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Research Progress on the Antioxidative Stress Effects of Astragalus Active Components and DJ-1 in Multiple Sclerosis

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Abstract: Multiple sclerosis (MS) is an autoimmune disease primarily affecting the white matter of the central nervous system (CNS), characterized by pathological changes of chronic multiple inflammatory demyelination. Its clinical manifestations alternate between relapses and remissions, showing a stepwise progressive development, which can lead to oligodendrocyte death, axonal disintegration, and ultimately neuronal loss, resulting in irreversible damage to neurological functions. In the early stage of onset, inflammatory responses cause excessive accumulation of reactive oxygen species (ROS), disrupting the dynamic balance of intracellular oxidative-antioxidative systems and triggering reactions in the central nervous system. The experimental autoimmune encephalomyelitis (EAE) mouse model is a classic animal model for multiple sclerosis. In traditional Chinese medicine (TCM), MS falls into the categories of "flaccidity syndrome" and "arthralgia syndrome." During the acute phase, the therapeutic principle focuses on eliminating pathogenic factors while supporting healthy qi; in the remission phase, the treatment emphasizes tonifying healthy qi and replenishing qi. *Astragalus membranaceus*, a commonly used and popular qi-tonifying herb in TCM clinical practice, possesses the effects of tonifying qi to consolidate healthy qi, replenishing qi and lifting yang, strengthening the defensive exterior to consolidate the superficial resistance, and promoting tissue regeneration to heal ulcers. The DJ-1 protein has antioxidative stress ability and potential crosstalk and synergistic relationships with *Astragalus membranaceus* in antioxidative stress and immune regulation. Therefore, both may become new targets for the prevention and treatment of multiple sclerosis in clinical practice. By consulting relevant literatures published from 2013 to 2025 in databases such as China National Knowledge Infrastructure (CNKI), Wanfang, VIP, and PubMed, this paper reviews the research progress of *Astragalus membranaceus* and its active components in the treatment of multiple sclerosis.

Keywords: *Astragalus membranaceus*; Multiple sclerosis (MS); DJ-1; Reactive oxygen species (ROS); Experimental autoimmune encephalomyelitis (EAE); Research progress

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1. Introduction

Multiple sclerosis (MS) is a chronic inflammatory demyelinating disease of the central nervous system. Lesions can involve brain tissue, spinal cord, optic nerve, etc., with multiple inflammatory demyelination, axonal degeneration, and gliosis as the main pathological characteristics [1]. It has a high incidence rate, recurrence rate, and disability rate, which can lead to various sequelae such as blindness, sensory disturbance, cognitive impairment, and even paralysis in patients, seriously affecting the quality of life and bringing a huge impact on patients, their families, and the entire society. Western medicine treatment of MS often uses hormones, immunosuppressants, etc., which impose a heavy economic burden and are associated with common adverse reactions. TCM treatment achieves therapeutic effects through overall regulation and coordination of yin and yang. Cluster analysis shows that *Astragalus membranaceus* is the most frequently used TCM in the treatment of multiple sclerosis [2].

By searching databases such as CNKI, Wanfang, VIP, and PubMed for relevant literatures from 2013 to 2025 with keywords including "*Astragalus membranaceus*", "active components", "multiple sclerosis", "EAE", "autoimmune encephalomyelitis", "DJ-1", and "ROS", more than 100 literatures were obtained. A total of 44 important relevant literatures were included in this paper to comprehensively discuss the related mechanisms and research progress of the active components of *Astragalus membranaceus* in the treatment of MS.

2. Pathogenesis of multiple sclerosis

The main pathological changes of MS include myelin destruction, blood-brain barrier (BBB) dysfunction, axonal injury, and neuronal loss. It is characterized by the formation of multiple demyelinating plaques in the white matter of the central nervous system, leading to neuroconductive dysfunction and neurodegeneration. Clinical classifications include relapsing-remitting MS (RRMS), primary progressive MS (PPMS), and secondary progressive MS (SPMS), among which RRMS is the most common [3]. There are multi-dimensional interactions between the nervous system and the immune system [4]. In diseases such as MS or when the BBB is damaged, a large number of peripheral adaptive immune cells such as T cells and B cells enter the central nervous system (CNS), leading to abnormal central immune responses and triggering inflammatory cascades. The specific cause of immune disorders remains unclear [5].

The pathogenesis of MS is also related to the imbalance of T cells and macrophages. The progression of MS is associated with the secretion of pro-inflammatory cytokines such as IL-12, TNF-α, IL-1β, and IFN-γ; the improvement of symptoms is associated with the secretion of anti-inflammatory cytokines such as IL-4 and IL-10. In addition, activated CD4+T cells can mediate and amplify inflammatory responses by producing pro-inflammatory factors such as IL-12 and TNF-α, and enter the central nervous system through the blood-brain barrier, thereby causing nerve damage [6]. B cells and other B cell subsets can affect the onset and outcome of MS through various different pathways. Its pathogenesis covers multiple aspects, including antibody production, cytokine release, formation of ectopic lymphoid tissue, and migration of B cells to the central nervous system [7].

Among them, a typical pathological feature of MS is the appearance of oligoclonal band antibodies [8]. Studies have shown that these oligoclonal band antibodies are not directed against CNS antibodies, and the target antigens generally include Myelin Oligodendrocyte Glycoprotein (MOG), Myelin Basic Protein (MBP), proteolipid protein, myelin-derived lipids, or other specific CNS antigens [9]. However, the increase

in antibody titers, such as MBP and MOG, also indicates the occurrence of specific immunity. In addition to the above target antigens, many other candidate antigens remain to be studied [10].

Activated B cells in the blood of MS patients can produce excessive pro-inflammatory cytokines. In 2017, Okada Y et al. found that B cells from MS patients produced insufficient IL-10 compared with healthy controls under CD40L stimulation.

The stable interaction between B cells and T cells is also an important factor driving MS. In addition, the degree of increased permeability of the BBB is positively correlated with the severity of the disease. In the MS animal model, experimental autoimmune encephalomyelitis (EAE), the expression of MMP-9, NADPH, ICAM-1, VCAM-1, etc., in mice is upregulated, leading to increased BBB permeability. Adhesion molecules and chemokines secreted by immune cells promote Th1/M1 macrophages to secrete pro-inflammatory factors into the central nervous system, thereby causing nerve damage.

3. Current status of multiple sclerosis treatment

At present, the global incidence of MS is increasing year by year, with significant differences in global distribution. The incidence and prevalence vary in different countries and regions. According to statistics, the global median prevalence of MS is 33 per 100,000 people, with the highest prevalence in North America and Europe, and the lowest in Asia and sub-Saharan African countries. The incidence rate in women is 2–3 times that in men. MS is characterized by multiple episodes, recurrence, and a high disability rate. With the continuous progression of the disease, it often leads to permanent disability in the end. This not only affects the quality of life of patients, brings a heavy care burden to their families, but also imposes a huge economic burden on the social medical system, including the consumption of medical resources and the increase in long-term care costs. Due to the complex etiology and pathogenesis of MS, there is still no effective cure for the disease so far. According to the 2023 guidelines, glucocorticoid therapy remains the first-line treatment for acute MS, while disease-modifying therapy (DMT) in the remission phase mainly includes immunosuppressants, immunomodulators, and antioxidants. However, due to individual differences among patients, for patients with poor response to hormone therapy, prolonged treatment does not benefit neurological recovery in the long run, but may trigger a series of serious adverse reactions. Moreover, MS is a lifelong disease. The remission phase mainly adopts DMT for immunomodulation and immunosuppressive therapy, but long-term use of immunosuppressants increases the risk of infection and cancer. Long-term use of hormone therapy is likely to lead to drug tolerance in patients, reduce their immunity, and cause relatively many side effects. In addition, due to the high price of hormone drugs, it increases the economic burden on patients, which is not conducive to the long-term treatment of patients. In this case, the combined use of traditional Chinese and Western medicine has a better curative effect, fewer side effects, and saves patients' medical costs. Multiple sclerosis belongs to the categories of "flaccidity syndrome" and "arthralgia syndrome" mentioned in *Huangdi Neijing·Suwen* (Yellow Emperor's Internal Classic·Plain Questions). In the remission phase, it advocates tonifying healthy qi and replenishing qi. *Astragalus membranaceus* has excellent effects of tonifying healthy qi and replenishing qi, enhancing patients' immunity and anti-inflammatory effects. It is used in the combined treatment of multiple sclerosis with traditional Chinese and Western medicine in clinical practice. Relevant literature shows that through the analysis of the medication rules of nationally famous TCM doctors in the treatment of MS, it is found that *Astragalus membranaceus* is included in the high-frequency drugs. When combined with other medicinal

materials into compound prescriptions, *Astragalus membranaceus* can exert a therapeutic effect on MS through mechanisms such as regulating immunity and anti-inflammation. These compound prescriptions include *Astragalus membranaceus*-*Cuscuta chinensis*-*Epimedium brevicornum*, *Astragalus membranaceus*-*Epimedium brevicornum*-*Codonopsis pilosula*, etc. The clustering of core drugs reflects the compatibility ideas of invigorating the spleen and eliminating dampness, tonifying the liver and kidney, resolving phlegm and promoting blood circulation, regulating qi and removing blood stasis, which embodies the characteristics of TCM in treating MS following "tonifying the kidney, resolving turbidity, detoxifying, and dredging collaterals."

4. Intervention effects of active components of *Astragalus membranaceus* on multiple sclerosis

Astragalus membranaceus is one of the commonly used qi-tonifying drugs in TCM clinical practice. It is the dried root of *Astragalus mongholicus* or *Astragalus membranaceus* of the Fabaceae family. It is warm in nature, sweet in taste, and belongs to the spleen and lung meridians. It has the effects of tonifying the middle and replenishing qi, promoting qi circulation to relieve bi syndrome, consolidating the exterior to stop sweating, inducing diuresis to reduce edema, promoting fluid production to nourish blood, and promoting tissue regeneration to heal ulcers. Fan Yongping, Shang Xiaoling, and others have used compound prescriptions containing *Astragalus membranaceus* in clinical applications, which have played a certain role in improving the symptoms of MS patients. The chemical components of *Astragalus membranaceus* mainly include three categories: polysaccharides, saponins, and flavonoids, which can exert multiple effects such as inhibiting inflammation, resisting oxidation, preventing cell apoptosis, regulating immune function, and maintaining cardiovascular health.

Astragalus membranaceus is known to contain 25 amino acids, including methionine, glutamic acid, leucine, γ-aminobutyric acid, etc., more than 20 trace elements such as selenium, zinc, iron, copper, etc., as well as other chemical components such as palmitic acid, palmitic acid glyceride, linoleic acid, linolenic acid, betaine, and bitter base. However, these are not the main components of the pharmacological effects of *Astragalus membranaceus*.

The flavonoids and polysaccharides in *Astragalus membranaceus* have the effects of scavenging free radicals and inhibiting oxidative stress. These components may indirectly regulate their antioxidative functions by enhancing the stability or activity of DJ-1, but the specific molecular mechanisms still need further research. Next, the intervention effects of the three main types of pharmacologically active compounds of *Astragalus membranaceus* in multiple sclerosis will be elaborated.

4.1. Astragalus polysaccharides

Astragalus polysaccharides can participate in the treatment of multiple sclerosis through antioxidative stress and anti-inflammatory responses. In the early stage of multiple sclerosis, inflammatory responses cause excessive accumulation of reactive oxygen species (ROS), which further disrupts the dynamic balance of the intracellular oxidative-antioxidative system and ultimately triggers chronic neuronal demyelination in the central nervous system through oxidative stress-mediated neurotoxic mechanisms. Experiments by Zhang Jingfang et al. have proved that Astragalus polysaccharides can increase the activity of superoxide dismutase and total antioxidant capacity. Zhong Ling et al. found that Astragalus polysaccharides can

reduce the malondialdehyde content and increase the peroxidase activity in mice, indicating that Astragalus polysaccharides have good antioxidative effects.

4.2. Astragalus saponins

Studies on the molecular mechanism of Astragaloside IV in regulating CD4+T cell differentiation have shown that this active component can significantly downregulate the secretion levels of pro-inflammatory cytokines IFN-γ, TNF-α, and IL-6, as well as the mRNA expression of RORγt transcription factor, while significantly upregulating the gene transcription levels of T-bet and Foxp3, thereby remodeling the differentiation pattern of CD4+T cell subsets. It inhibits the infiltration of autoreactive T cells into the central nervous system and effectively reduces neuroinflammatory damage. In vitro experiments have shown that Astragaloside IV can regulate the differentiation of Th17 cells and Treg cells. Treg cells are important regulatory T cells in the body that can inhibit T cells. In multiple sclerosis, they reduce the attack of T cells on normal autologous cells, indicating that Astragalus saponins can play an immunomodulatory role by regulating the differentiation of T cells, thereby fundamentally addressing the causes of multiple sclerosis. Phosphorylated myosin phosphatase target subunit 1 (p-MYPT1) is a substrate of Rho-associated protein kinase (ROCK), and its expression level is positively correlated with ROCK activity. After administration of Astragaloside IV, the phosphorylation level of MYPT1 in the spinal cord of EAE mice is significantly decreased, ROCK activation is inhibited, and neurons are protected, indicating that Astragalus saponins have a certain neuroprotective effect. Studies have shown that Astragaloside IV reduces neuroinflammation in EAE mice by inhibiting blood-brain barrier leakage. The mechanism involves reducing the levels of ROS and iNOS in the central nervous system, enhancing the activities of SOD and GSH-Px; in addition, Astragaloside IV can significantly inhibit the activation of microglia in the central nervous system in the EAE model, downregulate the transcription levels of pro-inflammatory cytokines IFN-γ, TNF-α, and IL-6, coordinately regulate the differentiation of CD4+T cell subsets, and inhibit their pathological infiltration into the central nervous system. Astragaloside IV can alleviate the clinical symptoms of EAE mice by inhibiting the proportion of CD4+T cell subsets expressing interferon-γ and interleukin-17, upregulating the percentage of CD4+T cell subsets expressing interleukin-10 and transforming growth factor-β, downregulating the expression of interferon-γ, interleukin-17, and interleukin-6 in the spinal cord and spleen, and upregulating the expression of the anti-inflammatory factor interleukin-4 in the spleen. Its mechanism is related to regulating the immune cell subsets in the spleen, thereby inhibiting the infiltration of inflammatory cells into the central nervous system and reducing myelin loss.

4.3. Astragalus flavonoids

Astragalus flavonoids have anti-inflammatory, antioxidative, immunomodulatory, and skeletal system protective effects. A large number of experiments have proved that Astragalus flavonoids have good anti-inflammatory effects. For example, Gu Minhua et al. found that formononetin can significantly inhibit the abnormal expression of inducible nitric oxide synthase, interleukin-6, and tumor necrosis factor-α, effectively reduce the inflammatory damage of the blood-brain barrier, and inhibit the transcription of MMPs. Xu Feng et al. found that calycosin-7-glucoside can significantly inhibit the proliferation of T lymphocytes and reduce macrophage toxicity, confirming that calycosin-7-glucoside has anti-inflammatory and immunosuppressive effects and can be used to prepare anti-inflammatory drugs and immunosuppressants. It is proven that Astragalus flavonoids have good anti-inflammatory effects. Based on antioxidative stress, Hu Zhiping et

al. found that formononetin can activate the Nrf2/HO-1 pathway, thereby reducing the water content and malondialdehyde level of brain tissue and increasing the superoxide dismutase level in a mouse model of traumatic brain injury, indicating that Astragalus flavonoids have a certain antioxidative effect. Yu Yifan et al. proved that total flavonoids of *Astragalus membranaceus* can promote the secretion of cytokines IL-6, IL-1β, IFN-γ, TNF-α, and cellular mediators NO and PGE2 in the supernatant of macrophage RAW264.7, and upregulate the expression of iNOS and COX-2 proteins, indicating that it has an immunomodulatory effect.

In summary, the three main types of pharmacologically active compounds of *Astragalus membranaceus* all have immunomodulatory and antioxidative stress effects, thereby achieving the prevention and treatment of multiple sclerosis. This includes symptomatic treatment of multiple sclerosis by inhibiting overactive T cells and reducing neuronal demyelination caused by oxidative stress. At the same time, *Astragalus membranaceus* also has anti-inflammatory effects, which can reduce inflammatory responses, thereby reducing damage to neurons, and has a certain protective and preventive effect on osteoporosis, which can reduce bone damage during hormone therapy, reduce the side effects of patients' medication, and improve the quality of life of patients.

5. Antioxidative stress mechanism of DJ-1

DJ-1 is a multifunctional protein widely distributed in organisms. It participates in many cellular life activities such as cell growth, antioxidative stress responses, and gene transcription. However, it has attracted increasing attention in enhancing cellular antioxidative stress, so it is also known as an antioxidative stress protein. Studies have found that DJ-1 may activate the Nrf2-ARE signaling pathway to resist myocardial cell oxidative stress injury induced by hypoxia/reoxygenation (H/R) in H9c2 cardiomyocytes.

DJ-1 can scavenge intracellular reactive oxygen species (ROS) through autoxidation; regulate the morphology and function of intracellular mitochondria to reduce intracellular ROS production; DJ-1 can also activate various molecular signaling pathways in cells, enhance the expression of cellular antioxidant enzymes, promote ROS scavenging, thereby restoring the cellular redox balance, and ultimately enhancing cellular antioxidation, promoting proliferation, and inhibiting cell apoptosis.

Studies have shown that during osteonecrosis of the femoral head (ONFH), through oxidative stress pre-experiments on bone marrow mesenchymal stem cells, it is found that DJ-1 protein exhibits significant antioxidative stress ability: DJ-1 exerts its effects through regulating the Keap1-Nrf2-ARE signaling pathway, phosphatidylinositol-3-kinase/protein kinase B (PI3K/Akt) signaling pathway, ERK1/2 signaling pathway, and mitochondrial antioxidative stress.

5.1. Antioxidative stress mechanism by regulating the Keap1-Nrf2-ARE signaling pathway

Under the induction of cellular reactive oxygen species, DJ-1, as a new antioxidative stress gene, can bind to the Keap1/Nrf2 complex and promote its dissociation. DJ-1 can bind to the inhibitory protein Keap1 to inhibit its interaction with Nrf2, thereby stabilizing the expression of Nrf2. Activated Nrf2 enters the nucleus and binds to Maf proteins to form dimers that recognize the corresponding ARE sequences, thereby initiating the transcription of antioxidant genes and promoting the expression of antioxidant proteins (**Figure 1**). At the same time, when DJ-1 is deficient, the stability of Nrf2 decreases, preventing the activation of the Nrf2/ARE signaling pathway and the expression of downstream antioxidant genes, leading to a significant increase in ROS levels and accumulation of ROS.

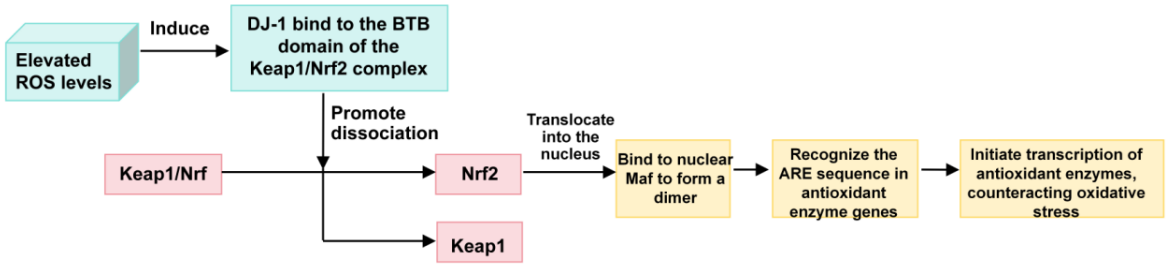

Figure 1. Mechanism of DJ-1 exerting antioxidative stress by regulating the Keap1-Nrf2-ARE signaling pathway. ROS: Reactive Oxygen Species; DJ-1: Parkinsonism Associated Degrees 7; Keap1: Kelch-Like ECH-Associated Protein 1; BTB: Bric-a-brac/Tramtrack/Broad complex; Maf: Small Maf Proteins; ARE: Antioxidant Response Element.

5.2. Antioxidative stress mechanism by regulating the PI3K/Akt signaling pathway

Overexpression of DJ-1 can significantly upregulate Thr308 in the PI3K/Akt signaling pathway to promote Akt phosphorylation. Phosphorylation modification at the Thr308 site is a key regulatory node for the activation of the Akt signaling pathway. The Thr308 site can mediate the phosphorylation activation of Akt through specific binding to phosphoinositide-dependent protein kinase 1 (PDK1). The Thr308 site of the Akt protein can bind to phosphoinositide-dependent protein kinase 1 to activate Akt, thereby promoting the phosphorylation of downstream target proteins to inhibit cell apoptosis and play a role in protecting nerve cells (**Figure 2**).

In addition, DJ-1 activates the PI3K/Akt pathway, thereby phosphorylating Nrf2 and promoting its nuclear translocation, further enhancing the antioxidant defense. Studies have shown that overexpression of DJ-1 can reduce the production of ROS through this pathway.

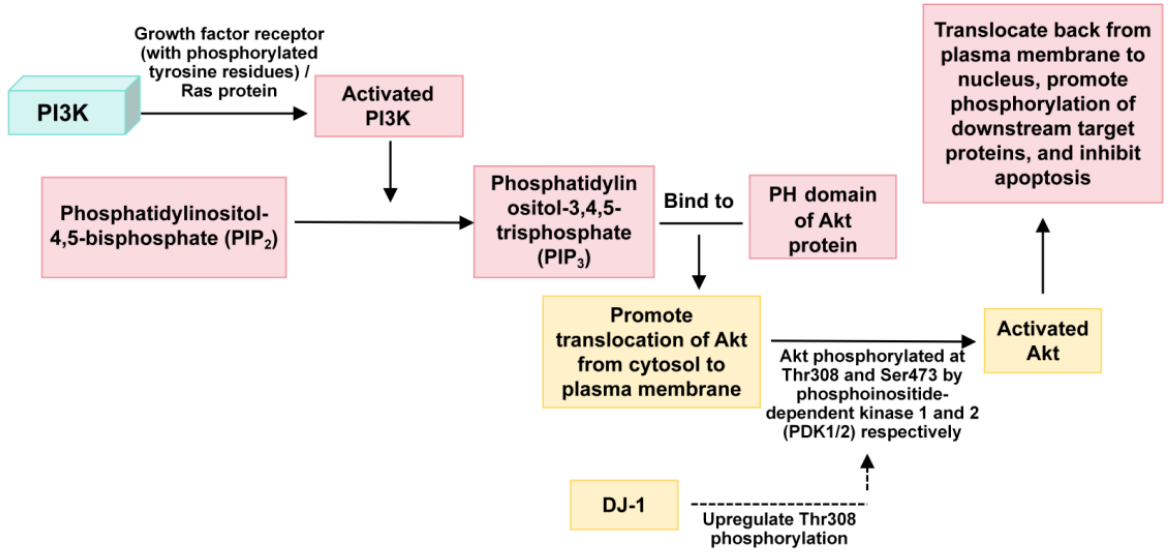

Figure 2. Antioxidative stress mechanism by regulating the PI3K/Akt signaling pathway. PI3K: Phosphatidylinositol-3-Kinase; Ras: Rat Sarcoma GTPase; Akt: Protein Kinase B; DJ-1: Parkinsonism Associated Degrees 7.

5.3. Antioxidative stress mechanism by regulating the extracellular signal-regulated kinase 1/2 (ERK1/2) signaling pathway

This protein can effectively mediate the nuclear translocation of ERK1/2 through direct interaction with ERK1/2. ERK1/2 entering the nucleus further activates members of the ETS oncogene transcription factor family and ultimately enhances ERK1/2 activity through regulating the gene transcription level of manganese superoxide dismutase (MnSOD) or binding to the C-Raf gene to promote the phosphorylation of C-Raf at Ser-338, thereby activating the MEK1 and ERK1/2 molecular signaling pathways. It can also bind to P53 under oxidative stress regulation to inhibit its transcriptional activity and reduce the inhibitory effect of P53 on ERK1/2 activity in the nucleus. Through the above direct or indirect ways, it enhances ERK1/2 activity, promotes the expression of downstream targets, and thus plays an antioxidative role (**Figure 3**).

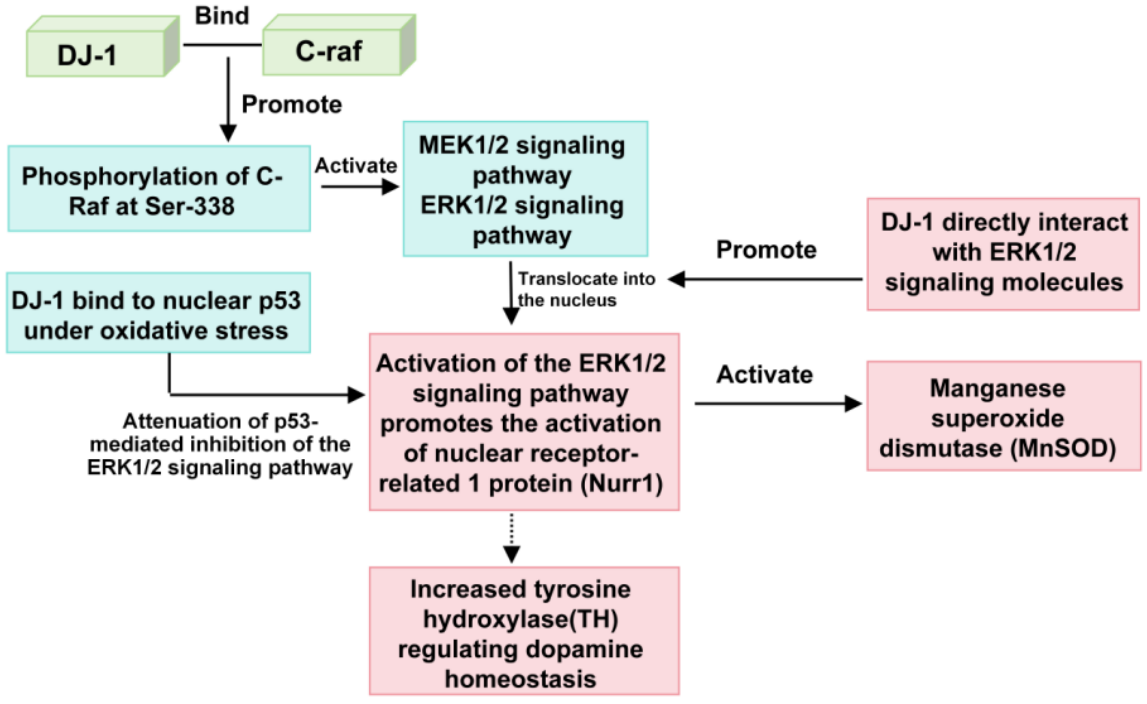

Figure 3. Mechanism of DJ-1 regulating the extracellular signal-regulated kinase 1/2 (ERK1/2) signaling pathway for antioxidative stress. DJ-1: Parkinsonism Associated Degrees 7; C-raf: v-Raf-1 Murine Leukemia Virus Oncogene Homolog 1 Gene; MEK1/2: Mitogen-Activated Protein Kinase Kinase 1/2; ERK1/2: Extracellular Signal-Regulated Kinase 1/2; P53: Tumor Protein 53.

5.4. Mechanism of DJ-1 regulating mitochondrial antioxidative stress

Studies have shown that when cells are stimulated by external factors, this protein can directly bind to Complex I by targeting the inner mitochondrial membrane and participate in regulating its enzyme activity. The protective effect of DJ-1 on Complex I can significantly improve mitochondrial function. Under oxidative stress conditions, the decrease in mitochondrial membrane potential and changes in membrane permeability may serve as key inducers for the mitochondrial translocation and localization of DJ-1 in the inner membrane system. DJ-1 also participates in mitochondrial regulation, which can phosphorylate mitochondrial dynamin-related protein 1 and avoid mitochondrial autophagic degradation.

6. Conclusion and prospect

Multiple sclerosis is a common, non-traumatic, disabling disease that can present various neurological symptoms, seriously affecting the quality of life of patients. Due to the incomplete clarification of its etiology and pathogenesis, the current clinical treatment methods are still relatively limited. Over the past two decades, with the progress of immunotherapy technology, Western medicine mainly inhibits inflammatory responses in clinical practice, while TCM commonly uses methods such as tonifying the middle and replenishing qi and inhibiting oxidative stress to reduce disease recurrence. As a traditional Chinese medicine, *Astragalus membranaceus* has gradually attracted attention in the prevention and treatment of MS. The authors believe that *Astragalus membranaceus* has broad application prospects in the treatment of multiple sclerosis. At the same time, the DJ-1 protein also has antioxidative stress effects. Whether there are potential cross and synergistic relationships between DJ-1 and *Astragalus membranaceus* in antioxidative stress and immune regulation, such as whether *Astragalus membranaceus* and DJ-1 can be combined to jointly intervene in the recurrence of multiple sclerosis? This can be used as a research direction for the authors to further study its mechanism of action, explore combined applications, develop innovative drugs, and carry out large-scale clinical trials, which is expected to provide safer and more effective treatment and recurrence prevention methods for patients with multiple sclerosis.

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Disclosure statement

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