



The Role of Medications in Treating Neurological Disorders like Parkinson's and Alzheimer's

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Abstract

Neurological disorders encompass a group of diseases characterized by the progressive degeneration of the nervous system that manifests with a wide spectrum of cognitive, motor, and mood disturbances. Among these disorders, Parkinson's and Alzheimer's diseases constitute the most prevalent and devastating clinical entities, affecting millions of individuals worldwide. Although advances have been achieved in the understanding of the pathogenic mechanisms of these diseases, considerable efforts are still necessary to develop therapeutic interventions that can prevent, slow, or reverse the damaging effects of the pathology. While the implementation of non-pharmacological strategies such as physical, cognitive behavioural therapies, and dedicated lifestyle changes play an important role, pharmacological treatments are essential to improve outcomes and mitigate the course of illness.

Parkinson's Disease (PD) is a progressive multisystem neurodegenerative disorder with unknown cause, characterized by loss of dopaminergic neurons in the substantia nigra pars compacta and other regions of the brain. The loss of DA neurons results in biochemical changes with heightened cholinergic tone and abnormal beta-amyloid (A β) metabolism, which both contribute to the manifestation of motor symptoms, including bradykinesia, rigidity, resting tremor, and postural instability. In addition, a variety of non-motor symptoms develop throughout the course of the disease, with many initially emerging prior to the onset of motor symptoms. In light of these features, the therapy of PD remains problematic and primarily symptomatic. The development of increasingly individualized dopaminergic and non-dopaminergic treatment options has largely improved the situation. Patients are exposed to elevated risks of adverse reactions and complications as a consequence of chronic dopaminergic therapy (Musey & Shivkumar, 2017). Treatment strategies should be planned carefully in order to reduce the incidence of adverse events, improve tolerability, and extend the therapeutic efficacy.



Alzheimer's disease (AD) is the most common neurodegenerative disorder in adults. It is characterized by progressive impairments in memory, behavior, and the ability to perform everyday tasks. The histopathological hallmarks of AD include extracellular amyloid plaques composed primarily of amyloid- β ($A\beta$) peptide and intracellular neurofibrillary tangles assembled from hyperphosphorylated tau proteins. The formation of these pathological protein aggregates compromises neuronal and synaptic function, thereby leading to neurodegeneration associated with cognitive deterioration and memory loss. Genetic factors contributing to AD susceptibility include mutations of the genes encoding the amyloid precursor protein (APP), Presenilin 1, and Presenilin 2. These mutations increase the production and aggregation of $A\beta$ peptides, thereby initiating the pathological processes underlying AD. A variety of potential risk factors associated with AD have been identified in recent years, such as low educational attainment, midlife hypertension, midlife obesity, hearing loss, depression, diabetes, physical inactivity, smoking, and social isolation. Pharmacological approaches remain the mainstay of AD treatment, predominantly focusing on symptom management to improve the quality of life for affected individuals (Thangwaritorn et al., 2024).

Keywords: Neurological disorder; Parkinson's disease; Alzheimer's disease; dopaminergic medication; cholinesterase inhibitor; glutamate modulator; cognitive-behavioral therapy; deep-brain stimulation

1. Introduction

Neurological disorders encompass a broad category of ailments affecting the brain, spinal cord, and nerves. Together, these diseases are among the leading causes of disability and death worldwide. Neurodegenerative diseases are the most prevalent class of neurological disorders, characterized by progressive neuronal loss that erodes motor, cognitive, and behavioral functions. Among these, Parkinson's disease (PD) and Alzheimer's disease (AD) are most widespread, generating immense social and economic burdens (Musey & Shivkumar, 2017). The two diseases differ in age of onset, clinical features, and neuropathological hallmarks yet share a chronic progressive course with no approved disease-modifying treatments. Several classes of medications are available to ameliorate symptoms. Dopaminergic agents—levodopa, dopamine receptor agonists, and monoamine oxidase type B inhibitors—are widely used to treat PD. Cholinesterase inhibitors and glutamate regulators prime the pharmacological armamentarium to alleviate symptoms in AD (Hickey & Stacy, 2011).

2. Overview of Neurological Disorders

Neurological disorders, which impair the structure, function, conduction, or chemistry of the nervous system, encompass stroke, multiple sclerosis, Alzheimer's disease, dementia,



Parkinson's disease, and many others (Musey & Shivkumar, 2017). They may occur congenitally, as a result of brain trauma or stroke, or manifest through degenerative processes later in life (Behl et al., 2022). Central nervous system diseases that resulting from restricted or poor blood supply are referred stroke, such as venous thrombosis and cerebral vasospasm. Multiple sclerosis is a chronic inflammatory demyelinating disorder of autoimmune origin, affecting more than 2.5 million people worldwide (Mathur et al., 2023). Alzheimer's and Parkinson's diseases are the two neurodegenerative disorders with the highest prevalence, affecting nearly 44 and 30 million individuals, respectively. In the United States in 2022, approximately 2 million individuals were affected by Alzheimer's, while about 1 million suffered from Parkinson's. Given the considerable impact of neurological disorders and significant gaps in therapeutic procedures, the present study focuses on Parkinson's and Alzheimer's diseases.

Parkinson's disease (PD) is a neurodegenerative disorder characterized by progressive loss of nigrostriatal dopaminergic neurons population. Parkinson's symptoms typically start unilaterally and spread bilaterally as the illness progresses, accompanied by other motor and non-motor deficits linked to dopamine abnormalities. Diagnosis relies on the presence of bradykinesia plus either rigidity, resting tremor, or postural instability. Medical therapies for motor symptoms include MAO-inhibitors, rasagiline, entacapone, tolcapone, and extended-release carbidopa-levodopa. Since many patients initially contact primary care physicians, they must determine appropriate candidates for therapy and select effective medications and doses in terms of diagnosis, ensuring sustained benefit while minimizing adverse effects. Multiple deficits in PD and Parkinsonian disorders are associated with dysfunctions of the neurotransmitter dopamine.

2.1. Definition and Classification

Neurological disorders encompass any condition affecting the nervous system, including the brain, spinal cord, or nerves. They may influence activities such as muscle control, vision, balance, and sensation. The term includes dementia, epilepsy, migraine, and stroke, among others. According to the World Health Organization (WHO), one billion people worldwide suffer from neurological disorders; moreover, it is estimated that neurological disorders account for 16.5% of global deaths, with a more significant impact on low- and middle-income countries. Relatedly, millions of people worldwide suffer from Parkinson's and Alzheimer's diseases, neurological conditions that greatly affect patients' quality of life as well as that of their caregivers (Musey & Shivkumar, 2017) (Thangwaritorn et al., 2024). According to the WHO, conclusive data exists on the major neurological disorders in Parkinson's and Alzheimer's.



2.2. Epidemiology

Neurological disorders comprise a spectrum of diseases affecting different organs and systems. Although relatively rare when compared to cancer or cardiovascular diseases (Mathur et al., 2023), the estimated prevalence stands at over 6% of the population (Musey & Shivkumar, 2017). Few resources are required to develop neurological pathologies, yet they often have drastic effects on the patient's regular life. Currently, there is an unmet demand to improve treatment and medication and enhance the quality of life of people affected by neurodegeneration and other neurological pathologies (Wang et al., 2013). Parkinson's and Alzheimer's diseases stand out as the two most common neurodegenerative diseases worldwide.

3. Parkinson's Disease

Parkinson's disease is a neurodegenerative disorder characterized by the progressive loss of nigrostriatal dopaminergic neurons. It manifests with motor symptoms such as bradykinesia, rigidity, resting tremor, and postural instability; cognitive impairment is also common. The diagnosis requires the presence of bradykinesia and at least one additional motor symptom: muscular rigidity, 4–6 Hz resting tremor, or postural instability not caused by primary visual, vestibular, cerebellar, or proprioceptive dysfunction. Many Parkinsonian symptoms can be attributed to abnormalities in dopamine neurotransmission (Musey & Shivkumar, 2017). Pharmacological treatment primarily targets dopamine deficiency, employing levodopa, which remains the most effective therapy despite motor complications including wearing-off and dyskinesias that emerge after long-term use in many patients. Because additional neurotransmitter systems, such as acetylcholine and glutamate, may also contribute to Parkinsonian symptoms, ongoing research seeks novel therapeutic strategies and technologies to improve disease management (Hickey & Stacy, 2011).

3.1. Pathophysiology

Parkinson's disease, first described as Shaking Palsy by James Parkinson in 1817, is a neurodegenerative disorder characterized by the progressive loss of nigrostriatal dopaminergic neurons. It presents with motor features such as bradykinesia, rigidity, resting tremor, or postural instability. Cognitive impairment often accompanies the motor manifestations. Similar to Parkinson's, Alzheimer's disease stands as one of the most prevalent neurodegenerative disorders in the elderly population.

Within the central nervous system, neurons utilize neurotransmitters—principally glutamate—to facilitate communication. The accumulation of neurotransmitters, particularly glutamate, in brain tissue can precipitate neuronal injury and subsequent cell death. Excessive stimulation of neurons triggers apoptotic pathways, thereby contributing to neurodegenerative cascades (Behl et al., 2022). The pathophysiology of Alzheimer's disease encompasses the



accrual of amyloid- β protein, aberrant phosphorylation of tau proteins, and pervasive neuroinflammatory processes. Microglia and astrocytes release neuroinflammatory mediators, including cytokines and chemokines, which modulate neuronal survival and plasticity. Oxidative stress further exacerbates neurodegeneration by generating reactive oxygen and nitrogen species that damage neuronal cells, compromise DNA integrity, and impair mitochondrial function. Mitochondrial dysfunction and oxidative injury synergistically contribute to disease progression.

The immune system, particularly microglial activation, plays a vital role in mediating neuroinflammation and subsequent neuronal damage. Epigenetic modifications, such as DNA methylation and histone acetylation, also influence the trajectory of neurodegenerative disorders. Agents targeting epigenetic regulators, notably histone deacetylase inhibitors, demonstrate therapeutic potential by ameliorating disease phenotypes. Collectively, these mechanisms underpin the clinical manifestations and therapeutic targets for Parkinson's and Alzheimer's diseases (Musey & Shivkumar, 2017).

3.2. Symptoms and Diagnosis

Parkinson's disease, a neurodegenerative disorder marked by the gradual loss of nigrostriatal dopaminergic neurons, presents with cardinal motor features such as bradykinesia, rigidity, resting tremor, and postural instability, often initiating asymmetrically. Non-motor symptoms, including cognitive impairment, further contribute to disability. Diagnostic criteria remain clinical, leading many primary care physicians to establish the diagnosis and commence treatment. Therapeutic challenges encompass the selection of appropriate pharmacologic agents and dosing schedules; medications are frequently employed at incorrect doses or intervals, necessitating a thorough understanding of drug-specific side-effect profiles and therapeutic rationales (Musey & Shivkumar, 2017). Parkinson's disease stems from the degeneration of dopaminergic neurons in the substantia nigra, an etiology influenced by aging, genetic, and environmental factors. The resultant striatal dopamine depletion precipitates movement disorders, with early manifestations comprising tremor, muscle rigidity, bradykinesia, and impaired motor coordination. Accompanying non-motor symptoms such as depression, constipation, and insomnia further diminish quality of life, underscoring the imperative for effective treatment modalities (Yu et al., 2017).

3.3. Current Treatment Options

Pharmacological treatment is the predominant approach for neurological disorders such as Parkinson's and Alzheimer's diseases. Pharmacotherapeutic options for Parkinson's include levodopa, dopamine agonists, monoamine oxidase type B inhibitors, amantadine, anticholinergics, and deep brain stimulation. Acetylcholinesterase inhibitors are the main choice of treatment for Alzheimer's disease, which also includes N-methyl-d-aspartate



receptor antagonist memantine. Since the mechanisms underlying these disorders are still unclear, the currently available pharmacological treatments have limited ability to provide relief from symptoms or to delay or stop neuronal degeneration (Thangwaritorn et al., 2024) (M. Ringman & L. Cummings, 2006).

3.4. Role of Medications

Parkinson's disease occurs due to the death of dopaminergic neurons in the substantia nigra and the presence of Lewy body inclusions (Behl et al., 2022). The disease affects approximately 2% of individuals older than 65 years. Symptoms include rigidity, bradykinesia, postural instability, and resting tremor, which only become overt after the loss of 50-80% of dopaminergic neurons in the substantia nigra. Diagnosis relies primarily on the presence of motor symptoms and a positive response to dopaminergic medications. Several treatment options are available to alleviate symptoms, including medications, deep brain stimulation, and physical therapy. The first line of treatment often involves dopaminergic agents. Medication types include levodopa/Carbidopa, dopamine agonists, and monoamine oxidase B (MAO-B) inhibitors. Levodopa, the most effective symptomatic treatment, converts to dopamine in the brain (Thangwaritorn et al., 2024).

4. Alzheimer's Disease

Alzheimer's disease (AD) is the most common neurodegenerative disorder among adults and is characterized by the deterioration of cognition, behavior, and functionality (Thangwaritorn et al., 2024). The hallmark histopathological feature is the accumulation of abnormal proteins that form neurofibrillary tangles and amyloid plaques, which impair neuronal and synaptic function (Lleó, 2007). Pathophysiological mechanisms consist of aberrant processing of the amyloid precursor protein (APP) and tau, generating beta-amyloid ($A\beta$) fibrils and neurofibrillary tangles. The deposition of these aggregates generally correlates with cognitive decline. Genetic contributors include mutations in the APP, Presenilin 1, and Presenilin 2 genes, all of which encode the amyloid beta peptide. Additional risk factors extend to low educational attainment, hypertension, obesity, hearing loss, depression, diabetes, physical inactivity, smoking, and social isolation. Early therapeutic strategies concentrated on alleviating the deficits of acetylcholine through the administration of cholinesterase inhibitors—agents that remain widely studied and continue to represent a key target for future interventions.

4.1. Pathophysiology

This paper focuses on the role that medications play in treating two widespread neurological disorders—Parkinson's and Alzheimer's. Neurological disorders are a diverse set of diseases involving the nervous system and the brain, and dopaminergic, cholinergic, and glutamatergic medications feature widely in their treatment.



Parkinson's disease is a common neurodegenerative disorder defined by bradykinesia, resting tremor, rigidity, and postural instability (Hickey & Stacy, 2011). Age is a significant risk factor, and developing treatments that address symptomatic relief and disease progression remains an important research priority (Musey & Shivkumar, 2017). Since 1968, levodopa has been the most effective symptomatic treatment, but its long-term use commonly induces motor complications such as wearing off, dyskinesias, and the on-off phenomenon. These complications may occur in 40 to 50% of patients treated for five to 10 years. Levodopa addresses the cardinal features of dopamine deficiency, but additional putative pathogenetic mechanisms in the disease involve other neuronal pathways mediated by acetylcholine, glutamate, and N-methyl-D-aspartate receptors. Because dopaminergic medications do not adequately control the full spectrum of symptomatology in PD, adjunctive non-dopaminergic therapies continue to be a focus of research.

4.2. Symptoms and Diagnosis

The diagnosis of Parkinson's disease relies on clinical observation rather than diagnostic tests. The hallmark features of the disease include a resting tremor (which decreases during movement and sleep), asymmetry (typically beginning on one side), bradykinesia (where patients perceive their movements as slowed), rigidity (resulting in resistance during passive limb movement), and postural instability (leading to frequent falls). These motor symptoms are accompanied by a spectrum of nonmotor manifestations such as constipation, anosmia, depression, REM sleep behavior disorder, and other sleep impairments, as well as fatigue. Cognitive decline also represents a prevalent and disabling aspect of the disease. Many of these clinical manifestations stem from abnormalities in dopamine neurotransmission, notably the degeneration of dopaminergic neurons within the nigrostriatal pathway (Musey & Shivkumar, 2017) (Yu et al., 2017).

4.3. Current Treatment Options

Several medications are currently approved by the US Food and Drug Administration for treating Parkinson's disease; however, there is no single drug regimen that effectively addresses the diverse symptoms of Parkinson's or Alzheimer's. In Parkinson's disease, levodopa combined with carbidopa remains the primary treatment. If a patient responding to levodopa develops dyskinesia or motor fluctuations, adjunct drugs such as dopamine agonists, catechol-O-methyltransferase (COMT) inhibitors, monoamine oxidase-B (MAO-B) inhibitors, or amantadine may be added (Thangwaritorn et al., 2024). For Alzheimer's disease, acetylcholinesterase inhibitors—including donepezil, galantamine, and rivastigmine—are prescribed, and memantine—a moderate-affinity, uncompetitive NMDA receptor antagonist—is an option for moderate-to-severe cases (M. Ringman & L. Cummings, 2006).



4.4. Role of Medications

Medications play a pivotal role in managing neurological disorders such as Parkinson's and Alzheimer's diseases. Parkinsonian disorders, traditionally classified based on the MDS classification, respond variably to drug treatments, with dopaminergic agents constituting the primary therapeutic strategy for Parkinson's disease. The pharmacological modulation involves numerous medication categories affecting various neurotransmitter systems. L-dopa remains the most efficacious drug for Parkinson's disease, enhancing voluntary movement and alleviating rigidity but exerting minimal influence on tremor. Administration practices seek to maintain a consistent plasma concentration to optimize symptomatic control and delay the onset of motor fluctuations. Alternatives and adjuncts to L-dopa encompass dopamine agonists—delivered via oral, transdermal, subcutaneous, or apomorphine infusions—MAO-B inhibitors, COMT inhibitors, anticholinergics, and amantadine, each contributing to the dopaminergic milieu through distinct mechanisms. In Alzheimer's disease, therapeutic agents augment cholinergic transmission, predominantly through cholinesterase inhibitors such as donepezil, galantamine, and rivastigmine, given the elevated acetylcholine metabolism observed in afflicted patients. Memantine, an NMDA receptor antagonist, often complements cholinesterase inhibitors by targeting glutamatergic pathways implicated in neurodegeneration. Beyond pharmacological interventions, therapeutic strategies encompass physical exercise, cognitive-behavioral therapy, speech therapy, occupational therapy, and dietary considerations, thereby supporting comprehensive patient management and addressing motor and non-motor symptoms inherent to both disorders (Thangwaritorn et al., 2024) (Behl et al., 2022).

5. Types of Medications Used

Drugs for neurological and psychiatric disorders that affect the central nervous system which are often classified according to the chemical structures of the active ingredient. Parkinson's disease is a hypokinetic movement disorder characterized by progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta. Re-establishment of dopamine levels in the basal ganglia remains the most effective symptomatic treatment. The main classes of medications used are discussed to facilitate the clinician's understanding of their uses, benefits, limitations and risks (Musey & Shivkumar, 2017). The principal drugs used include levodopa, dopamine agonists, monoamine oxidase type B inhibitors and catechol-orthomethyltransferase inhibitors. Alzheimer's disease is the most common neurodegenerative disorder among adults characterized by the accumulation of abnormal aggregates of specific proteins in the brain (Thangwaritorn et al., 2024). Aggregates of the amyloid plaques and neurofibrillary tangles impair neuronal and synaptic function and play a central role in cognitive decline. The limited availability of acetylcholine is one of the earliest



alterations that was considered a strategic target and cholinesterase inhibitors have remained by far the most widely evaluated drugs for Alzheimer's disease.

5.1. Dopaminergic Medications

Dopaminergic medications form the core pharmacological approach to Parkinson's disease, a neurodegenerative disorder marked by motor symptoms such as bradykinesia, resting tremor, rigidity, and postural instability (Hickey & Stacy, 2011). Aging remains the principal risk factor contributing to the progressive loss of nigrostriatal dopaminergic neurons underlying the symptomatology. Levodopa, introduced in 1968, continues as the most potent agent for motor symptom relief, though long-term use frequently results in motor complications including wearing off, dyskinesias, and "on-off" phenomena. Contemporary management often incorporates adjunct dopaminergic agents: dopamine agonists such as ropinirole and pramipexole, monoamine oxidase type B (MAO-B) inhibitors like rasagiline and selegiline, catechol-O-methyltransferase (COMT) inhibitors including entacapone and tolcapone, as well as ancillary anticholinergic and N-methyl-D-aspartate (NMDA) antagonists (Musey & Shivkumar, 2017). Appropriate selection and dosing of this pharmacopoeia enable clinicians to optimize symptomatic control while mitigating adverse effects. Research initiatives target the development of disease-modifying therapies addressing the pathophysiological basis of dopamine neuron degeneration, with several candidates advancing through phase 2 or later clinical evaluation (Murakami et al., 2023).

5.2. Cholinesterase Inhibitors

Cholinesterase inhibitors prevent the degradation of acetylcholine and thereby improve cholinergic neurotransmission; an important consideration in Alzheimer's disease (AD), where acetylcholine-producing neurons degenerate (Vecchio et al., 2021). Three reversible cholinesterase inhibitors—donepezil, galantamine, and rivastigmine—have reached the market and are widely used in mild to moderate AD (M. Ringman & L. Cummings, 2006). Tacrine, the first marketed acetylcholinesterase (AChE) inhibitor, was withdrawn due to hepatotoxicity.

Cholinesterase inhibitors produce modest symptomatic benefits on cognition for a limited period. Adverse effects such as nausea, diarrhea, vomiting, muscle cramps, anorexia, and dizziness occur frequently but rarely require treatment discontinuation. Cardiac arrhythmias and other cardiac side effects, which may lead to treatment interruptions, are less common. Absolute contraindications to cholinesterase inhibitors are lacking, but caution is advised in patients at risk of gastrointestinal bleeding or with pre-existing bradycardia.

5.3. NMDA Receptor Antagonists

Numerous pharmacological agents address cognitive, psychiatric, and motor difficulties in neurological disorders. Attention to NMDA receptor antagonists emerged as memantine's



neuroprotection against excitotoxicity appeared to benefit Alzheimer's disease (AD) (Vanle et al., 2018). Subsequent research suggested additional NMDA antagonists could benefit Parkinson's disease (PD). In general, NMDA receptors act as glutamate-dependent cation channels with major roles in long-term potentiation and synaptic plasticity, the cellular basis of learning and memory. The pharmacology and therapeutic value relate to subunit composition and subcellular location, including extrasynaptic sites emerging with normal ageing (Olatunde Egunlusi & Joubert, 2024). Placements within a basal ganglia network underlie the relatively greater roles of glutamate in basal ganglia structure and function in PD.

PD stems from nigrostriatal dopamine depletion, placing a premium on agents restoring or supplementing dopamine-mediated transmission throughout the basal ganglia network. Despite clinical enthusiasm, favourable effects often reflect non-selective or rapid-release agents rather than agents arranged to smooth delivery. Consequently, prescriptions often alternate between different drug preparations to exploit each class' relative advantages and to ensure an acceptable benefit-to-risk ratio. In addition to dopaminergic agents, selegiline and rasagiline slow dopamine denaturation. Initially regarded as monotherapy agents, amantadine and anticholinergic agents give additional symptomatic relief when combined with a dopaminergic agents. When accounting for beneficial but less-prominent effects and adverse effects, the relatively few agents currently available respond to the general classes of dopamine, acetylcholine, and putative glutamate. Other classes remain older, marginal, or under investigation.

5.4. Antidepressants and Antipsychotics

Antidepressants and antipsychotics find utility in mitigating the neuropsychiatric manifestations associated with certain neurological disorders. Depression in Parkinsonism may respond to serotonin-selective reuptake inhibitors. Caution is warranted in using antipsychotics due to an increased risk of sudden death among subjects with dementia, particularly those with dementia with Lewy bodies. Conventional antipsychotics such as haloperidol, flupentixol, and pimozide should be avoided owing to their potential to worsen extrapyramidal symptoms. Atypical antipsychotics with a more favorable profile for Parkinsonism-induced symptoms include clozapine, quetiapine, risperidone, olanzapine, and amisulpride.

The presence of hallucinations, delusions, or psychosis can markedly diminish the quality of life for individuals afflicted with Parkinson's and Alzheimer's diseases. Reduced dopamine concentrations within the mesocortical-limbic pathways appear to contribute to the emergence of psychosis in Parkinson's disease. In the case of Alzheimer's disease, acetylcholine deficiency affecting these pathways may serve as a predisposing factor for such psychiatric disturbances. Consequently, raising cerebral dopamine and acetylcholine



concentrations through pharmacological means has been shown to alleviate these symptoms. In Parkinson's disease, the aim is to augment dopamine levels, whereas in Alzheimer's disease, enhancing acetylcholine concentrations is the therapeutic focus.

6. Mechanisms of Action

Dopaminergic, cholinergic, and glutamatergic receptors modulate the rate of cognitive information processing, mood, motor function, and muscle tone. Each of these plays a significant role in the symptomatology of those with Parkinson's or Alzheimer's disease. Dopamine deficiency usually gives rise to stooped posture, reduced voluntary movement, and muscle rigidity; acetylcholine deficits explain the inability to sustain a motor task over time in conjunction with the memory and attention deficits observed in Parkinson's and Alzheimer's disease. Neurodegeneration associated with these diseases and other chronic neurological disorders is complex. It involves N-methyl-D-aspartate (NMDA) glutamate receptor-induced excitotoxicity, oxidative stress, mis-metabolism of oxygen and/or dopamine, iron dyshomeostasis, and decreased endogenous neuroprotective molecules such as brain-derived neurotrophic factor and testosterone. Scopolamine and other anticholinergic drugs evoke amnesia and related cognitive deficits, whereas cholinesterase inhibitors boost acetylcholine levels and exert pro-cognitive effects. Amantadine and memantine are NMDA receptor antagonists that also trigger dopamine release and inhibit the reuptake of dopamine; they improve cognitive, affective, and motor deficits in patients with neurological disorders (Behl et al., 2022) (B. H. Youdim, 2010).

6.1. Dopamine Replacement

Dopamine replacement represents the most frequently prescribed treatment for Parkinson's disease (PD). Levodopa (L-DOPA), a dopamine precursor, remains a cornerstone therapy (A. MacDonald & Monchi, 2011). Since dopamine itself cannot cross the blood-brain barrier, levodopa bypasses this limitation by entering the brain and converting to dopamine, thereby enhancing dopaminergic signaling. A notable complication of levodopa therapy, especially during prolonged use, involves the development of dyskinesias. To mitigate this risk, levodopa is usually co-administered with a dopa decarboxylase inhibitor, which prevents peripheral conversion of levodopa to dopamine. This combination not only diminishes peripheral dopamine-related side effects but also allows for a reduction in the therapeutic levodopa dose. Dopamine receptor agonists provide an alternative strategy by directly stimulating dopamine receptors. These agents may exert their effects independently of presynaptic dopaminergic neurons or stimulate residual neuronal cells to release stored dopamine. Apomorphine, a nonselective dopamine receptor agonist, is employed for the acute management of severe "off" episodes—periods when medication effects wane, and PD symptoms emerge. The pharmacological profile of dopamine replacement therapies reflects the intricate balance required to address the substantial loss of dopamine in the dorsal



striatum, which is profoundly depleted throughout all stages of PD, versus the relatively spared ventral striatum—particularly in early disease. Administration is titrated primarily to compensate for dorsal striatal dopamine deficiency supporting motor functions; this can lead to dopamine overdose in the ventral striatum, potentially impairing cognitive processes. Clinical observations corroborate the complementary roles of dopamine replacement, with improvements documented in attention shifting, response selection, and the manipulation of working memory contents. Conversely, verbal fluency, prospective remembering, and action planning, which deteriorate in the unmedicated state, are ameliorated under treatment.

6.2. Acetylcholine Modulation

Cholinesterase inhibitors are the first class of drugs approved for the treatment of Alzheimer's disease (AD). They partially or completely block the metabolic breakdown of the neurotransmitter acetylcholine, thus increasing its levels in the brain. Three different inhibitors are currently approved by the U.S. Food and Drug Administration for treatment of mild to moderate AD. Two of these drugs are tacrine and donepezil, described above for their dopaminergic neuroprotective effects. The third agent is rivastigmine, a derivative of physostigmine that inhibits both acetylcholinesterase and butyrylcholinesterase. The mechanism of this drug class is believed to promote cholinergic activity in the brain. However, the mechanism of action is not yet clearly defined and these agents have shown modest improvement in cognition and behavioral symptoms compared to placebo in AD patients. Common side effects include nausea, vomiting, and diarrhea, which often limit dose escalation.

Donepezil has also been approved for treatment of Parkinson's disease dementia (see above). While cholinesterase inhibitors have not been shown to improve the cognitive function of non-demented PD patients, rivastigmine has in one small study shown some effect on levodopa-induced tremor. It has been suggested that the cholinergic system has a role in the mechanism of tremor in PD and clinical trials using cholinesterase inhibitors for their antitremor effect in PD patients with mild or no dementia may be warranted.

6.3. Glutamate Regulation

The development of new therapies for PD remains a challenge despite improved understanding of molecular and cellular mechanisms. Studies of glutamate receptor changes during the disease provide an opportunity to identify novel treatments. While the complexity of basal ganglia circuitry precludes simple solutions, targeting specific glutamate receptor subtypes within distinct pathways may be effective for selective design of new agents (Zhang et al., 2019).

Because there are different subtypes, selective targeting of metabotropic glutamate receptors might avoid the side effects associated with antagonists of AMPA, NMDA, or kainate



receptors (Gasparini et al., 2013). New compounds acting on these receptors are currently in clinical development or already available. The understanding of glutamate receptor involvement in PD pathophysiology thus opens new avenues for therapeutic intervention.

Analysis of glutamate concentrations, receptors, and associated intracellular signaling cascades remains a priority for the discovery of more effective, clinically translatable medications to control PD and LID. Identification of new molecules or biologicals targeting glutamate receptors may provide innovative tools to limit progression of PD.

7. Efficacy of Medications

When assessing the latest treatment options for Parkinson's disease (PD) and Alzheimer's disease (AD), it seems prudent to focus on the role of medications because the categories of the drugs used are essentially the same. Both conditions are neurological disorders classified as neurodegenerative: in PD, the death of cells in an area of the brain called the substantia nigra inhibits the production of dopamine — affecting movement, balance, coordination, speech, and other functions. By contrast, in AD, the degeneration of the hippocampus inhibits the production of acetylcholine — disrupting memory formation and language identification. Both disorders also involve the neurotransmitter glutamate. To compensate for the double effect of these diseases, both dopaminergic and anticholinergic drugs are necessary and hence, the therapeutic options for these two brain disorders share many resemblances.

Parkinson's disease results when dopaminergic neurons that are responsible for the production of dopamine stop functioning. These unbalanced levels of dopamine create a distinctive slowing down of voluntary movements; in parallel, symptoms like abnormal body posture, shaking of fingers and hands, rigidity, and reduced facial expression are present. Because the biochemical approach to Parkinson's is based on the fact that there is a deficiency of dopamine in the corpus striatum, the primary aim of the drug treatment is to replenish dopamine levels by either increasing the biosynthesis of dopamine or stimulating the dopamine receptors or decreasing the metabolism of dopamine — that is, increasing the levels of dopamine already present. In this sense, treatment is briefly summarized, highlighting the benefit of pharmacotherapy in increasing the quality of life of patients, even though no diagnosis is available until more than 50% of dopaminergic neurons are dead.

7.1. Clinical Trials and Studies

Clinical trials play a pivotal role in identifying pharmacological treatments for neurological disorders (E. Oxford et al., 2020). Alongside animal models, clinical trials enable the assessment of a chemical compound's therapeutic potential before regulatory approval and widespread clinical use. These trials proceed from initial human testing through three progressive phases. Phase I evaluates safety and tolerance and establishes suitable dosage levels. Phase II investigates efficacy and dose range in the target patient population. Phase III



compares the new treatment against current standard-of-care therapies. Successful completion of Phase III trials usually leads to Marketing Authorization Application for regulatory approval.

Phase III trial datasets are publicly registered at ClinicalTrials.gov, facilitating awareness of ongoing trial activity and subsequent research evaluations of medication effectiveness. Following short-to medium-term trial durations, long-term observational studies monitor safety profiles and disease progression under treatment (McFarthing et al., 2020). Such studies provide critical evidence of sustained clinical benefit and risks influencing treatment selection.

Over the past decades, clinical trials evidence has guided the evolution of medications to manage symptoms and enhance quality of life for Parkinson's and Alzheimer's disease patients. Several anti-parkinsonian and cognition-enhancing medications have reached the market, yet no clinically approved option exists to halt or reverse either disorder. The recent toll extracted by the COVID-19 pandemic underscores the urgency of developing disease-modifying therapies.

Recruitment to prospective medication trials faces barriers including hesitancy about potential side effects, a concern pronounced among the elderly population targeted by neurodegenerative medications (Calamia et al., 2016). Optimized communication of study information—presenting risk and benefit data with unambiguous, relatable terminology—may increase participation rates, thereby expediting the discovery of improved therapeutic options.

7.2. Long-term Outcomes

Long-term studies of drug interventions commonly extend beyond one year to evaluate efficacy and safety. However, randomized trials administered open-label cholinergic treatments to untreated subjects, assessing outcomes accordingly (Ballard et al., 2008). The benefits of neuroleptic treatment are inherently limited by such study designs, and it is difficult to exclude the occurrence of significant but very rare adverse effects. A proportion of patients discontinue treatment due to adverse events (Hickey & Stacy, 2011). The absence of specific treatment for Alzheimer's disease means that cholinesterase inhibitors are rarely discontinued due to disease progression. Conversely, disease progression remains the predominant reason for cessation of anti-parkinsonian medication.

8. Side Effects and Management

Side effects often limit benefits of pharmacotherapies, but they can be minimized by taking recommended doses and having regular follow-ups with healthcare providers. For example, patients should keep their levodopa dose low initially, in order to minimize levodopa-induced dyskinesia.



Dopaminergic medications may induce nausea, hypotension, confusion, hallucinations, and dyskinesias. Excess glutamate activity causes excitotoxicity. This toxic action causes calcium influx and boosts intracellular calcium, triggering activation of enzymes, which in turn cause destruction of neuronal structures. This glutamatergic excitotoxicity may be responsible for the symptoms in some neurodegenerative diseases. The medications currently approved to treat Alzheimer's include the cholinesterase inhibitors donepezil, galantamine, and rivastigmine; a glutamate regulation medication, memantine; and combination donepezil/memantine.

8.1. Common Side Effects

Whether the approach to pharmacological treatment is symptomatic, slowing of disease progression, or neuroprotection, the goal is to stabilize the patient with durable benefit and minimal adverse effects. Most transitory side effects are not life-threatening but need to be anticipated, recognized, discussed, and managed promptly to avoid compromised therapy.

Before treatment, the patient should be tested for psychosis or cognitive changes. Hallucinations and illusions may be part of the disease or related to concomitant medications or illness. The onset of hallucinations usually indicates the need for dose reduction. Some patients attempt to self-taper sine die. These patients remain at risk of side effects but also become increasingly disabled by underdosing. Complementary treatments, including acupuncture and herbal vitamin therapy, are often used, and herbals can interact with several prescribed Parkinson's medications. Monitoring should include early follow-up to assess for side effects and efficacy, with more frequent checks during medication initiation and titration. The aim is to treat to an optimal response and dose while minimizing dose-limiting adverse events (Musey & Shivkumar, 2017).

8.2. Strategies for Management

Contemporary management of Parkinson's and Alzheimer's diseases is primarily pharmacological. Parkinson's disease necessitates chronic administration of dopaminergic drugs; the prevalent therapy is levodopa, a dopamine precursor that traverses the blood-brain barrier, with its prolonged use potentially leading to harmful effects. Alzheimer's disease lacks curative treatment, and currently available drugs yield minimal benefits. Cholinergic therapy aims to alleviate the deficit of acetylcholine secretion using acetylcholinesterase inhibitors (e.g., donepezil, rivastigmine, galantamine) that impede the degradation of acetylcholine in the synaptic cleft. No glutamatergic lowering drug has been approved yet, although some are under investigation (e.g., memantine), which mitigate disease progression by modulating the excitotoxic effect of glutamate.

Addressing these neurological disorders also demands physical and cognitive behavioral therapies, alongside lifestyle changes. Given Parkinson's and Alzheimer's diseases holder



impact on patients' and caregivers' quality of life, treatment should be guided by an ethical framework configured to meet temporal needs, encompassing juridical considerations, informed consent, advance directives, and end-of-life decisions. These diseases pose significant challenges in neurological patients' care and health economics.

9. Future Directions in Treatment

As the understanding of Parkinson's and Alzheimer's diseases deepens, innovative treatments beyond current medications are under development (Thangwaritorn et al., 2024). Personalized medicine is also becoming more prominent, recognizing the variability in patient responses. In preparation for broader application, enhanced assessment tools and therapies, including computerized technologies, computerized technologies might play a valuable interim role (ASAKAWA et al., 2019).

9.1. Emerging Therapies

Standard-of-care pharmaceutical agents for Alzheimer's disease (AD) and Parkinson's disease (PD) cannot modify progression. Hence, novel therapeutic development remains a priority. Emerging treatments for these chronic, debilitating neurodegenerative disorders include monoclonal antibodies targeting toxic protein accumulation and neuroprotective agents aiming to slow neuronal degeneration (Thangwaritorn et al., 2024).

9.2. Personalized Medicine

Personalized medicine for AD aims to improve diagnosis, monitor disease progression, and evaluate treatment efficacy. Precision medicine uses a person-centred assessment by a multidisciplinary team to incorporate information about biological factors, lifestyle, and caregiving environment to allow the patient to maximize independence, choice, and control over their own situation (Svob Strac et al., 2021). Patient stratification is an essential step based on demographic, genetic, clinical, imaging, biomarker, and environmental data. Forward-looking technologies such as induced pluripotent stem cells can recapitulate polygenic AD and help identify relevant genetic and environmental factors, as well as provide a scalable platform to screen large libraries of potential drugs and their combinations. Early intervention opens the window of opportunity to slow or prevent AD when changes may be most modifiable (J. Montine & S. Montine, 2015). Several behavioural and pharmacological approaches, such as physical activity, brain stimulation, diet, and targeted drugs, fit this scenario. For moderate and severe stages of AD, promising therapeutic strategies rely on drugs targeting neural inflammation and to a lesser extent neural regeneration. Future personalised approaches will probably also target the gut microbiota and epigenetic modifications with potential roles in the onset and progression of AD.



10. Role of Non-Pharmacological Interventions

Non-pharmacological interventions may complement medication therapy for Parkinson's and Alzheimer's diseases (Sung Kim, 2017). Some psychostimulation programs combined with pharmacological treatments can help patients maintain cognitive stability; further research is needed to enhance such interventions and develop easily adopted, affordable treatments. Since 2005, clinical guidelines have emphasized nonpharmacological treatments for the behavioral and psychological symptoms of dementia (BPSD) because of concerns about antipsychotic risks and benefits (Azermi, 2015). Options include cognitive training, rehabilitative care, activities of daily living (ADL), music therapy, massage, physical activity, and education or training for professionals and caregivers. Most interventions show promise, but additional research is warranted. Reality orientation has limited evidence for effectiveness, whereas cognitive stimulation shows moderate benefits for cognition, ADL, behavior, and mood. Reminiscence therapy exerts positive effects but lacks sufficient support to recommend its widespread use; validation therapy generally provides no significant benefits. Aromatherapy may reduce agitation, but the supporting evidence is inconsistent. Massage and music therapy lack robust data, and light therapy does not improve cognition, sleep, or behavior in this population. Exercise programs enhance functional ability, physical health, and mood. A Cochrane review concluded that psychosocial interventions can decrease antipsychotic use, although the overall effect size remains unclear.

10.1. Physical Therapy

Parkinson's disease (PD) is a neurodegenerative disorder characterized by progressive loss of dopaminergic neurons in the nigrostriatal pathway (Musey & Shivkumar, 2017). Resulting motor symptoms—including bradykinesia, rigidity, resting tremor, and postural instability—often begin unilaterally and spread contralaterally. Cognitive impairment frequently accompanies the motor deficits. Abnormal dopamine neurotransmission underlying many of these symptoms guides medical therapy. In the absence of curative intervention, effective physical therapy and rehabilitation play important roles throughout the course of the condition. PD frequently involves gait impairments such as difficulties in initiation and termination of walking, shuffling gait, reduced joint mobility, stooped postural alignment, and compromised balance, which elevates the risk of falls and postural instability (Lee et al., 2024). To address these challenges, physical therapy options include electrical modalities (functional electrical stimulation and electrical muscle stimulation), neurorehabilitation techniques (proprioceptive neuromuscular facilitation and Bobath therapy), aerobic and resistance training, treadmill exercise, balance and gait training, and hydrotherapy.



10.2. Cognitive Behavioral Therapy

Cognitive behavioral therapy is a psychological treatment effective for a range of health problems, including neurological disorders such as Parkinson's and Alzheimer's diseases. Numerous clinical trials suggest it is as effective as medication for many of these conditions and that it may be a valuable supplement to pharmacotherapy.

Parkinson's disease is a neurological disorder characterised by midbrain dopamine depletion and nigrostriatal transmission deficits, leading to diminished movement control. It represents a public health challenge, emphasising the need for effective treatments to lessen motor and non-motor symptoms (McNamara & Durso, 2006). Treatments for Parkinson's include motor-specific and adjunct interventions; among these, dopaminergic agents like levodopa remain the primary method for symptom relief.

Alzheimer's disease is a central nervous system disorder and the leading cause of dementia worldwide. It presents an escalating societal concern due to its rising incidence in ageing populations. Cholinesterase inhibitors and membrane stabilisers represent the primary medicinal approaches to treatment (Mathys, 2018).

10.3. Lifestyle Modifications

Treatment of Parkinson's disease (PD) is largely symptomatic, with levodopa remaining the most effective therapy. Adjuvant therapies may include monoamine oxidase B inhibitors, catechol-O-methyltransferase inhibitors, amantadine, and anticholinergics. Advanced brain stimulation therapies, such as deep brain stimulation, are used in patients with motor fluctuations and tremor refractory to medical treatment. In addition to dopaminergic therapies, some benefit may be obtained from physical, speech, and occupational therapy (Nag & A. Jelinek, 2019). Numerous non-pharmacological therapies have been studied in Alzheimer's disease (AD); of these, physical activity and cognitive behavioral therapy have the best evidence of benefit, along with techniques to improve sleep quality.

11. Patient and Caregiver Perspectives

Parkinson's and Alzheimer's diseases are the two most common progressive neurodegenerative disorders resulting in a pervasive decline in a variety of cognitive, behavioural, motor, and functional symptoms (Cristina Muñoz-Contreras et al., 2022). They represent the two most common neurological disorders with an exceptionally high burden of disease in Western society. During the 12-month period preceding this publication, over 140 000 research articles have been published on these two neurodegenerative diseases, with the number of articles steadily increasing over recent years. Approximately 7.5 million people in the United States live with Parkinson's or Alzheimer's disease, and considered in monetary terms, the combined annual cost of treating individuals with Parkinson's and Alzheimer's



disease lies in the hundreds of billions of dollars. Effective treatment of neurological disorders would therefore be of immense benefit to the global population.

Caregiver presence is central in promoting adherence to pharmacological treatment for patients with Alzheimer's disease and other types of dementia. Drug-related problems could be prevented or solved and caregivers' satisfaction with the provided pharmaceutical care is high. Parkinson's disease is a complex and multifaceted condition with heterogeneous motor and nonmotor symptoms, which can present a challenge to effective management. A feedback loop of patient identification, diagnosis, choice of therapy, providing information about the disease, and patient monitoring is required to effectively manage the disease (Hermanowicz et al., 2019). Insufficient communication between the patient and physician can result in suboptimal treatment and affect clinical outcomes.

11.1. Quality of Life Considerations

The ability to perform activities of daily living commonly deteriorates in patients with Parkinson's disease. As the disease progresses, other activities such as eating and contacting the outside world become increasingly difficult, resulting in reduced quality of life (K. Hanna & Cronin-Golomb, 2011). Anxiety is commonly experienced by Parkinson's patients across the disease course, and along with depression contributes to poor quality of life in the disorder (K. Hanna & Cronin-Golomb, 2012). A variety of medications may improve quality of life in Parkinson's and Alzheimer's diseases; however, in specific situations, some are ineffective and somewhat toxic. Understanding the mechanisms of action and benefits of these treatments could yield hope for better quality of life as the diseases progress.

11.2. Support Systems and Resources

Neurological disorders, with symptoms such as seizures, movement problems, muscle weakness, and dementia, affect 1 in 6 people worldwide. In the UK, the economic and social costs associated with neurological disorders are estimated at £4.4 billion per year. Despite many disorders remaining poorly understood, an increasing number can be managed effectively with medication, particularly Parkinson's and Alzheimer's, which account for nearly half of the prescriptions dispensed for neurological disorders each year.

Parkinson's is a neurodegenerative disorder characterized by the progressive loss of nigrostriatal dopaminergic neurons. It is classically associated with four motor features: bradykinesia, rigidity, resting tremor, and postural instability. Typically, symptoms emerge unilaterally and progress to bilateral involvement. Cognitive impairment is common and can be particularly disabling. Many of the motor and non-motor deficits relate to abnormalities in dopamine neurotransmission. Several classes of medications have been developed to manage these deficits, with the effectiveness of any individual agent depending critically on dose and scheduling (Musey & Shivkumar, 2017).



12. Ethical Considerations in Treatment

Ethical challenges permeate the treatment of advanced neurological disorders like Parkinson's and Alzheimer's diseases. Informed consent often becomes problematic because the majority of patients cannot meaningfully participate in decisions about initiating or withdrawing treatment. Decisions frequently remain confined to physician–family–caregiver discussions. Examples include use of feeding tubes and assisted ventilation in late-stage Alzheimer's, where treatment may prolong life without restoring cognitive abilities; studies indicate no significant survival benefit for those undergoing such interventions. Ethical decision-making in these contexts also extends beyond autonomy, encompassing considerations common to other terminal illnesses but complicated by patients' inability to communicate wishes.

12.1. Informed Consent

'Informed consent' is a process recognized in the doctor–patient relationship whereby doctors enable and support decisions about taking medication. Doctors must convey to patients, simply and fully, the available options: recommendations, benefits and potential side effects, alternatives and consequences of refusing. Psychiatric illnesses often involve impaired insight and/or judgment, which complicates informed consent. Consider advice tailored carefully for a patient, relayed through a family member. A doctor cannot simply focus on legalistic questions—"Do you understand? Have I explained enough?"—but must seek genuine comprehension. Part of informed consent is assessing patients' decision-making capacity—their ability to process information, understand the consequences of choices, and communicate decisions. In daily practice, physicians should endeavor to provide comprehensive explanations and support, while appreciating that some individuals may face challenges in understanding or decision-making; the aim is to promote informed and nuanced decision-making.

During consultation, the doctor may assess patients' capacity by analyzing their interaction, including how they respond to questions and whether doubts or difficulties in understanding arise. Upon brief presentation of therapeutic options, one can generally identify those who comprehend and those who do not. In complex or significant cases, formal evaluations by trained specialists may be necessary. Various written-informed-consent forms and information sheets exist for patients and relatives; however, habilitation to provide consent requires personal assessments by the attending doctor, based on a thorough understanding of the patient's condition and circumstances.

12.2. End-of-Life Decisions

Neurologists are frequently called upon to continue the management of complex long-term neurological conditions during the dying phase, providing both symptom control and advice



on the continuation or discontinuation of disease-specific medications. People with neurodegenerative conditions can deteriorate and die unexpectedly, occasionally during sleep. Such potential has important implications for care planning, specifically the completion of anticipatory care directives to avoid unwanted interventions, particularly cardiopulmonary resuscitation. Death due to a more protracted decline is also common, and recognized general indicators are described. Where death is anticipated, the enactment of relevant care plans supports decisions about next steps, guidance that may be reinforced by the neurologist, who can confirm the likely prognosis and advise on medication options (Nair et al., 2022).

National Institute for Health and Care Excellence (NICE) guidelines advocate honest and open communication with patients and their families, to provide reassurance and help all parties understand what to expect. Medications required for the dying phase are usually prescribed preemptively, with subcutaneous injection being the preferred route when oral administration is no longer feasible. Anticipatory medications are aimed at controlling symptoms such as pain, breathlessness, agitation, respiratory secretions, and nausea. In cases of complex regimen or uncertainty, engagement with specialist palliative care services is recommended.

Disease-specific medication needs warrant particular consideration, especially for Parkinson's disease where psychomotor function depends heavily on the regular administration of dopaminergic treatment. When the prognosis is measured in weeks, the use of rotigotine transdermal patches or subcutaneous apomorphine infusion offers a mechanism to maintain therapy; whereas if the prognosis is reduced to days, treatment generally focuses on standard palliative medications including midazolam (Bhansali et al., 2023). At this time, symptoms are most commonly related to respiratory secretions, pain, agitation, and fever.

For epilepsy, midazolam remains the first-line anticonvulsant and can be supplemented with additional drugs administered via subcutaneous infusion. Hydration and nutrition are highly problematic issues, since a diminishing desire to eat or drink is a natural part of the dying process that should be recognized and accepted by those providing care. Routine mouth care and small sips of fluid can nevertheless alleviate dry mouth. For patients receiving artificial feeding, reductions or complete cessation of enteral feeding may be appropriate, with reassurance offered to family and carers that dehydration is not a cause of distress. Spiritual and religious needs should be respected, and appropriate emotional support provided throughout, recognizing the wide range of complex emotional and practical issues that surround the death of a patient with a longstanding neurodegenerative disease.



13. Conclusion

Neurological disorders are conditions of the brain, spine, and associated nerves that disrupt normal functioning of the nervous system. They are classified as acute or chronic, depending on the persistence of symptoms; chronic neurological disorders can cause long-term and permanent damage. Diagnosis typically requires either physical examination or diagnostic tests, such as CT or MRI, but neurological tests or lumbar punctures are only rarely used. Due to their high prevalence and WHO projections of increased future incidence, Parkinson's and Alzheimer's diseases were selected as subjects. Both diseases manifest in aged individuals but differ in mechanisms and symptoms.

Parkinson's disease is caused by low levels of the neurotransmitter dopamine and is characterized by tremors, rigidity, poor posture, muscle weakness, and fatigue. Alzheimer's disease results from the low levels of acetylcholine neurotransmitter in the brain, and its symptoms include memory loss, confusion, mood swing, disorientation, and irritability. Various medications are available to alleviate symptoms, and some can slow the disease rate by addressing deficient neurotransmitters or blocking related receptors. Non-pharmacological therapies—such as physical, occupational, speech, cognitive behavioral, and recreation therapies—and lifestyle modifications are important to help patients retain cognitive and physical functions and maintain self-reliance.

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