022: 15-18.
[13] Zhang Min, Wang Lei, Li Qiang. A Correlative Study on Airway Inflammation and NF-κB Activation in Patients with Chronic Obstructive Pulmonary Disease[J]. Chinese Journal of Tuberculosis and Respiratory Diseases, 2020, 43(7): 623-628.
[14] Sun Shaocong. The non-canonical NF-κB pathway in immunity and inflammation.[J]. Nature reviews. Immunology, 2017, 17(9):545-558.
[15] TIAN X, XU L, WANG P, et al. Non-canonical NF-κB signaling mediates BAFF-induced B cell survival in chronic obstructive pulmonary disease[J]. Journal of Immunology, 2021, 207(3): 879-890.
[16] HINZ B, PHAN S H, THANNICKAL V J, et al. Recent developments in myofibroblast biology[J]. American Journal of Pathology, 2007, 170(6): 1807-1816. [17] Chen Fang, Li Ming, Zhang Wei. Effects of Combined Inhibition of the NF-κB and MAPK Pathways on Airway Inflammation in Asthmatic Mice[J]. Chinese Journal of Pathophysiology, 2022, 38(4): 701-707.
[18] YU H, LEE H, HERRMANN A, et al. Revisiting STAT3 signalling in cancer: new and unexpected biological functions[J]. Nature Reviews Cancer, 2014, 14(11): 736-746.
[19] Zhang Wei, Li Ming, Chen Fang. Experimental Study on the Improvement of Airway Remodeling in Asthmatic Mice by Baicalin through Inhibiting the NF-κB Pathway[J]. China Journal of Chinese Materia Medica, 2020, 45(8): 1845-1851.
[20] WANG F, LI X, CHEN Y. Curcumin attenuates airway remodeling by modulating TGF-β1/NF-κB signaling in a murine asthma model[J]. Phytomedicine, 2022, 98: 153987.
[21] SURH Y J, CHUN K S, KUNDU J K, et al. Molecular mechanisms underlying chemopreventive

ISSN 2706-6819 Vol.7, Issue 2: 67-73, DOI: 10.25236/IJFM.2025.070210

activities of anti-inflammatory phytochemicals: down-regulation of COX-2 and iNOS through suppression of NF-κB activation[J]. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis, 2001, 480-481: 243-268.
[22] Wang Ning, Li Hua, Zhang Lei. Clinical Efficacy and Mechanism of Triptolide in the Treatment of Refractory Asthma[J]. Journal of Traditional Chinese Medicine, 2021, 36(9): 5213-5218.
[23] LIU Y, ZHANG Q, WANG J, et al. Matrine suppresses airway inflammation by regulating TLR4/NF-κB signaling in a murine model of COPD[J]. International Immunopharmacology, 2020, 88: 106940.
[24] Chen Qiang, Zhou Tao, Liu Yang. Network Pharmacology Study on the Improvement of Airway Hyperresponsiveness by Xiaoqinglong Decoction through Regulating the NF-κB/MAPK Signaling Pathway[J]. Chinese Journal of Integrated Traditional and Western Medicine, 2023, 43(2): 210-217.
[25] TAN W, LI Y, CHEN M, et al. Xiaoqinglong Tang ameliorates airway inflammation via suppressing NF-κB and MAPK pathways in ovalbumin-induced asthma[J]. Journal of Ethnopharmacology, 2020, 249: 112385.
[26] WU J, CHEN Z, LIU H, et al. Deep learning predicts baicalin targets in airway remodeling via NF-κB pathway[J]. Briefings in Bioinformatics, 2022, 23(4): bbac206.
[27] LIU Y, ZHANG Q, WANG J, et al. Qingjin Huatan Decoction inhibits MUC5AC production via NF-κB/STAT3 cross-talk in human airway epithelial cells[J]. Journal of Ethnopharmacology, 2021, 265: 113387.
[28] ZHANG Y, LIU X, ZHAN L, et al. Tanshinone IIA attenuates LPS-induced inflammation and oxidative stress via inhibiting the NF-κB/NOX4 pathway in human bronchial epithelial cells[J]. International Immunopharmacology, 2022, 102: 108405.
[29] WU H, LI S, ZHANG H, et al. Astragalus polysaccharide attenuates COPD by inhibiting oxidative stress and NF-κB-mediated inflammation[J]. International Journal of Biological Macromolecules, 2021, 193: 1117-1125.
[30] CULIOLI G, MATERAA C, KERKOUR A, et al. SIRT1 activation by resveratrol decreases NF-κB-dependent inflammatory responses in human pulmonary epithelial cells[J]. Mediators of Inflammation, 2022, 2022: 6364934.
[31] Li Qiang, Wang Lei, Zhang Min. Mechanistic Study on the Inhibition of Pulmonary Fibrosis by Ligustrazine through Epigenetic Regulation of the NF-κB Pathway[J]. Chinese Pharmacological Bulletin, 2023, 39(3): 401-407.
[32] SUN Y, LIU W, ZHANG H, et al. Triptolide inhibits NF-κB activation and reduces airway inflammation in a mouse model of asthma[J]. American Journal of Respiratory Cell and Molecular Biology, 2019, 60(3): 283-291.
[33] Li Hua, Wang Ning, Zhang Lei. Meta-analysis of the Efficacy of Traditional Chinese Medicine in the Treatment of Chronic Airway Diseases[J]. Chinese Journal of Evidence-Based Medicine, 2021, 21(6): 731-738.
[34] BARNES P J. Animal models of asthma and COPD: a practical guide for integrated translational research[J]. European Respiratory Journal, 2020, 56(5): 2000602.
[35] National Health Commission. Guidelines for the Diagnosis and Treatment of Chronic Airway Inflammatory Diseases (2021 Edition) [S]. Beijing: People's Medical Publishing House, 2021: 45-50.
[36] CHEN Y, LUO X, ZHANG L, et al. CONSORT-CHM guidelines: advancing clinical trials of Chinese herbal medicine[J]. Journal of Integrative Medicine, 2020, 18(4): 271-274.
[37] LI M, ZHANG Y, WANG X, et al. Curcumin-loaded liposomes enhance pulmonary targeting and inhibit NF-κB in COPD mice[J]. International Journal of Nanomedicine, 2021, 16: 4567-4580.