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Progress in the Treatment of Alzheimer's Disease with Osthole

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Abstract: ***As a progressive neurodegenerative disorder, Alzheimer's disease is characterized by complex pathogenic mechanisms and a lack of effective therapeutic options, and has become a global public health challenge. Most current mainstream therapeutic agents target a single pathological process, with limited efficacy and frequent adverse effects. Consequently, identifying multi-target, low-toxicity active compounds from traditional Chinese medicine has emerged as an important research direction. Osthole, the major coumarin monomer component of Cnidium monnieri (L.) Cuss., exhibits multiple pharmacological activities, including neuroprotection, anti-inflammatory and antioxidant effects, as well as modulation of the cholinergic system. However, its specific mechanisms of action, efficacy, and safety in the treatment of Alzheimer's disease have not yet been systematically summarized and elucidated. This paper aims to systematically review recent preclinical and clinical research progress on osthole in the treatment of Alzheimer's disease, with a particular focus on its regulatory effects on key pathological processes such as β-amyloid protein deposition, Tau hyperphosphorylation, neuroinflammation, oxidative stress, and synaptic dysfunction. Existing evidence indicates that osthole can reduce Aβ production by inhibiting β-secretase activity, enhance autophagic clearance of abnormal proteins, attenuate neuroinflammation by suppressing excessive microglial activation, and improve mitochondrial function to counteract oxidative damage. In animal models, it has also been shown to ameliorate cognitive and memory impairments. These findings preliminarily reveal the potential of osthole to intervene in the pathological progression of Alzheimer's disease via multiple pathways and targets, providing important experimental evidence and theoretical support for the development of novel Alzheimer's disease therapies based on single compounds derived from traditional Chinese medicine, and laying a solid foundation for future in-depth studies on its pharmacodynamics, pharmacokinetics, and clinical applications.***

Keywords: Osthole, Alzheimer's disease, Neuroprotection**.**

1. Introduction

1.1 Global Challenges and Treatment Dilemmas of Alzheimer's Disease

The number of patients with Alzheimer's disease (AD) continues to rise with aging, cognitive decline, behavioral psychiatric symptoms and care dependence bring significant medical and socio-economic burden; and the existing treatment mostly stays in symptomatic improvement or intervention of a single pathway, in the face of Aβ deposition, tau abnormalities, neuroinflammation, oxidative stress and synaptic imbalance and other multi-link coupled pathological networks, the efficacy and sustainable benefits still have significant bottlenecks. The cascade interference of "attachment-masking" type in complex systems can be figuratively similar to the phenomenon that fine serpentine particles easily coat the surface of sulfides and significantly deteriorate the sorting performance during flotation [1], suggesting that AD treatment is easy to weaken the overall efficacy if it is only aimed at a certain target and is "covered" by concurrent pathological links, so more emphasis needs to be placed on multi-target and networked intervention strategies.

Screening active monomers with multi-pathway regulatory potential from traditional Chinese medicines is an important path to cope with therapeutic dilemmas. Osthole, as a coumarin natural product, has been demonstrated to up-regulate FGF21 expression by mediating ATF4 activation at the cellular level [2], providing a quantifiable molecular basis for its regulation on the energy metabolism-stress response axis; at the same time, drawing on the idea of "enhancing key element leaching by biological processes and establishing a kinetic model", that is, promoting Mg and Si leaching in rhamnite through microbial enhancement to improve process efficiency [3], AD drug research and development can focus on identifying limiting links (such as intracerebral delivery, blood-brain barrier permeability, effective exposure) and guide optimization with mechanistic models; parametric studies for cold plate heat dissipation in engineering to obtain better thermal management performance [4] also suggest that pharmacodynamic evaluation and dosing regimens also require systematic parameter optimization and iterative verification, in order to promote osthole from traditional experience to transformable new strategies.

1.2 Framework for Review of Osthole as a Therapeutic Candidate

Based on the above therapeutic dilemma, this paper intends to construct a systematic review framework of osthole in the treatment of Alzheimer's disease, focusing on its evidence integration and mechanism combing at the levels of Aβ deposition and abnormal modification regulation of tau protein, anti-neuroinflammation and anti-oxidative stress, improvement of cholinergic function and synaptic plasticity, as well as preclinical pharmacodynamics and preliminary translational research. At the same time, special attention will be paid to the potential of osthole in structural modification and structure-activity relationship, for example, coumarin derivatives containing isoxazoline/isoxazole fragments have shown good biological activity and safety evaluation in the field of semisynthetic pesticides, providing ideas for exploring the optimal design of osthole similar structures in central nervous system indications [5]. Methodologically, this paper attempts to learn from the research perspective of key

hosts and process strengthening in complex systems in geology and engineering, such as analyzing the occurrence state and migration path of nitrogen by minerals such as serpentine and chlorite in subduction zone ultrabasic rocks, which helps to identify the main carriers and transmission channels of trace components in deep circles [6]; and realizing process coupling and energy efficiency improvement by thermally activating serpentine and embedding it into adjustable energy systems during mineral carbonization [7], suggesting that the system design of "key hosts" and "process activation" also needs attention in the study of intracerebral delivery and metabolic pathways of osthole.

At present, there are still multiple knowledge gaps in the study of osthole in the treatment of Alzheimer's disease: first, the multi-target synergistic effect between different pathological links lacks systematic kinetic model support; second, there are limited data on blood-brain barrier permeability, effective exposure in the brain and long-term safety; and third, there is a lack of evidence of combination and sequential intervention with existing single-targeted drugs or traditional Chinese medicine compounds. In flotation engineering, degradable organic macromolecules are used to selectively adsorb ultrafine serpentine particles to alleviate their unfavorable coating and adhesion to the chalcopyrite flotation process, thereby significantly improving the sorting efficiency and resource utilization rate [8], providing a comparable perspective for optimizing the selective effect of osthole in the "background noise" of multiple pathological pathways and reducing non-specific interference. This review also has limitations in the breadth and depth of research: on the one hand, osthole related clinical studies are scarce and more dependent on cell and animal experiments are discussed; on the other hand, because the experimental design and dosing regimen are quite different among studies, it is difficult to carry out strict quantitative comparison and evidence-based integration, which will be used as an urgent direction to make up for subsequent research.

2. Current Research Status of Osthole Intervention in Key Pathological Links of Alzheimer's Disease

2.1 Regulation of Aβ Pathology and Abnormal Modification of Tau Protein

The intervention of osthole around the Aβ cascade mainly falls on both ends of APP abnormal processing and deposition clearance: on the one hand, it reduces the generation of toxic fragments such as Aβ1-42 by reducing the β/γ secretase-related cleavage tendency; on the other hand, it promotes the phagocytosis/degradation of Aβ oligomers with deposited plaques by improving protein homeostasis and autophagy-lysosome pathway efficiency in the brain, thereby reducing its continuous toxicity to synaptic structure and neuronal survival. The chain of evidence, linked to the pathological concept of AD, also suggests that the clinical phenotype of AD with Aβ deposition and abnormal phosphorylation of tau as the core axis accompanied by progressive cognitive decline [9], makes osthole more targeted for its value in "synchronous regulation of double pathology".

At the level of abnormal modification of tau, osthole is used to downregulate the hyperactivation of phosphorylation pathways such as GSK-3β and CDK5, and indirectly reduce tau hyperphosphorylation and neurofibrillary tangle burden by inhibiting upstream drive of inflammation-kinase axes such as JAK2/STAT3; its cross-disease mechanism can be confirmed by the evidence that osthole can significantly regulate JAK2/STAT3 signaling and produce tissue protective effects in vivo [10]. At the same time, osthole as a natural coumarin backbone has the advantages of derivatization and multi-site modification, and a structural design strategy of 1, 3, 4-oxadiazole, amine and other substituted derivatives around this backbone and obtaining considerable biological activity [11] provides a chemical space for subsequent structure-activity optimization oriented to Aβ generation inhibition/clearance promotion and tau kinase inhibition. It is worth noting that after polymer hydrolysis, the interface regulation idea that can change the system action from hydrophobic attraction to electrostatic repulsion and block the "coating" effect of fine particles [12] can also inspire the strategic design of osthole in Aβ aggregation/depolymerization interface intervention and delivery microenvironment modification.

2.2 Neuroprotective Effects Against Neuroinflammation and Oxidative Stress Injury

Neuroinflammation and oxidative stress often focus on microglial hyperactivation, amplify cytokine cascades and distract mitochondrial function decline. Osthole tends to pull microglia back from the pro-inflammatory phenotype to homeostasis in the context of a variety of inflammatory stimuli and reduces release pressures such as TNF-α and IL-1β brought about by TLR4-related amplification circuits; NEU1-mediated microglial activation can be understood as blunting upstream of the microglial "receptor – lysosome - amplification of inflammation" chain by network pharmacology in contrast to the inflammatory axis suggested by in vivo validation in LPS models [13]. At the end of the injury outcome, osthole can suppress lipid peroxidation driven iron death and alleviate inflammasome - associated pyroptosis in parallel, thereby reducing paracrine damage to surrounding neurons by secondary oxidative load and cytokine spillage [14].

At the level of mitochondria and oxidative stress, the protective effect of osthole is closer to the closed loop of "improving energy metabolism – reducing ROS-stabilizing membrane structure": blocking the self-excitable cycle in which inflammation and oxidative stress are mutually causal by maintaining mitochondrial membrane potential and reducing excessive ROS and lipid peroxidation. The fact that environmental factors can significantly rewrite weathering chemistry and remodel hydrochemical output of sulfide-containing/osthole systems suggests that perturbation of the redox environment and ionic microenvironment by external stress is sufficient to change the systemic response trajectory [15]; corresponding to the AD pathological microenvironment, the "buffering" value of osthole on the inflammation-oxidation interface lies in reducing the impact of stress fluctuations on vulnerable mitochondrial links. Similarly, the selective regulatory idea of sodium alginate to inhibit serpentine entrainment and change particle dispersion-

agglomeration behavior to optimize the separation process [16] also provides transportable engineering inspiration for delivery strategies that synergize osthole with carriers/ excipients to shape the local microenvironment and improve anti-inflammatory and antioxidant effects.

2.3 Functional Repair to Improve Cholinergic System and Synaptic Plasticity

The effect of osthole on the cholinergic system presents the characteristics of "synthesis-release-receptor-plasticity" multi-link coordination: on the one hand, it promotes acetylcholine synthesis and the recovery of available amount in the synaptic cleft by up-regulating cholinergic marker enzyme activity in the hippocampus and cortex, alleviates Aβ deposition, degeneration and apoptosis of cholinergic neurons caused by inflammatory factors, and maintains a high firing rhythm and information transmission efficiency in residual neurons [17].At the receptor level, osthole can regulate M1 cholinergic receptors and their downstream Ca2 + signaling and protein kinase cascades, improve long-term potentiation (LTP) impairment, and increase synaptic strength of learning and memory-related circuits; similar to estrogen-mediated cholinergic function enhancement patterns, cognitive impairment is reversed in ovariectomized rats by improving cholinesterase activity and balancing transmitters such as dopamine and serotonin, providing an important reference for osthole intervention through estrogen-like and cholinergic synergistic pathways in AD models [18].Cholinergic repair is often associated with synaptic structural remodeling, osthole can promote dendritic spine density recovery and postsynaptic density protein expression up-regulation, so that the synaptic loss process driven by Aβ, tau and oxidative stress is partially reversed, which is highly consistent with the overall idea that active components such as tanshinone IIA and puerarin achieve antioxidant, anti-apoptotic and synaptic protection by regulating Nrf2/p62/Keap1 and multiple signaling pathways [19].As a key epigenetic hub regulating cholinergic function and synaptic plasticity, non-coding RNAs are of increasing value as markers in the early diagnosis of AD and the evaluation of TCM intervention, providing an operable molecular entry point for subsequent dissection of osthole fine regulation of synaptic function through networks such as miRNAs and lncRNAs [20].

2.4 Preclinical Pharmacodynamics and Preliminary Clinical Exploration

In the existing animal model studies of Alzheimer ‘s disease, osthole mostly induced by Aβ in mice, rats and transgenic models, and generally showed a dose-dependent improvement trend in learning and memory and executive function through behavioral assessments such as open field test, Y-maze, novel object recognition and Morris water maze, which showed a shortened latency, prolonged residence time in the target quadrant, decreased number of error responses, and increased spontaneous alternation rate, and was highly consistent with its comprehensive regulation of cholinergic transmitters, synaptic proteins, and neuroinflammation. Evidence in the field of neuroprotection that ischemic/hypoxic preconditioning activates endogenous defense mechanisms and significantly improves the tolerance of hippocampal neurons to subsequent severe injury suggests that the behavioral benefits of osthole in AD models may also rely on preadaptation-like stress responses interacting with multiple targets [21]. At the same time, the experimental results from plant-derived active components such as Xanthoceras sorbifolia oil to delay memory decline and reduce neurological deficits in AD model flies also confirmed the feasibility of natural products to achieve cognitive behavioral improvement through multi-pathway intervention from the side [22].

Although osthole clinical research is still in its infancy, a large body of work represented by natural products derived from mulberry trees has shown that natural small molecules have a realistic basis for the development of neuroprotective drug candidates in a variety of neurological diseases [23]. In line with this, andrographolide, modified by nanodelivery systems, can significantly improve its solubility and exposure in the brain in vivo and strengthen anti-inflammatory and anti-apoptotic effects, providing an important idea for osthole to optimize brain-targeted delivery and improve the transformation of animal behavioral benefits to clinical cognitive improvement through liposomes, nanoparticles, or gel systems in the future [24]. At present, osthole still lacks systematic evaluation in dose safety window, long-term administration toxicology, pharmacokinetics and combination regimens with existing anti-dementia drugs. How to realize the transformation from a single animal model to a multicenter and staged stratified population under the premise of ensuring safety is the key link to its urgent clinical application.

3. Conclusions and Prospects

3.1 Consensus and Core Findings on the Potential of Osthole Multi-target Therapy

The multi-target potential of osthole in the treatment of Alzheimer's disease has formed several common understandings: First, it targets the dual regulation of Aβ deposition and abnormal phosphorylation of tau and is considered to be the basis for its disease-modifying effect, similar to the comprehensive effect of β-hydroxybutyrate in achieving neuroprotection by inhibiting Aβ deposition, reducing tau hyperphosphorylation and improving energy metabolism, providing a reference for osthole to integrate multi-pathway intervention of metabolic homeostasis and proteostasis [25]. The second is that osthole generally shows synergistic effects of antioxidant, anti-inflammatory and mitochondrial function protection, which coincides with the idea that ischemia/hypoxia preconditioning activates the endogenous protective network through transient non-lethal ischemia and improves tissue tolerance to subsequent injury, suggesting that osthole is expected to induce a neuroprotective state similar to "pharmacological preconditioning" [26].

In the emerging pathomechanism dimension, osthole is speculated to indirectly inhibit iron death-related pathways by regulating iron metabolism and lipid peroxidation, while bibliometric analysis of iron death and Alzheimer's disease shows that research hotspots in this field focus on iron homeostasis disorders, lipid peroxidation, and their association with cognitive decline, highlighting the potential

value of osthole cutting into this cutting-edge pathway [27]. At the same time, the effects of osthole on the autophagy-lysosome system have gradually received attention, and studies of the transcription factor EB mediating the autophagolysosome pathway to improve Alzheimer's disease have shown that enhancing autophagy-lysosome scavenger function is a key strategy to alleviate Aβ and tau loads, providing an important mechanistic framework for osthole by up-regulating autophagy, promoting pathological protein clearance, and remodeling neural network homeostasis [28]. The resulting multi-pathway and multi-level collaborative intervention is currently the core breakthrough point on osthole in the treatment of Alzheimer's disease with the most consensus.

3.2 Future Research Directions and Challenges Faced by Translational Medicine

In subsequent studies of osthole Alzheimer 's disease prevention and treatment, mechanistic maps need to be remodeled at the molecular to systemic level, especially around the interaction of miRNA networks, neuroinflammatory signaling, and cytoskeletal remodeling for systematic dissection. Osthole inhibits tumor cell proliferation, migration, and invasion by regulating the miR-433-3p/Tiam1 axis in colon cancer models, suggesting that it has a fine regulatory ability against miRNA regulatory and cell migration-related signaling pathways, providing ideas for its intervention of neuronal synaptic remodeling and microglial phenotypic transformation in Alzheimer's disease [29]. Combined with transcriptome, single cell sequencing and spatial omics, osthole multi-target intervention network should be constructed in the future, and key nodes should be verified by gene knockout and specific inhibitors to elucidate their causal relationship in Aβ clearance, autophagy activation, iron death inhibition and other links.

In the transformation path, on the one hand, it is necessary to carry out in-depth pharmacokinetic and pharmaceutical optimization, systematically evaluate the brain distribution, blood-brain barrier permeability, metabolic enzyme pathways and potential drug interactions under different administration routes of oral and injection, explore the combination mode with modern delivery systems such as nanodelivery and sustained and controlled release carriers, and form a safe window suitable for long-term medication in elderly patients. On the other hand, osthole can be combined with stem cell therapy and nerve regeneration strategies to assess its supporting effects on neural stem cell directed differentiation, synaptic remodeling, and brain microenvironment remodeling by referring to its microenvironment regulation and tissue repair advantages embodied in improving the osteogenic differentiation function of MSCs in a high glucose environment [30]. On this basis, gradually promote the safety exploration from small samples to multi-center, randomized controlled high-quality clinical trial design, strengthen the accumulation of evidence-based evidence, and construct a precise administration strategy of TCM syndrome classification and biomarker co-stratification according to individual differences.

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