MEDS Chinese Medicine (2025)
Clausius Scientific Press, Canada

DOI: 10.23977/medcm.2025.070103
ISSN 2616-1753 Vol. 7 Num. 1

# *α-Synuclein's Multifunctionality in Parkinson's Disease: From Pathogenic Mechanisms to Therapeutic Targets*

**Yu Ruishan[a,*], He Jiaqi[b], Du Nan[c], Peng Tao[d], Wang Gaofen[e]**

*Shaanxi University of Chinese Medicine, Xianyang City, Shaanxi Province, China*
*[a]yrs515620@163.com, [b]1015453992@qq.com, [c]572490028@qq.com, [d]1596915306@qq.com, [e]2962318415@qq.com*
**Corresponding author: yrs515620@163.com*

***Keywords:*** α-synuclein, Parkinson's disease, pathogenic mechanisms, therapeutic targets

***Abstract:*** Parkinson's disease (PD), a prevalent neurodegenerative disorder, affects over 6 million individuals worldwide. Its incidence rates are positively correlated with age, showing an increasing trend as age advances. α-Synuclein plays a central role in the initiation and progression of Parkinson's disease. The mechanisms underlying its transition from a physiological to a pathological state are highly complex and diverse. This article conducts an in-depth investigation of α-synuclein, providing a detailed analysis of its molecular structural characteristics and physiological functions. It focuses on elucidating its pathological mechanisms in the progression of Parkinson's disease, including aggregation and fibrillation, effects on cellular organelles, induction of neuroinflammation, and cell death. Additionally, it offers a comprehensive review of current therapeutic strategies targeting α-synuclein, aiming to provide new insights and directions for the treatment of Parkinson's disease and to promote further research and development in related fields.

## 1. Introduction

Parkinson's disease is a common neurodegenerative disorder that affects middle-aged and elderly individuals. The characteristic pathological changes include the progressive degeneration of dopaminergic neurons in the substantia nigra and the formation of Lewy bodies. With the continuous reduction of dopamine in the striatum and the ongoing imbalance between dopamine and acetylcholine neurotransmitters, clinical symptoms in patients progressively worsen. The main manifestations are motor symptoms such as tremors, rigidity, bradykinesia, and postural instability, as well as non-motor symptoms such as sleep disturbances, olfactory disorders, autonomic dysfunction, and cognitive and psychiatric disorders. Epidemiological survey data indicate that the prevalence of Parkinson's disease among individuals aged 65 and above globally is between 1% and 2%. Currently, the prevalence of Parkinson's disease among individuals aged 65 and above in China is 1.7%, which is at the global average level. However, with the continuous exacerbation of an aging society, by 2030, the number of individuals with Parkinson's disease in China may rise to 5 million, which would account for nearly half of the global number of individuals with Parkinson's disease[1-3].α-Synuclein is a soluble protein expressed in the presynaptic and perinuclear regions of

the central nervous system. It is closely associated with the pathogenesis of Parkinson's disease and related functional impairments. Under certain conditions, it misfolds and aggregates into toxic species, leading to neuronal damage and degeneration[4]. Despite years of research, the pathogenesis of Parkinson's disease has not been fully elucidated. However, the abnormal conformational transitions and aggregation of α-synuclein (α-Syn) are widely recognized as one of the core factors in the pathogenesis of Parkinson's disease (PD). In-depth investigation of its structural and functional characteristics is expected to provide new scientific foundations and therapeutic targets for the early diagnosis, precise treatment, and prevention strategies of the disease.

## 2. Structure and Function of α-Synuclein

### 2.1. Protein Structure

α-Synuclein is a 140-amino acid (aa) protein encoded by the SNCA gene, with a molecular weight of approximately 14 kDa. It is a natively unfolded protein that features three significant structural domains: namely, the amphipathic N-terminal domain (1–60 aa), the hydrophobic central domain (non-amyloid component, NAC, 61–95 aa), and the hydrophilic C-terminal domain (96–140 aa). The N-terminal and NAC regions contain seven highly repetitive sequences (KTKEGV). Upon binding to membrane structures, these sequences can form amphipathic α-helical structures that mediate the association of α-Syn with negatively charged phospholipid membranes. The N-terminal region harbors six genetic mutation sites that cause familial Parkinson's disease (PD). The C-terminal region contains two post-translational modification sites that are associated with α-synucleinopathies[5-7].

### 2.2. Physiological Functions

α-Synuclein is involved in regulating synaptic vesicle transport and exocytosis. It plays a role in modulating neurotransmitter release, synaptic function, and synaptic plasticity. However, its physiological functions under normal conditions are not yet fully understood [8].Studies have found that α-synuclein acts as a molecular chaperone for the soluble N-ethylmaleimide-sensitive factor attachment protein receptor (SNARE) complex. It can influence the assembly and distribution of the SNARE complex by controlling its degradation. The SNARE complex is directly responsible for facilitating the release of various neurotransmitters, including dopamine[9].Furthermore, α-synuclein possesses molecular chaperone-like activity, which can assist in the proper folding of other proteins and prevent their misfolding and aggregation. This helps maintain protein homeostasis within the cell. Additionally, α-synuclein plays a role in the cell's antioxidant response. It is capable of protecting cells from oxidative damage by scavenging free radicals, thereby ensuring the normal physiological functions and survival of cells.

## 3. Pathological Mechanisms of α-Synuclein in Parkinson's Disease

### 3.1. Aggregation and Fibrillation

In the brains of Parkinson's disease (PD) patients, the abnormal aggregation and fibrillation of α-synuclein (α-Syn) are key steps in the disease's progression. Under physiological conditions, α-synuclein predominantly exists as monomers. However, under the influence of genetic mutations, environmental toxins, and oxidative stress, its conformation is altered. The increasing β-sheet structure leads to intermolecular interactions and the formation of oligomers. These oligomers

further aggregate to form protofibrils, eventually forming mature fibrillar aggregates, which are the main components of Lewy bodies and Lewy neurites. These aggregates are not only cytotoxic in themselves, but also capable of disrupting normal physiological processes within cells, such as affecting the function of organelles and hindering the normal metabolism and transport of proteins, thereby causing neuronal damage and death.

### 3.2. Impacts on Mitochondria

With the advancement of biochemical research techniques, α-synuclein has been identified as the primary protein component of Lewy bodies[10]. Mitochondria are the key organelles for cellular energy production, and their function determines cellular status. Mitochondrial dysfunction is considered a core initiating factor in dopaminergic neuronal damage and is also one of the significant pathological features of Parkinson's disease[11]. Impaired mitochondrial function leads to cellular energy metabolism disorders, causing neurons to be unable to obtain a sufficient energy supply. This, in turn, accelerates the death process of neurons and exacerbates the progression of Parkinson's disease.

### 3.3. Impacts on the Endoplasmic Reticulum

The endoplasmic reticulum is a key organelle for protein folding, modification, and transport within the cell. Abnormalities in α-synuclein can impact endoplasmic reticulum function. Pathological aggregation of α-synuclein can induce endoplasmic reticulum stress responses. This imbalance in endoplasmic reticulum homeostasis leads to the accumulation of unfolded and misfolded proteins, which in turn induces endoplasmic reticulum stress and triggers apoptosis. This process also plays a significant role in the pathogenesis of Parkinson's disease[12].

### 3.4. Neuroinflammatory Responses

Significant neuroinflammation is present in the brains of Parkinson's disease (PD) patients, and α-synuclein is one of the key factors in triggering neuroinflammation. Lewy bodies, dopaminergic neuron damage, and neuroinflammation are typical pathological changes in PD. α-Synuclein, released from necrotic neurons, can be recognized by microglia and activate inflammatory signaling pathways, increasing the production of inflammatory factors and leading to immune responses and antigen clearance.However, chronic PD pathology leads to the presence of a large number of pathological microglia, which lose the ability to recognize and clear antigens. This results in chronic neuroinflammation, exacerbating neuronal damage and motor dysfunction and promoting the progression of Parkinson's disease[13].

### 3.5. Activation of Cell Death Pathways

The abnormal aggregation and toxicity of α-synuclein can activate multiple cell death pathways, leading to substantial neuronal loss. This is one of the ultimate outcomes of Parkinson's disease progression. Among these pathways, apoptosis is one of the important modes of neuronal death. α-Synuclein can activate apoptosis signaling pathways through both the mitochondrial pathway and the death receptor pathway, promoting the activation of apoptosis-related proteins within cells, such as caspase family proteases, thereby initiating the apoptotic program.Furthermore, the autophagy-lysosome pathway plays a significant role in Parkinson's disease. Moderate autophagy can clear abnormal α-synuclein and damaged organelles within the cell, providing a protective effect on cells. However, in Parkinson's disease, the abnormal aggregation of α-synuclein may

disrupt the autophagy process, leading to impaired autophagic flux, preventing the timely clearance of harmful substances within the cell, and ultimately leading to cell death.Recent studies have also revealed that α-synuclein may be associated with other cell death pathways, such as necroptosis and ferroptosis. These cell death pathways are interwoven and collectively promote the death process of neurons in Parkinson's disease. However, the specific molecular mechanisms still require further in-depth investigation.

## 4. Therapeutic Strategies Targeting α-Synuclein

### 4.1. Immunotherapy

In the field of neurodegenerative diseases, immunotherapeutic strategies targeting α-synuclein (α-Syn) are gaining increasing attention. The aggregation of α-synuclein is closely associated with the occurrence of synucleinopathies, such as Parkinson's disease (PD). The goal of immunotherapy is to activate the body's immune system to specifically recognize and eliminate abnormal α-synuclein, thereby slowing the progression of the disease[14].Studies suggest that the pathological propagation of α-synuclein may be similar to that of prions. This finding offers a new perspective for immunotherapy. In clinical studies, PD01A, a specific active immunotherapy targeting aggregated α-synuclein, has been evaluated for its safety and tolerability in patients with Parkinson's disease. Furthermore, using an adeno-associated virus (AAV)-mediated approach to enable the expression of the 306C7B3 antibody in the central nervous system offers a novel strategy for immunotherapy. In genetic models of α-synucleinopathy, AAV-mediated expression of 306C7B3 can significantly improve survival rates and achieve effective antibody concentrations in the cerebrospinal fluid[15].In summary, immunotherapeutic strategies targeting α-synuclein are actively being developed. Although these strategies face challenges, studies have demonstrated their therapeutic potential, providing new directions for the future treatment of Parkinson's disease and other synucleinopathies.

### 4.2. Small Molecule Inhibitors

Small molecule inhibitors are designed to reduce the toxic effects of α-synuclein by disrupting its aggregation process or inhibiting its interactions with other proteins. To date, a variety of small molecules capable of inhibiting α-synuclein aggregation have been identified, each with distinct mechanisms of action. As representative therapeutic agents for Parkinson's disease (PD), small molecule inhibitors of α-synuclein could also be considered as potential precursor compounds for PET tracers. Modifying existing compounds to specifically bind to α-synuclein and enable crossing of the blood-brain barrier and cell membranes is also an effective strategy[16].

### 4.3. Gene Therapy

Gene therapy employs genetic engineering techniques to modify or regulate genes associated with α-synuclein in patients, with the aim of treating Parkinson's disease. Gene therapeutic strategies primarily focus on reducing the expression levels of α-synuclein, inhibiting the formation of α-synuclein aggregates, promoting the degradation of these aggregates, and preventing their propagation. For example, reducing the expression of α-synuclein through RNA interference (RNAi) technology can decrease its accumulation and toxicity within cells. Furthermore, the use of gene-editing technologies such as the CRISPR/Cas9 system to directly target the SNCA gene represents a potential therapeutic approach.In preclinical studies, gene therapeutic strategies have demonstrated potential in mitigating neurotoxicity induced by α-synuclein. For instance, viral

vector-mediated gene transfer allows for the direct delivery of therapeutic genes to the brain, reducing the expression of α-synuclein or enhancing its clearance. Additionally, nanoparticles as non-viral vectors are being explored for gene therapy to improve the efficiency and safety of gene delivery[17].Although gene therapy has shown significant potential in the treatment of Parkinson's disease, it is still in the stages of experimental research and early clinical trials, facing numerous challenges such as gene delivery efficiency, off-target effects, immune responses, and ethical considerations that need to be addressed.

### 4.4. Other Emerging Treatment Modalities

Beyond the aforementioned traditional treatment strategies, the treatment approaches for Parkinson's disease are continuously evolving and innovating. In recent years, several emerging treatment methods have emerged, offering new hope for the treatment of Parkinson's disease. For example, cell-based therapeutic approaches, including stem cell transplantation and neural precursor cell transplantation, have shown promise. These cells can differentiate into dopaminergic neurons, replacing damaged neurons in Parkinson's disease patients. They can also secrete bioactive substances such as neurotrophic factors, promoting the survival and functional recovery of surrounding neurons.Additionally, treatment regimens for Parkinson's disease typically focus on dopamine replacement and symptom relief. However, current treatments can lead to adverse side effects, and there remains a significant unmet clinical need for treatments that provide disease modification and address levodopa-resistant symptoms[18].Nanotechnology can be applied in the therapeutic research of Parkinson's disease. Nanomaterials can serve as drug carriers to deliver therapeutic agents precisely to the affected brain regions, enhancing the efficacy of drugs and reducing their side effects. For example, nanoparticles can encapsulate drugs or nucleic acid molecules targeting α-synuclein. With surface modifications that allow them to pass through the blood-brain barrier, these nanoparticles can achieve targeted therapy against α-synuclein.However, these emerging treatment methods are mostly still in the laboratory research or early clinical trial stages. Further in-depth research and validation of their safety and efficacy are required.

## 5. Advances in Research on α-Synuclein and Parkinson's Disease

### 5.1. Novel Research Findings

Recent scientific research has revealed that the distribution and aggregation of α-synuclein within cells may be regulated by various intracellular signaling pathways. Specifically, changes in the activity of certain kinases and phosphatases may affect the phosphorylation status of α-synuclein, thereby altering its aggregation propensity. Research conducted using animal models of Parkinson's disease has shown that inhibiting the activity of specific kinases can reduce the phosphorylation of α-synuclein, which may help to decrease the risk of its aggregation into Lewy bodies. Furthermore, studies indicate that the interaction network between α-synuclein and other proteins may be more complex than previously thought. These newly identified protein interaction partners may play a significant role in the pathological processes of α-synuclein, offering potential targets for the development of new therapeutic approaches.

### 5.2. Exploration of Diverse Research Directions

#### 5.2.1. Fundamental Research Directions

①Conduct an in-depth investigation into the structural and functional relationships of

α-synuclein by utilizing high-resolution structural analysis techniques, such as cryo-electron microscopy. This will further elucidate the details of its conformational changes in both normal and pathological states, thereby providing a more precise structural basis for understanding its aggregation mechanisms.

②Investigate the intricate regulatory mechanisms of α-synuclein within cells, including the regulatory factors and signaling pathways involved in its synthesis, modification, and degradation, as well as its interaction network with other intracellular molecules. For instance, examine the interactions between α-synuclein and molecular chaperones, such as heat shock proteins, and how these interactions influence the folding and aggregation states of α-synuclein.

③Further investigate the mechanisms by which α-synuclein functions in synaptic plasticity. Synaptic plasticity is a crucial process that enables neurons to adapt to environmental changes and store information. α-Synuclein may influence synaptic transmission efficiency and plasticity changes through its interactions with postsynaptic receptors or related signaling molecules. A deeper understanding of this process could help reveal the underlying mechanisms of cognitive and motor dysfunction in patients with Parkinson's disease.

④Investigate the specific functions and pathological changes of α-synuclein in various brain regions. There are variations in the degree of involvement and pathological manifestations across different brain regions in patients with Parkinson's disease. Studying the functional changes of α-synuclein in specific brain regions—such as the substantia nigra, striatum, and cortex—and its interactions with local neural circuits can aid in gaining a deeper understanding of the region-specific pathological mechanisms of the disease.

### 5.2.2. Clinical Research Directions

①Conduct large-scale epidemiological studies to further identify the risk factors associated with α-synuclein-related Parkinson's disease. This includes examining the associations with environmental factors, lifestyle, and other aspects related to α-synuclein abnormalities, in order to provide a basis for disease prevention.

②Optimize the design of clinical trials for therapeutic strategies targeting α-synuclein to enhance the accuracy and reliability of therapeutic outcome assessments. For example, seek more sensitive and specific biomarkers for monitoring changes in α-synuclein levels or associated pathological processes during treatment.

③Investigate the relationship between α-synuclein-related biomarkers and disease progression, as well as therapeutic response in patients with Parkinson's disease. By conducting long-term follow-up of patients and collecting clinical data and biological samples, analyze the correlation between biomarkers such as α-synuclein, its phosphorylated forms, and oligomer levels, and patient motor and non-motor symptoms, disease progression rate, and responsiveness to different therapeutic drugs. This can provide guidance for personalized medicine.

④Explore early diagnostic methods based on the detection of α-synuclein. Currently, the diagnosis of Parkinson's disease primarily relies on clinical symptoms and lacks specific biomarkers for early diagnosis. Developing highly sensitive and specific α-synuclein detection technologies, such as novel immunoassay methods and biosensors, could enable early diagnosis of Parkinson's disease. This would facilitate early intervention and help slow the progression of the disease.

### 5.2.3. Insights from Research for Treatment

New research findings offer novel insights into the treatment of Parkinson's disease. For instance, based on research into the regulatory mechanisms of α-synuclein phosphorylation, it is possible to develop small molecule inhibitors targeting relevant kinases or phosphatases as potential

therapeutic agents. By modulating the interactions between α-synuclein and other proteins, it may be possible to prevent its aggregation and the spread of toxicity.Furthermore, a deeper understanding of intracellular regulatory mechanisms also aids in the development of more effective gene therapeutic strategies for the precise regulation of the expression and function of α-synuclein-related genes. Regarding the mechanisms of α-synuclein in synaptic plasticity and various brain regions discovered in basic research, future therapeutic strategies may consider interventions targeting specific brain regions or neural circuits to improve patients' cognitive and motor functions.In clinical research, more accurate biomarkers and early diagnostic methods will aid in selecting appropriate patients for early treatment and in monitoring treatment outcomes in real-time, allowing for timely adjustments to therapeutic plans.

## 6. Future Research Outlook

Although significant progress has been made in the study of α-synuclein and Parkinson's disease, numerous challenges remain to be addressed. Future research will require an integrated approach utilizing multidisciplinary techniques, including molecular biology, cell biology, neuroimaging, and bioinformatics, to delve into the complex molecular mechanisms of α-synuclein in Parkinson's disease.At the basic research level, further elucidate the structural dynamics of α-synuclein and its interaction network with other biomolecules, identify its key roles in intracellular signal transduction pathways, and provide more precise targets for the development of novel therapeutic agents. Concurrently, enhance research on animal models of Parkinson's disease to model the pathological processes and clinical characteristics of the disease, and to more comprehensively evaluate the efficacy and safety of potential therapeutic approaches.In clinical research, conduct multicenter, large-scale clinical trials to optimize treatment plans, enhance therapeutic outcomes, and reduce the incidence of adverse effects. Furthermore, with the advancement of precision medicine, it is anticipated that personalized treatment plans can be developed based on individual patient differences, such as genetic background and pathological characteristics of α-synuclein, to formulate personalized treatment plans, elevate the standard of care for Parkinson's disease, and improve patients' quality of life.In summary, there is still significant room for exploration of α-synuclein in the field of Parkinson's disease research, with the potential to bring new breakthroughs to the treatment of Parkinson's disease.

## 7. Conclusion

α-Synuclein plays a multifaceted role in the pathogenesis of Parkinson's disease, ranging from the disruption of its normal physiological functions to the pathological changes induced by its abnormal aggregation. These changes involve cellular and molecular events at multiple levels, including mitochondrial dysfunction, endoplasmic reticulum stress, neuroinflammation, and the activation of various cellular death pathways. A deeper understanding of the mechanisms by which α-synuclein functions in Parkinson's disease provides a solid theoretical foundation for the development of therapeutic strategies targeting α-synuclein[19].Currently, although various therapeutic approaches targeting α-synuclein have made certain advancements in preclinical studies and early-stage clinical trials, the realization of effective clinical treatment still faces numerous challenges. In future research, there is a need to further delve into the structural and functional relationships of α-synuclein, its intricate regulatory mechanisms within cells, as well as its complex interaction network with other biomolecules and signaling pathways. This will aid in the identification of new therapeutic targets and the development of more precise drug action sites, providing a scientific basis for the precision treatment of Parkinson's disease. Concurrently, collaborative efforts across disciplines, including neuroscience, biochemistry, molecular biology,

and clinical medicine, will accelerate breakthroughs and advancements in the treatment of Parkinson's disease.

## References


*[1] Zhang Z, Roman G, Hong Z, et al. Parkinson's disease in China: Prevalence in Beijing, Xian, and Shanghai [J]. Lancet, 2005, 365(9459): 595 - 597.*

*[2] De Lau L M, Breteler M M. Epidemiology of Parkinson's disease [J]. Lancet Neurol, 2006, 5(6): 525 - 535.*

*[3] Alves G, Forsaa E B, Pedersen K F, et al. Epidemiology of Parkinson's disease [J]. J. Neurol, 2008, 255(5): 18 - 32.*

*[4] Wang J Y, Ren B B, Ma S N, et al. Mechanism of α-synuclein -induced mitochondrial damage in Parkinson's disease [J]. Chinese Journal of Tissue Engineering Research, 2025, 29(17): 3668 - 3674.*

*[5] Hallacli E, Kayatekin C, Nazeen S, et al. The Parkinson's disease protein alpha - synuclein is a modulator of processing bodies and mRNA stability[J]. Cell, 2022, 185(12): 2035 - 2056.*

*[6] Zhang S, Zhu R, Pan B, et al. Post - translational modifications of soluble α - synuclein regulate the amplification of pathological α – synuclein [J]. Nat Neurosci, 2023, 26(2): 213 - 225.*

*[7] Tong J, Huang Y T, Li L X, et al. New research progress on the physiological functions and pathological roles of α-synuclein [J]. China Medical Herald, 2024, 37(3): 18 - 26. DOI: 10.19296/j.cnki.1008 - 2409.2024 - 03 - 003.*

*[8] Bendor JT, Logan TP, Edwards RH. The function of α - synuclein. Neuron, 2013, 79(6): 1044 - 1066.*

*[9] Burre J, Sharma M, Tsetsenis T, et al. c Alpha - synuclein promotes SNARE - complex assembly in vivo and in vitro. Sciene, 2010, 329(5999): 1663 - 1667.*

*[10] SIMON C, SOGA T, OKANO HJ, et al. α - Synuclein - mediated neurodegeneration in Dementia with Lewy bodies: the pathophysiology of a paradox. Cell Biosci. 2021; 11(1):196.*

*[11] MALPARTIDA AB, WILLIAMSON M, NARENDRAP D, et al. Mitochondrial Dysfunction and Mitophagy in Parkinson's Disease: From Mechanism to Therapy. Trends Biochem Sci. 2021; 46(4):329 - 343.*

*[12] Li Y H, Mou Z, Cui L H. Research progress on the regulation of the endoplasmic reticulum quality control system and its relationship with Parkinson's disease[J]. Advances in Physiological Science, 2024, 55(3): 278 - 284. DOI: 10.20059/j.cnki.pps.2023.11.1139.*

*[13] Cardinale A, Calabrese V, De Iure A, et al. Alpha - synuclein as a prominent actor in the inflammatory synaptopathy of Parkinson's disease. Int J Mol Sci, 2021, 22(12): 6517.*

*[14] Han S D, Cui M. Research progress on immunotherapy for synucleinopathies[J]. Chinese Journal of Clinical Neurosciences, 2018, 26(1): 86 - 95.*

*[15] Dühs, M., Blazevic, D., Rechtsteiner, P., Kenny, C., Lamla, T., Low, S., Savistchenko, J., Neumann, M., Melki, R., Schönberger, T., Stierstorfer, B., Wyatt, D., Igney, F., & Ciossek, T. (2023). AAV-mediated expression of a new conformational anti-aggregated α-synuclein antibody prolongs survival in a genetic model of α-synucleinopathies. NPJ Parkinson's disease, 9(1), 91. https://doi.org/10.1038/s41531-023-00542-9*

*[16] Yu H Y, Yan J J, Li W. Research progress on α-synuclein imaging agents and small molecule inhibitors as therapeutic drugs[J]. Journal of Shenyang Pharmaceutical University, 2022, 39(2): 233 - 242. DOI: 10.14066/j.cnki.cn21 - 1349/r.2020.0858.*

*[17] Sandoval, I. M., Marmion, D. J., Meyers, K. T., & Manfredsson, F. P. (2021). Gene Therapy to Modulate Alpha-Synuclein in Synucleinopathies. Journal of Parkinson's disease, 11(s2), S189–S197. https://doi.org/10.3233/JPD-212679*

*[18] Elkouzi, A., Vedam-Mai, V., Eisinger, R. S., & Okun, M. S. (2019). Emerging therapies in Parkinson disease - repurposed drugs and new approaches. Nature reviews. Neurology, 15(4), 204–223. https://doi.org/10.1038/s41582-019-0155-7*

*[19] Sun S N, Xiao Q, Fan L S, et al. Current research status of α-synuclein and Parkinson's disease[J]. The Chinese Journal of Clinical Pharmacology, 2023, 39(8): 1201 - 1205. DOI: 10.13699/j.cnki.1001 - 6821.2023.08.029.*