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Research Progress on Traditional Chinese Medicine Regulating MAPK/p38 Signaling Pathway for Psoriasis Based on Network Pharmacology

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Abstract: ***Psoriasis is a chronic, recurrent immune-inflammatory skin disorder characterized by abnormal proliferation of keratinocytes and inflammatory infiltration of the dermis as its hallmark pathological features. Abnormal phosphorylation of the MAPK/p38 pathway promotes the release of pro-inflammatory factors such as IL-17 and IL-23, triggering an inflammatory cascade that contributes to disease progression. Western medical treatments can provide short-term symptom relief but are associated with high recurrence rates and significant adverse effects. Traditional Chinese medicine (TCM) offers therapeutic advantages due to its multi-component and multi-target mechanisms. Network pharmacology combined with molecular docking techniques enables a systematic elucidation of the action mechanisms of TCM formulations. This article focuses on the MAPK/p38 pathway to review relevant TCM interventions targeting this pathway in psoriasis management, analyze existing research limitations, explore future research directions, and provide theoretical insights for mechanistic studies and novel drug development in psoriasis treatment.***

Keywords: Psoriasis, Traditional Chinese medicine, MAPK/p38 signaling pathway, Network pharmacology.

1. Introduction

Psoriasis falls under the category of "Baibi" in traditional Chinese medicine (TCM). It features a protracted clinical course and high recurrence rate. In recent years, its incidence has kept rising with a tendency toward younger onset. The main clinical manifestations include erythema, silvery-white scales and infiltrative thickening of skin lesions. The core pathogenesis lies in the imbalance of Th17/Treg cells and excessive activation of the IL-17/IL-23 inflammatory axis, which induces abnormal proliferation of keratinocytes and persistent cutaneous inflammation [1]. Western medicine treatments including topical agents, immunosuppressants and biological agents can rapidly relieve acute-phase symptoms, yet they are confronted with limitations such as high recurrence rate, adverse reactions and considerable treatment costs [2-3]. TCM holds that the major pathogeneses of Baibi are blood heat, blood stasis and blood dryness, and corresponding therapeutic approaches of clearing heat and cooling blood, activating blood circulation to dissipate stasis, as well as nourishing blood and moistening dryness are adopted. TCM can regulate immune and inflammatory status holistically, and exhibits prominent advantages in reducing recurrence and ensuring medication safety [4]. Along with advances in modern research on TCM, network pharmacology combined with molecular docking technology can elucidate the multi-component and multi-target characteristics of TCM compound prescriptions, making it a vital research tool for signaling pathway mechanisms. Existing studies have verified that the MAPK/p38 signaling pathway serves as a key target pathway for TCM to intervene in inflammatory lesions of psoriasis [5]. Multiple TCM compound prescriptions can block inflammatory cascade reactions and ameliorate pathological lesions of psoriasis by suppressing abnormal activation of this pathway [6]. This paper integrates recent literature concerning network pharmacology, molecular docking and basic experiments, reviews research progress on TCM compound prescriptions treating psoriasis via targeting the MAPK/p38 pathway, analyzes deficiencies in current studies, and provides references for further mechanism exploration and clinical medication.

2. Correlation Mechanism Between MAPK/ p38 Signaling Pathway and Psoriasis

2.1 Structure and Biological Functions of MAPK/p38 Signaling Pathway

Mitogen-activated protein kinases (MAPKs) are a widely distributed serine/threonine protein kinase family in eukaryotic cells, responsible for transmitting extracellular signals intracellularly and regulating cell proliferation, differentiation, apoptosis, immune responses, and inflammatory stress. They play a pivotal role in the pathological processes of various inflammatory skin diseases and autoimmune disorders. The MAPK family is primarily divided into three major subfamilies: ERK, JNK, and p38 MAPK, with pathways that are both independent and subject to cross-regulation. Among these, the MAPK/p38 pathway is a critical subtype mediating cutaneous inflammation and immune stress, and has been extensively studied in psoriasis research. p38 comprises four subtypes: p38α, p38β, p38γ, and p38δ. p38α (MAPK14) is highly expressed in skin tissues and immune cells, serving as the central target underlying inflammatory damage in psoriasis [7- 8]. Under physiological conditions, p38 exists in a non-phosphorylated resting state; upon stimulation by inflammation or oxidative stress, it undergoes phosphorylation via the MAPKKK-MAPKK-MAPK three-step cascade, forming the activated form p-p38. Activated p-p38 enters the nucleus, activating transcription factors such as AP-1 and NF-κB, thereby promoting the expression of pro-inflammatory genes including IL-17, IL-23, TNF-α, and IL-6, and inducing inflammatory cell infiltration, abnormal proliferation of keratinocytes, and angiogenesis,

thereby driving the progression of chronic inflammatory skin diseases such as psoriasis.

2.2 Molecular Mechanism of MAPK/p38 Pathway Mediating Psoriasis Onset

Immunological dysregulation and persistent inflammatory activation constitute the core pathogenesis of psoriasis, with abnormal activation of Th17 cells and hyperactivity of the IL-17/IL-23 inflammatory axis persisting throughout the disease course. The MAPK/p38 pathway, as a critical upstream regulatory pathway of this inflammatory axis, participates in psoriasis progression through multiple mechanisms including immune response, cell proliferation, inflammatory infiltration, and vascular remodeling. At the immunological level, excessive activation of the MAPK/p38 pathway promotes dendritic cell secretion of IL-23, induces differentiation of naive T cells into Th17 cells, and leads to massive release of IL-17 and TNF-α, thereby establishing a vicious inflammatory cycle that disrupts cutaneous immune homeostasis [9]. At the epidermal level, aberrant pathway activation induces excessive proliferation of keratinocytes with impaired apoptosis, resulting in acanthosis and dyskeratosis, which manifest as characteristic scaly erythematous lesions [6]; activated keratinocytes release chemokines that recruit inflammatory cells to infiltrate the dermis, exacerbating local damage. At the vascular level, the MAPK/p38 pathway stimulates angiogenesis, causing vasodilation and congestion in the superficial dermis, further aggravating erythema and inflammation. Numerous basic experiments and clinical studies have demonstrated significantly elevated p-p38 expression in psoriatic lesions and model mice, with sustained pathway activation whose intensity correlates positively with PASI scores and inflammatory severity, confirming the MAPK/p38 pathway as a pivotal target in psoriasis pathogenesis.

3. Research Status of TCM Regulating MAPK/p38 Pathway for Psoriasis Based on Network Pharmacology

3.1 Application Advantages of Network Pharmacology and Molecular Docking Technology in TCM Research on Psoriasis

Traditional Chinese medicine (TCM) formulations exhibit characteristics of multi-component, multi-target, and multi-pathway synergistic effects, making it challenging to fully elucidate their anti-inflammatory mechanisms through studies focusing on a single target. Network pharmacology, grounded in systems biology and bioinformatics, constructs an interaction network linking "TCM components – disease targets – signaling pathways," aligning with the holistic perspective of TCM [10]. This approach enables rapid screening of core active constituents and key pathways involved in psoriasis treatment, addressing the limitations of traditional pharmacological studies where mechanistic explanations remain unclear.

Molecular docking serves as a critical validation method for network pharmacology prediction results, enabling the simulation of small molecule-target protein binding modes and affinities to assess the binding potential between components and targets at the molecular level. The combined application of these two techniques facilitates elucidation of the mechanism by which traditional Chinese medicines targeting the MAPK/p38 pathway exert therapeutic effects against psoriasis, thereby establishing a comprehensive research framework encompassing "component–target–pathway–therapeutic efficacy" and providing a scientific basis for the use of traditional Chinese medicine in psoriasis treatment.

3.2 Standard Research Paradigm and Core Procedures of Network Pharmacology for TCM Treating Psoriasis

Current network pharmacological studies on traditional Chinese medicine (TCM) for psoriasis treatment have established standardized research protocols: screening active components and targets of TCM, identifying disease-related targets for psoriasis, conducting target intersection analysis, constructing protein interaction networks, performing GO/KEGG enrichment analyses, and completing target validation. These studies predominantly utilize databases such as TCMSP, GeneCards, and OMIM to screen blood-soluble active components of TCM based on criteria of $OB \geq 30\%$ and $DL \geq 0.18$; subsequent aggregation of psoriasis-related targets yields potential therapeutic targets through intersection identification [11-12].

The intersection targets were imported into STRING to construct protein interaction networks, and core targets such as MAPK14 (p38), TNF, and IL-6 were screened using Cytoscape topology analysis [13-14]. Enrichment analyses were conducted with Metascape to identify targets involved in processes such as inflammatory responses and cell proliferation regulation, thereby pinpointing inflammatory pathways including MAPK/p38 and IL-17 [15]. Combined with molecular docking and animal experiment validation, this approach establishes a research paradigm of "big data prediction – molecular simulation – experimental validation," providing a standardized framework for studying the pathway mechanisms in traditional Chinese medicine [16].

3.3 Mechanism Study of Yinxianping Tablets Targeting MAPK/p38 Pathway Based on Network Pharmacology

Yinsheping Tablets are a commonly used traditional Chinese medicine for the clinical treatment of plaque psoriasis, demonstrating stable efficacy and good safety profile. Relevant online pharmacological studies have shown that Yinsheping Tablets contain multiple anti-inflammatory active components capable of targeting numerous disease targets in psoriasis, with MAPK14 (p38) as the primary target, indicating that the MAPK/p38 pathway is a key therapeutic target for psoriasis.

KEGG enrichment analysis revealed that the core targets of Yinxi Ping tablets are predominantly enriched in classic psoriasis inflammatory pathways such as MAPK, IL-17, and TNF, with the MAPK/p38 pathway showing the most significant enrichment [10]. GO functional analysis indicated that these targets are involved in biological processes including inflammatory responses and cell proliferation regulation, which closely aligns with the pathological characteristics of psoriasis. Molecular docking results

confirmed that the active components of Yinxi Ping exhibit strong affinity for p38 protein, enabling stable binding and providing a molecular basis for targeted modulation of this pathway [16].

Subsequent animal experiments further demonstrated that Yinsheping tablets effectively inhibit the phosphorylation and activation of the MAPK/p38 pathway, reduce the expression of pro-inflammatory factors IL-17 and IL-23, mitigate inflammatory infiltration and abnormal proliferation of keratinocytes in skin lesions, and significantly improve pathological damage in psoriasis. These findings corroborate the results of network pharmacology predictions and systematically elucidate its multi-component, multi-target anti-inflammatory mechanism [17].

3.4 Research Progress of Network Pharmacology on Other Anti-Psoriasis TCM Compound Prescriptions

Apart from Yinxianping Tablets, multiple classic clinical compound prescriptions have been confirmed by network pharmacology to regulate the MAPK/p38 pathway in a targeted manner. Network pharmacology research on Xijiao Dihuang Decoction for blood-heat type psoriasis shows that its core targets are significantly enriched in the MAPK signaling axis, and molecular docking verifies stable binding capacity of key components with the p38 target [18]. Heat-clearing and blood-cooling prescriptions centered on the herb pair "Paeonia Radix Alba – Salvia Miltiorrhiza – Moutan Cortex" also feature target networks highly enriched in the MAPK/p38 and TNF inflammatory pathways, and improve abnormal proliferation of HaCaT cells by downregulating pro-inflammatory cytokines [19-20]. In addition, blood-activating and stasis-resolving herb pair Danggui-Chuanxiong [21] and Guizhi Fuling Pill [22] have been proven to act on this pathway. International scholars have carried out network pharmacology and experimental research on formulas including Sinhyotaklisan and She-Chuang-Si-Wu -Tang, showing that such prescriptions can inhibit the Th17/IL-17 axis by regulating the STAT3/MAPK pathway, further verifying that targeted regulation of the MAPK/p38 pathway is a common core mechanism for multiple anti-psoriasis compound prescriptions to ameliorate psoriasis [23-24].

3.5 Experimental Research on TCM Compound Prescriptions Regulating MAPK/p38 Pathway

Traditional Chinese medicine formulations for the clinical treatment of psoriasis generally adhere to the therapeutic principles of clearing heat and cooling blood, promoting blood circulation to resolve stasis, detoxifying, and moistening dryness. These formulations can regulate inflammation through multiple pathways, modulate immune responses, and inhibit excessive proliferation of keratinocytes. Network pharmacology studies have demonstrated that targeted inhibition of aberrant activation of the MAPK/p38 signaling pathway constitutes a common core mechanism underlying the efficacy of such compound formulations in improving skin lesions.

Yinxieping Tablets are a clinically commonly used traditional Chinese medicine for the treatment of psoriasis, with well-established efficacy. Relevant experiments have demonstrated that this medication significantly reduces the expression of p-p38 in skin lesions of model mice, inhibits excessive activation of the MAPK/p38 pathway, lowers levels of IL-17 and IL-23, alleviates erythema, scaling, and histopathological damage in skin lesions, and exhibits dose-dependent therapeutic effects [25].

4. Problems and Deficiencies

Although existing research has made certain progress, this field still faces several limitations. First, pathway studies remain relatively limited in scope: most investigations focus solely on the MAPK/p38 pathway, neglecting its cross-regulatory interactions with key pathways such as JAK-STAT [26] PI3K/Akt [27], and NF-κB, thereby failing to fully elucidate the comprehensive, multi-targeted, and networked therapeutic advantages of traditional Chinese medicine. Second, animal models exhibit significant homogeneity: current studies predominantly employ imiquimod-induced acute psoriasis models, which fail to replicate the chronic, recurrent nature of clinical disease, resulting in limited translational value for experimental findings. Third, network pharmacology research lacks depth: most studies only complete target screening and intersection prediction, often exhibiting a bias toward prediction over validation, and lack integrated evidence from cellular and animal experiments [28]. Fourth, clinical mechanism research remains scarce: existing studies are primarily based on basic experiments, lacking large-scale, multicenter controlled clinical trials, and few reports validate p38 pathway activation levels using patient skin lesions or serum samples. Fifth, research innovation is insufficient: current paradigms are overly rigid, often confined to superficial efficacy assessments and pathway validation, with inadequate exploration of deeper mechanisms such as protein phosphorylation modifications, transcription factor regulation, and immune cell differentiation, leading to limited research depth.

5. Summary and Prospect

In summary, aberrant activation of the MAPK/p38 signaling pathway constitutes the core molecular mechanism underlying chronic inflammation, immune dysregulation, and abnormal proliferation of keratinocytes in psoriasis, playing a pivotal role throughout disease onset, progression, and recurrence. It represents a key therapeutic target for psoriasis management. Traditional Chinese Medicine (TCM) demonstrates extensive clinical experience, stable efficacy, and favorable safety profiles in treating psoriasis. Leveraging its synergistic regulatory effects across multiple components, targets, and pathways [29], TCM can inhibit phosphorylation and activation of the MAPK/p38 pathway, block inflammatory cascades, reduce pro-inflammatory cytokines such as IL-17 and IL-23, correct Th17 immune imbalance, suppress abnormal epidermal proliferation and dermal inflammatory infiltration, thereby comprehensively improving psoriatic lesions and pathological damage. This approach exhibits significant advantages in long-term prevention, treatment, and reduction of recurrence rates, offering broad clinical application potential. Existing fundamental studies have confirmed the efficacy of TCM targeting the MAPK/p38 pathway in psoriasis treatment,

providing robust experimental support for mechanistic research and clinical application.

To address the current limitations in this field of research, by integrating cutting-edge technologies such as network pharmacology, multi-omics, and molecular biology, future efforts can comprehensively enhance the research framework. This will advance the study of TCM mechanisms in treating psoriasis toward precision medicine, clinical applicability, and systematic development, thereby facilitating clinical translation and novel drug discovery.

First, advance research on multi-pathway collaborative regulation. By integrating transcriptomic and proteomic technologies, systematically elucidate the cross-regulatory mechanisms between pathways such as MAPK/p38, NF-κB, and PI3K/Akt, thereby overcoming the limitations of single-pathway studies [30].

Second, promote the deep integration of basic research with clinical applications. Conduct clinical controlled follow-up studies to measure changes in p-p38 expression in skin lesions before and after treatment, establish a correlation model between pathway markers and the PASI score, thereby filling the gap in clinical mechanism data [31].

Third, deepen comprehensive validation studies of network pharmacology across the entire research chain. Standardize the research workflow to precisely identify core active components and key targets in traditional Chinese medicine formulations [32], employ mass spectrometry technology to determine blood-soluble active constituents, elucidate the correspondence between "components – targets – pathways – therapeutic effects," and refine the mechanistic research framework [33].

Fourth, elucidate the underlying mechanisms to enhance research innovation. Go beyond superficial efficacy validation and conduct in-depth investigations into the molecular mechanisms governing upstream and downstream regulation of the MAPK/p38 pathway, protein phosphorylation modifications, and immune cell differentiation. Utilize cutting-edge technologies to comprehensively interpret the scientific rationale behind traditional Chinese medicine (TCM) in treating tinea [34], thereby providing theoretical foundations for precision TCM therapy and the development of targeted novel drugs for psoriasis.

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