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Drug Repositioning for Neurodegenerative Diseases Alzheimers Disease and Parkinsons Disease

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Abstract: *Neurodegenerative diseases are a group of chronic, progressive, and debilitating disorders characterized by the gradual loss of structure and function of neurons in the central nervous system. Among these disorders, Alzheimer's disease (AD) and Parkinson's disease (PD) are the most prevalent and represent a significant global health burden, particularly among the aging population. Alzheimer's disease is primarily associated with progressive memory loss, cognitive decline, and behavioral disturbances, whereas Parkinson's disease is characterized by motor dysfunction, including tremors, rigidity, bradykinesia, and postural instability, along with various non-motor symptoms. Despite extensive research and advances in medical science, currently available therapies provide only symptomatic relief and do not effectively halt or reverse disease progression. Therefore, there is an urgent need for the development of novel therapeutic strategies that can address the underlying pathological mechanisms of these disorders.*

Drug repositioning, also known as drug repurposing, has emerged as a promising and cost-effective approach for the discovery of new therapeutic applications for existing drugs. This strategy involves identifying approved or investigational drugs that can be utilized for indications other than those for which they were originally developed. Compared to conventional drug discovery processes, drug repositioning significantly reduces the time, cost, and risk associated with drug development because the safety, pharmacokinetic, and toxicological profiles of these drugs are already well established. Consequently, drug repositioning has gained considerable attention as a viable approach for accelerating the availability of effective treatments for neurodegenerative diseases.

The present project focuses on the role of drug repositioning in the management of Alzheimer's disease and Parkinson's disease. The study explores the pathophysiology of these disorders, including the accumulation of amyloid-beta plaques, tau protein hyperphosphorylation, neuroinflammation, oxidative stress, mitochondrial dysfunction, protein aggregation, and dopaminergic neuronal degeneration. Understanding these mechanisms provides a foundation for identifying existing drugs capable of targeting multiple pathways involved in neurodegeneration. Various classes of repositioned drugs, including antidiabetic agents, antihypertensive drugs, anti-inflammatory agents, anticancer drugs, antimicrobial agents, and neuroprotective compounds, have demonstrated potential therapeutic benefits in preclinical and clinical studies.

Several repositioned drugs such as metformin, pioglitazone, nilotinib, ambroxol, exenatide, rasagiline, and other pharmacological agents have shown promising results in improving neuronal survival, reducing neuroinflammation, enhancing mitochondrial function, promoting autophagy, and mitigating protein aggregation. These mechanisms may contribute to slowing disease progression and improving the quality of life of affected patients. Advances in computational biology, bioinformatics, artificial intelligence, molecular docking, network pharmacology, and systems biology have further enhanced the identification and validation of potential repositioning candidates, thereby facilitating more efficient drug discovery processes.

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This project also discusses the advantages, challenges, and future perspectives of drug repositioning for neurodegenerative diseases. While drug repurposing offers substantial benefits, including reduced development costs and shorter regulatory pathways, challenges such as limited efficacy, intellectual property concerns, and the need for robust clinical validation remain significant. Nevertheless, continued research and technological advancements are expected to expand the opportunities for identifying effective repositioned drugs for Alzheimer's disease and Parkinson's disease.

In conclusion, drug repositioning represents a highly promising strategy for addressing the unmet therapeutic needs associated with neurodegenerative disorders. By leveraging existing pharmacological agents and modern computational approaches, drug repurposing has the potential to accelerate the development of disease-modifying therapies for Alzheimer's disease and Parkinson's disease. The successful implementation of this approach may significantly improve patient outcomes, reduce healthcare burdens, and contribute to the advancement of neurodegenerative disease management in the future.

Neurodegenerative diseases represent a major challenge to modern healthcare systems due to their increasing prevalence, complex pathogenesis, and limited therapeutic options. Among these disorders, Alzheimer's disease (AD) and Parkinson's disease (PD) are the two most common neurodegenerative conditions affecting millions of people worldwide. These diseases are characterized by progressive neuronal loss, resulting in irreversible impairment of cognitive, behavioral, and motor functions. Alzheimer's disease is primarily associated with memory deficits, impaired learning, cognitive dysfunction, and dementia, whereas Parkinson's disease predominantly affects movement control due to the degeneration of dopaminergic neurons in the substantia nigra. The growing aging population has significantly increased the incidence of these disorders, creating an urgent need for effective and affordable therapeutic interventions.

Traditional drug discovery and development for neurodegenerative diseases is a lengthy, expensive, and high-risk process that often requires more than a decade of research and billions of dollars in investment. Furthermore, many newly developed drug candidates fail during clinical trials due to inadequate efficacy or safety concerns. As a result, researchers have increasingly focused on drug repositioning as an alternative strategy to accelerate the identification of novel treatments for neurodegenerative disorders. Drug repositioning, also known as drug repurposing, involves the investigation of approved, discontinued, or investigational drugs for new therapeutic indications beyond their original clinical use. This approach offers several advantages, including reduced development time, lower research costs, established safety profiles, and increased probability of regulatory approval.

The pathological mechanisms underlying Alzheimer's disease and Parkinson's disease are highly complex and involve multiple interconnected pathways. In Alzheimer's disease, major pathological features include extracellular accumulation of amyloid-beta plaques, intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein, oxidative stress, mitochondrial dysfunction, synaptic degeneration, neuroinflammation, and neuronal death. Similarly, Parkinson's disease is characterized by the progressive loss of dopaminergic neurons, aggregation of alpha-synuclein protein in Lewy bodies, mitochondrial impairment, oxidative damage, neuroinflammatory responses, and disruption of neurotransmitter balance. These overlapping molecular pathways provide numerous opportunities for identifying existing drugs that can target common mechanisms involved in neurodegeneration.

Recent advances in pharmacology, molecular biology, genomics, proteomics, and computational drug discovery have facilitated the identification of several promising repositioned drugs for neurodegenerative diseases. Antidiabetic drugs such as metformin, pioglitazone, and glucagon-like

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peptide-1 (GLP-1) receptor agonists have demonstrated neuroprotective effects by improving insulin signaling, reducing inflammation, and enhancing neuronal survival. Antihypertensive agents, including angiotensin receptor blockers and calcium channel blockers, have shown potential in improving cerebral blood flow and reducing neurodegenerative progression. Anti-inflammatory drugs, antimicrobial agents, anticancer medications, and lipid-lowering drugs have also emerged as potential candidates due to their ability to modulate cellular pathways associated with neuronal damage and protein aggregation.

The application of advanced computational techniques, including artificial intelligence, machine learning, network pharmacology, molecular docking, and bioinformatics analysis, has revolutionized the field of drug repositioning. These technologies enable researchers to identify novel drug-target interactions, predict therapeutic efficacy, and optimize candidate selection with greater precision. Furthermore, systems biology approaches have improved the understanding of disease networks and molecular mechanisms, allowing for the discovery of multitarget therapeutic agents capable of addressing the multifactorial nature of Alzheimer's disease and Parkinson's disease.

Clinical and preclinical studies have identified several repositioned drugs with significant therapeutic potential. Agents such as nilotinib, exenatide, ambroxol, rasagiline, isradipine, and various anti-inflammatory compounds have demonstrated beneficial effects in reducing neurodegeneration, enhancing neuronal function, promoting autophagy, improving mitochondrial health, and decreasing pathological protein accumulation. Although some repositioned drugs have shown promising outcomes in early-stage clinical trials, additional large-scale studies are required to establish their long-term efficacy, safety, and clinical applicability.

The present project comprehensively reviews the concept, principles, methodologies, and therapeutic significance of drug repositioning in the treatment of Alzheimer's disease and Parkinson's disease. It highlights the molecular basis of neurodegeneration, current therapeutic limitations, strategies for identifying repositioned drug candidates, and the role of emerging technologies in accelerating drug discovery. The project also examines the challenges associated with drug repurposing, including intellectual property issues, regulatory barriers, optimization of dosage regimens, and validation through clinical trials.

In conclusion, drug repositioning has emerged as a highly innovative and practical strategy for addressing the unmet medical needs of neurodegenerative diseases. By utilizing existing pharmacological agents with known safety profiles, this approach has the potential to significantly reduce the time and cost required for therapeutic development. Continued advancements in computational biology, translational medicine, and clinical research are expected to expand the repertoire of repositioned drugs available for Alzheimer's disease and Parkinson's disease. The successful implementation of drug repositioning strategies may ultimately lead to the development of effective disease-modifying therapies, improved patient outcomes, enhanced quality of life, and reduced socioeconomic burden associated with neurodegenerative disorders.

Neurodegenerative disorders constitute one of the most significant causes of disability, dependency, and mortality among the elderly population worldwide. These disorders are characterized by the progressive degeneration and eventual loss of neurons in specific regions of the brain and nervous system, leading to irreversible impairment of cognitive, behavioral, sensory, and motor functions. Alzheimer's disease (AD) and Parkinson's disease (PD) account for the majority of neurodegenerative cases and pose substantial medical, social, and economic burdens on patients, caregivers, and healthcare systems. Despite decades of intensive research, the development of effective disease-modifying therapies remains a major challenge due to the multifactorial nature of these disorders and the complexity of the human brain.

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Alzheimer's disease is the leading cause of dementia and is characterized by progressive memory impairment, cognitive decline, language dysfunction, behavioral abnormalities, and loss of independence in daily activities. The disease is associated with pathological hallmarks including extracellular deposition of amyloid-beta plaques, intracellular accumulation of neurofibrillary tangles composed of hyperphosphorylated tau protein, synaptic dysfunction, mitochondrial abnormalities, oxidative stress, and chronic neuroinflammation. Parkinson's disease, the second most common neurodegenerative disorder, is characterized by resting tremors, muscular rigidity, bradykinesia, postural instability, and a variety of non-motor symptoms such as depression, cognitive impairment, sleep disturbances, and autonomic dysfunction. The pathological basis of Parkinson's disease involves degeneration of dopaminergic neurons in the substantia nigra, accumulation of alpha-synuclein aggregates, mitochondrial dysfunction, impaired protein degradation pathways, and inflammatory responses within the central nervous system.

Current pharmacological treatments for Alzheimer's disease and Parkinson's disease primarily focus on symptomatic management rather than addressing the underlying causes of neurodegeneration. Cholinesterase inhibitors and NMDA receptor antagonists used in Alzheimer's disease provide temporary improvement in cognitive symptoms but fail to prevent neuronal loss. Similarly, levodopa and dopamine agonists used in Parkinson's disease effectively alleviate motor symptoms but do not stop disease progression. The limited success of conventional therapeutic approaches has highlighted the urgent need for innovative strategies capable of identifying effective disease-modifying treatments within a shorter timeframe and at a lower cost.

Drug repositioning has emerged as a transformative approach in modern pharmaceutical research aimed at discovering new therapeutic applications for existing drugs. This strategy capitalizes on previously approved, clinically tested, or investigational compounds whose pharmacological properties, pharmacokinetics, toxicity profiles, and safety data are already well documented. By bypassing several early stages of traditional drug development, drug repositioning substantially reduces research expenditure, shortens development timelines, minimizes the risk of clinical failure, and accelerates patient access to potentially beneficial therapies. The increasing availability of biomedical databases, electronic health records, omics technologies, and computational resources has further enhanced the feasibility and success of drug repositioning initiatives.

The rationale for drug repositioning in neurodegenerative diseases is based on the observation that multiple pathological pathways contribute to neuronal degeneration. Processes such as oxidative stress, mitochondrial dysfunction, neuroinflammation, impaired autophagy, protein misfolding, excitotoxicity, insulin resistance, vascular abnormalities, and dysregulated cellular signaling are common to both Alzheimer's disease and Parkinson's disease. Existing drugs developed for metabolic disorders, cardiovascular diseases, infectious diseases, cancer, autoimmune conditions, and psychiatric illnesses have been found to interact with these pathways, making them attractive candidates for repurposing. Consequently, numerous pharmacological agents have been investigated for their ability to protect neurons, enhance synaptic plasticity, improve mitochondrial function, reduce inflammatory responses, and prevent pathological protein aggregation.

Recent studies have demonstrated the neuroprotective potential of several repositioned drugs. Antidiabetic agents such as metformin, pioglitazone, liraglutide, semaglutide, and exenatide have shown promising effects on insulin signaling pathways, glucose metabolism, and neuroinflammation. Anticancer drugs such as nilotinib have been investigated for their capacity to enhance autophagic clearance of toxic protein aggregates. Antihypertensive medications, including angiotensin receptor blockers and calcium channel blockers, have exhibited protective effects through improved cerebral circulation and reduction of oxidative damage. Antimicrobial agents, anti-inflammatory compounds,

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immunomodulators, and lipid-lowering drugs have also demonstrated potential therapeutic benefits by targeting diverse mechanisms involved in neurodegenerative pathology.

The advancement of computational drug repositioning has significantly accelerated the identification of novel therapeutic candidates. Artificial intelligence, machine learning algorithms, network pharmacology, molecular docking simulations, transcriptomic analysis, proteomic profiling, and systems biology approaches are increasingly being utilized to predict drug-disease associations and uncover previously unrecognized therapeutic opportunities. These technologies facilitate the integration of large-scale biological datasets, enabling researchers to identify multitarget drugs capable of simultaneously modulating several pathological pathways involved in neurodegeneration. Such approaches are particularly valuable for complex diseases like Alzheimer's disease and Parkinson's disease, where single-target interventions often fail to produce meaningful clinical outcomes.

This project provides a comprehensive overview of the principles, methodologies, scientific basis, and clinical relevance of drug repositioning for neurodegenerative diseases. It examines the molecular mechanisms responsible for neuronal degeneration, explores the therapeutic potential of repositioned drugs currently under investigation, and discusses the role of emerging technologies in accelerating drug discovery. Furthermore, it highlights the challenges associated with clinical translation, including dosage optimization, blood-brain barrier penetration, patient variability, regulatory considerations, and long-term safety evaluation.

The growing success of drug repositioning demonstrates its potential to bridge the gap between basic neuroscience research and clinical application. By exploiting the therapeutic versatility of existing drugs and integrating advanced computational tools with experimental validation, drug repositioning offers a practical and economically viable pathway toward the development of novel treatments for Alzheimer's disease and Parkinson's disease. Continued interdisciplinary collaboration among neuroscientists, pharmacologists, clinicians, bioinformaticians, and regulatory agencies will be essential for translating these discoveries into effective therapies capable of slowing disease progression, preserving neurological function, and improving the quality of life of millions of patients affected by neurodegenerative disorders worldwide.(1,3,5,8)

Keywords: Neurodegenerative diseases

I. INTRODUCTION

Neurodegenerative diseases are a diverse group of chronic, progressive, and debilitating disorders characterized by the gradual degeneration and irreversible loss of neurons in the central and peripheral nervous systems. These diseases lead to the deterioration of cognitive, motor, sensory, and behavioral functions, significantly affecting the quality of life of affected individuals. As the global population continues to age, neurodegenerative disorders have emerged as a major public health concern due to their increasing prevalence, high healthcare costs, and substantial social and economic impact. Among the various neurodegenerative disorders, Alzheimer's disease (AD) and Parkinson's disease (PD) are the most common and extensively studied conditions, affecting millions of people worldwide.

The human nervous system is composed of highly specialized neurons responsible for transmitting signals that regulate movement, memory, learning, emotions, perception, and various physiological functions. Unlike many other cell types, mature neurons possess limited regenerative capacity. Consequently, neuronal damage or death often results in permanent functional impairment. Neurodegenerative diseases are characterized by the progressive loss of neuronal structure and function, leading to cognitive decline, motor dysfunction, behavioral disturbances, and eventually disability and dependence on caregivers. The exact causes of neurodegeneration remain incompletely understood;

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however, genetic, environmental, metabolic, and age-related factors are known to contribute significantly to disease development and progression.

Alzheimer's disease is the most prevalent cause of dementia and accounts for approximately 60–80% of all dementia cases worldwide. It is a progressive neurological disorder primarily affecting memory, cognition, language, reasoning, and problem-solving abilities. The disease typically begins with mild memory impairment and gradually progresses to severe cognitive dysfunction, personality changes, behavioral abnormalities, and complete dependence on caregivers. Pathologically, Alzheimer's disease is characterized by the accumulation of extracellular amyloid-beta plaques and intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein. These pathological features trigger synaptic dysfunction, neuronal loss, oxidative stress, mitochondrial damage, and chronic neuroinflammation, ultimately leading to widespread brain atrophy.

The burden of Alzheimer's disease is increasing at an alarming rate due to rising life expectancy and population aging. Current therapeutic options, including acetylcholinesterase inhibitors and N-methyl-D-aspartate (NMDA) receptor antagonists, provide only symptomatic relief and do not effectively halt disease progression. Consequently, there is a pressing need to identify novel therapeutic approaches capable of targeting the underlying pathological mechanisms responsible for neurodegeneration.

Parkinson's disease is the second most common neurodegenerative disorder after Alzheimer's disease. It is a chronic and progressive movement disorder primarily caused by the degeneration of dopaminergic neurons in the substantia nigra pars compacta region of the brain. The resulting deficiency of dopamine leads to characteristic motor symptoms, including resting tremor, muscular rigidity, bradykinesia (slowness of movement), and postural instability. In addition to motor manifestations, Parkinson's disease is associated with a wide range of non-motor symptoms such as depression, anxiety, sleep disturbances, cognitive impairment, autonomic dysfunction, and gastrointestinal abnormalities.

The pathological hallmark of Parkinson's disease is the formation of Lewy bodies, intracellular inclusions composed mainly of aggregated alpha-synuclein protein. Emerging evidence suggests that mitochondrial dysfunction, oxidative stress, neuroinflammation, impaired autophagy, lysosomal abnormalities, and protein misfolding play critical roles in disease pathogenesis. Despite significant advances in understanding the molecular mechanisms of Parkinson's disease, currently available treatments such as levodopa, dopamine agonists, monoamine oxidase inhibitors, and deep brain stimulation primarily address symptoms rather than preventing neuronal degeneration.

The complexity of Alzheimer's disease and Parkinson's disease presents significant challenges for traditional drug discovery. Developing a new drug from initial discovery to market approval typically requires 10–15 years of research and development and involves substantial financial investment. Furthermore, the success rate of candidate drugs in neurodegenerative disease clinical trials remains extremely low. Many investigational compounds fail due to inadequate efficacy, toxicity concerns, poor blood-brain barrier penetration, or inability to demonstrate meaningful clinical benefits. These challenges have encouraged researchers to explore alternative strategies for identifying effective therapies more rapidly and efficiently.

One such innovative strategy is drug repositioning, also referred to as drug repurposing, drug rediscovery, or therapeutic switching. Drug repositioning involves identifying new therapeutic indications for existing drugs that have already been approved for other diseases or have undergone extensive clinical evaluation. Rather than developing entirely new chemical entities, drug repositioning seeks to leverage existing pharmacological knowledge, safety profiles, pharmacokinetic data, and clinical experience to accelerate therapeutic development. This approach has gained considerable attention in recent years as a practical solution to overcome the limitations associated with conventional drug discovery.

The concept of drug repositioning is not new. Several successful examples exist in pharmaceutical history where drugs originally developed for one indication were later found to possess therapeutic benefits for entirely different diseases. The success of these repositioned drugs has demonstrated the potential of this strategy to shorten development timelines, reduce costs, minimize risks, and improve the probability of regulatory approval. Since the safety and

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toxicity profiles of repositioned drugs are already established, researchers can often bypass early stages of drug development and focus directly on evaluating efficacy for the new indication.

Drug repositioning is particularly attractive for neurodegenerative diseases because many pathological mechanisms involved in Alzheimer's disease and Parkinson's disease overlap with biological pathways implicated in other medical conditions. Processes such as oxidative stress, chronic inflammation, mitochondrial dysfunction, impaired autophagy, insulin resistance, vascular abnormalities, protein aggregation, apoptosis, and immune dysregulation are common to multiple diseases. Therefore, drugs originally developed for diabetes, hypertension, cancer, autoimmune disorders, infectious diseases, and metabolic syndromes may possess therapeutic properties capable of modifying neurodegenerative processes.

Recent advances in neuroscience, molecular biology, pharmacology, genomics, proteomics, transcriptomics, metabolomics, and bioinformatics have significantly enhanced the identification of potential drug repositioning candidates. High-throughput screening technologies allow researchers to evaluate thousands of compounds rapidly, while computational approaches facilitate the prediction of novel drug-target interactions. Artificial intelligence, machine learning, network pharmacology, systems biology, and molecular docking studies have emerged as powerful tools for discovering previously unrecognized therapeutic opportunities and accelerating the repositioning process.

Among the various classes of repositioned drugs investigated for neurodegenerative diseases, antidiabetic agents have attracted considerable interest. Increasing evidence suggests that insulin resistance and impaired glucose metabolism contribute to neurodegeneration. Drugs such as metformin, pioglitazone, liraglutide, semaglutide, and exenatide have demonstrated neuroprotective effects through mechanisms involving improved insulin signaling, reduced oxidative stress, enhanced mitochondrial function, and suppression of inflammatory pathways. Similarly, antihypertensive drugs, anti-inflammatory agents, statins, anticancer drugs, and antimicrobial compounds have shown promising results in experimental and clinical studies.

Another important aspect of drug repositioning is its potential to identify multitarget therapeutic agents. Neurodegenerative diseases are multifactorial disorders involving numerous interconnected molecular pathways. Traditional single-target therapies often fail to provide significant clinical benefits because they address only one aspect of disease pathology. In contrast, repositioned drugs frequently exhibit pleiotropic effects, enabling them to modulate multiple pathological mechanisms simultaneously. Such multitarget approaches may offer greater therapeutic efficacy and improve clinical outcomes in patients with Alzheimer's disease and Parkinson's disease.

The blood-brain barrier (BBB) represents a major obstacle in the treatment of neurological disorders. Many therapeutic agents are unable to reach effective concentrations within the brain due to restricted permeability across the BBB. Drug repositioning provides an opportunity to identify existing drugs that already possess favorable pharmacokinetic properties and adequate brain penetration. This advantage can significantly improve the likelihood of successful therapeutic intervention in neurodegenerative diseases.

Despite its considerable promise, drug repositioning also faces several challenges. Differences in dosage requirements, disease-specific pharmacodynamics, intellectual property considerations, regulatory hurdles, and the need for large-scale clinical validation can complicate the development process. Additionally, positive findings in preclinical studies do not always translate into clinical success. Therefore, rigorous experimental evaluation and well-designed clinical trials remain essential for confirming the safety and efficacy of repositioned drugs in neurodegenerative disorders.

The growing integration of artificial intelligence, precision medicine, biomarker-based diagnostics, and personalized therapeutic approaches is expected to further expand the potential of drug repositioning in the coming years. By combining advanced computational methods with clinical and experimental research, scientists can identify more effective treatment strategies tailored to individual patient characteristics and disease mechanisms.

Therefore, drug repositioning has emerged as a highly promising, cost-effective, and time-efficient strategy for the development of novel therapies for Alzheimer's disease and Parkinson's disease. By utilizing existing drugs with established safety profiles and exploring their potential neuroprotective properties, this approach offers new opportunities for addressing the unmet medical needs associated with neurodegenerative disorders. Continued research

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in this field may contribute significantly to the discovery of disease-modifying treatments capable of slowing neurodegeneration, preserving neurological function, improving patient outcomes, and reducing the global burden of Alzheimer's disease and Parkinson's disease.

The twenty-first century has witnessed remarkable advances in medical science, biotechnology, and pharmaceutical research; however, neurodegenerative diseases continue to represent one of the greatest unresolved challenges in modern medicine. These disorders are characterized by the progressive deterioration of neuronal cells, resulting in the gradual loss of brain function and ultimately leading to severe disability and death. Unlike many acute illnesses that can be cured or effectively managed, neurodegenerative diseases are chronic, progressive, and largely irreversible. The increasing life expectancy of the global population has contributed significantly to the rising prevalence of these disorders, making them a major cause of morbidity, mortality, and healthcare expenditure worldwide.

Neurodegeneration refers to the gradual destruction of neurons, the specialized cells responsible for transmitting information throughout the nervous system. Neurons play a critical role in cognition, memory, movement, sensation, emotional regulation, and autonomic control. Since mature neurons possess limited regenerative capacity, neuronal damage often leads to permanent functional deficits. The degeneration of specific neuronal populations gives rise to distinct clinical manifestations depending on the affected region of the brain. As neurodegeneration progresses, affected individuals experience worsening symptoms that interfere with daily activities, social interactions, occupational performance, and overall quality of life.

Among the various neurodegenerative disorders, Alzheimer's disease (AD) and Parkinson's disease (PD) represent the most prevalent and extensively researched conditions. Together, these diseases affect tens of millions of individuals globally and impose substantial emotional, social, and economic burdens on patients, families, caregivers, and healthcare systems. The increasing incidence of these diseases is closely associated with population aging, making neurodegenerative disorders one of the most important public health concerns of the present century.

Alzheimer's disease is a progressive neurodegenerative disorder that primarily affects cognitive functions. It is the leading cause of dementia worldwide and is characterized by progressive memory impairment, decline in executive function, language disturbances, impaired judgment, personality changes, and loss of independent functioning. The disease usually begins insidiously with mild forgetfulness but gradually advances to severe cognitive deterioration and complete dependence on caregivers. Histopathological examination of the Alzheimer's brain reveals characteristic abnormalities, including extracellular amyloid-beta plaques and intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein. These pathological structures disrupt neuronal communication, induce oxidative stress, impair mitochondrial function, activate inflammatory pathways, and ultimately result in neuronal death and brain atrophy.

The burden of Alzheimer's disease extends beyond medical complications. Patients frequently require long-term care, institutionalization, and continuous support from family members and healthcare professionals. The psychological stress experienced by caregivers often contributes to depression, anxiety, and financial hardship. As global populations continue to age, the prevalence of Alzheimer's disease is expected to increase dramatically, emphasizing the urgent need for innovative therapeutic interventions capable of delaying, preventing, or reversing disease progression.

Parkinson's disease is another major neurodegenerative disorder characterized primarily by movement abnormalities resulting from degeneration of dopaminergic neurons in the substantia nigra region of the midbrain. Dopamine is an essential neurotransmitter involved in regulating voluntary movement, motivation, reward processing, and various cognitive functions. The progressive depletion of dopamine leads to classical motor symptoms such as resting tremor, muscular rigidity, bradykinesia, postural instability, gait disturbances, and impaired coordination. In addition to motor dysfunction, Parkinson's disease is increasingly recognized as a multisystem disorder involving non-motor manifestations such as cognitive decline, depression, anxiety, sleep disorders, autonomic dysfunction, sensory abnormalities, and gastrointestinal disturbances.

A defining pathological feature of Parkinson's disease is the presence of Lewy bodies, intracellular inclusions primarily composed of misfolded alpha-synuclein protein. The accumulation of these protein aggregates contributes to neuronal

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dysfunction, impaired cellular homeostasis, mitochondrial abnormalities, oxidative damage, neuroinflammation, and activation of apoptotic pathways. Emerging evidence suggests that pathological alpha-synuclein may spread between neurons in a prion-like manner, contributing to disease progression throughout the nervous system. This complex pathophysiology presents significant challenges for the development of effective disease-modifying therapies.

The underlying mechanisms responsible for neurodegeneration are multifactorial and involve intricate interactions among genetic, environmental, metabolic, and cellular factors. Oxidative stress plays a central role in neuronal injury through the excessive production of reactive oxygen species (ROS), which damage proteins, lipids, and nucleic acids. Mitochondrial dysfunction further exacerbates neuronal vulnerability by impairing energy production and increasing oxidative damage. Chronic neuroinflammation mediated by activated microglia and astrocytes contributes to neuronal degeneration through the release of pro-inflammatory cytokines and neurotoxic mediators. Additionally, abnormal protein aggregation, impaired autophagy, disrupted proteasomal degradation, calcium dysregulation, excitotoxicity, vascular dysfunction, and metabolic abnormalities collectively contribute to disease pathogenesis.

One of the major obstacles in neurodegenerative disease treatment is the complexity of the central nervous system. The human brain contains approximately 86 billion neurons interconnected through trillions of synaptic connections. This intricate network regulates countless physiological and cognitive processes. Therapeutic interventions must therefore target highly specific molecular pathways while minimizing adverse effects on normal neurological function. Furthermore, the blood-brain barrier serves as a highly selective protective interface that restricts the entry of many potentially beneficial drugs into the brain. Consequently, numerous therapeutic candidates fail to achieve adequate concentrations within target tissues, limiting their clinical effectiveness.

Traditional drug discovery has historically been the primary approach for developing treatments for neurodegenerative diseases. However, this process is associated with substantial limitations. The development of a novel drug typically requires extensive preclinical research, multiple phases of clinical trials, and regulatory approval processes that may span more than a decade. The overall cost of bringing a new drug to market often exceeds billions of dollars. Moreover, neurodegenerative diseases have one of the highest drug failure rates in pharmaceutical research, with many promising compounds demonstrating limited efficacy during clinical evaluation. These challenges have created an urgent demand for alternative strategies capable of accelerating therapeutic development while reducing financial risk.

Drug repositioning has emerged as a highly attractive solution to these challenges. Drug repositioning refers to the identification and development of new therapeutic applications for existing drugs that have already been approved for clinical use or have undergone significant stages of development. Unlike conventional drug discovery, which begins with entirely new chemical entities, drug repositioning leverages existing knowledge regarding drug safety, pharmacokinetics, pharmacodynamics, formulation characteristics, and manufacturing processes. This significantly reduces development costs, shortens timelines, and increases the likelihood of successful clinical translation.

The scientific rationale behind drug repositioning for neurodegenerative diseases is supported by the observation that many pathological pathways involved in Alzheimer's disease and Parkinson's disease are also implicated in other medical conditions. For example, chronic inflammation, oxidative stress, mitochondrial dysfunction, insulin resistance, immune dysregulation, and abnormal cellular signaling contribute to numerous disorders including diabetes, cardiovascular diseases, autoimmune diseases, metabolic syndromes, and cancer. Consequently, drugs originally developed to treat these conditions may possess previously unrecognized neuroprotective properties capable of modifying disease progression in neurodegenerative disorders.

Recent years have witnessed remarkable progress in identifying repositioned drugs with potential therapeutic benefits for Alzheimer's disease and Parkinson's disease. Antidiabetic agents such as metformin, pioglitazone, exenatide, liraglutide, and semaglutide have attracted significant attention due to their ability to improve insulin signaling, reduce inflammation, enhance mitochondrial function, and promote neuronal survival. Epidemiological studies have suggested a strong association between insulin resistance and neurodegeneration, leading some researchers to describe Alzheimer's disease as "Type 3 Diabetes." These findings have stimulated extensive investigation into the neuroprotective potential of antidiabetic medications.

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Similarly, antihypertensive drugs have demonstrated potential benefits through mechanisms involving improved cerebral perfusion, reduced oxidative stress, and modulation of inflammatory pathways. Statins, originally developed for hypercholesterolemia, have been investigated for their anti-inflammatory and vascular protective effects. Certain anticancer drugs have shown promise in promoting the clearance of toxic protein aggregates through activation of autophagic pathways. Antimicrobial agents, immunomodulators, and anti-inflammatory compounds have also emerged as potential candidates for repositioning due to their ability to target specific mechanisms involved in neurodegeneration.

The rapid advancement of computational biology has transformed the field of drug repositioning. Modern technologies such as artificial intelligence, machine learning, big data analytics, network pharmacology, molecular docking, transcriptomics, proteomics, metabolomics, and systems biology enable researchers to analyze vast amounts of biological information and identify novel drug-disease relationships. These approaches facilitate the prediction of therapeutic targets, drug interactions, and disease pathways with unprecedented accuracy. Computational drug repositioning significantly reduces the time required for candidate identification and enhances the efficiency of experimental validation.

Network pharmacology has emerged as a particularly valuable tool for studying complex diseases such as Alzheimer's disease and Parkinson's disease. Rather than focusing on single molecular targets, network pharmacology examines interactions among multiple genes, proteins, signaling pathways, and pharmacological agents. This systems-level perspective is especially relevant for neurodegenerative diseases, which involve numerous interconnected pathological processes. Repositioned drugs capable of modulating multiple targets simultaneously may offer greater therapeutic efficacy than conventional single-target approaches.

Another important advantage of drug repositioning is the potential to support personalized medicine. Genetic variability among patients influences disease susceptibility, progression, and therapeutic response. Advances in genomics and biomarker discovery enable researchers to identify patient-specific molecular profiles that may predict responsiveness to particular repositioned drugs. This personalized approach may improve treatment outcomes and minimize adverse effects by tailoring therapy to individual biological characteristics.

Despite its numerous advantages, drug repositioning is not without challenges. Clinical efficacy must still be rigorously demonstrated through well-designed clinical trials. Differences in dosing regimens, treatment duration, target populations, and disease-specific pharmacology require careful investigation. Regulatory considerations, intellectual property issues, and commercial incentives may also influence the development and adoption of repositioned therapies. Furthermore, successful translation from laboratory findings to clinical practice requires collaboration among researchers, clinicians, pharmaceutical industries, and regulatory authorities.

The future of drug repositioning in neurodegenerative diseases appears highly promising. Continued integration of artificial intelligence, precision medicine, biomarker-guided therapy, advanced imaging technologies, and systems pharmacology is expected to accelerate the discovery of novel therapeutic opportunities. As scientific understanding of neurodegenerative mechanisms continues to expand, drug repositioning may play a crucial role in bridging the gap between laboratory research and clinical application.

Therefore, drug repositioning represents a revolutionary and practical approach for addressing the urgent need for effective treatments for Alzheimer's disease and Parkinson's disease. By utilizing existing drugs with established safety profiles and exploring their potential neuroprotective effects, this strategy offers a faster, more economical, and scientifically robust pathway for therapeutic innovation. The successful implementation of drug repositioning approaches has the potential to transform the management of neurodegenerative diseases, improve patient outcomes, reduce healthcare burdens, and contribute significantly to the advancement of neurological medicine in the coming decades.(7,8,11,12,15)

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II. NEED OF STUDY

• Existing treatment options mainly provide symptomatic relief and do not effectively stop or reverse disease progression.
• There is a significant unmet medical need for disease-modifying therapies that can target the underlying causes of neurodegeneration.
• Traditional drug discovery is a lengthy, expensive, and high-risk process that requires extensive research and development.
• The high failure rate of new drug candidates in clinical trials necessitates alternative approaches for therapeutic development.
• Drug repositioning offers a cost-effective and time-saving strategy by utilizing drugs with already established safety and pharmacokinetic profiles.
• Repositioned drugs can potentially reach patients faster because much of the preclinical and clinical safety data is already available.
• Alzheimer's disease and Parkinson's disease share several common pathological mechanisms, including oxidative stress, neuroinflammation, mitochondrial dysfunction, and protein aggregation, making them suitable targets for drug repurposing approaches.
• Several existing drugs used for diabetes, hypertension, cancer, and inflammatory disorders have shown promising neuroprotective effects in experimental studies.
• The identification of new therapeutic uses for approved drugs may reduce the financial burden associated with pharmaceutical research and development.
• Drug repositioning can accelerate the availability of effective treatments for patients suffering from neurodegenerative disorders.
• Understanding the molecular mechanisms involved in neurodegeneration may help identify novel therapeutic targets and repositioning opportunities.
• Advances in artificial intelligence, bioinformatics, molecular docking, and network pharmacology have enhanced the ability to discover new drug-disease relationships.
• Repositioned drugs may target multiple pathological pathways simultaneously, providing greater therapeutic benefits than conventional single-target therapies.
• The increasing prevalence of dementia and movement disorders creates a pressing need for innovative and sustainable treatment strategies.
• Neurodegenerative diseases impose a substantial socioeconomic burden on patients, caregivers, healthcare systems, and society.
• Early intervention using effective repositioned drugs may improve patient quality of life and delay disease progression.
• Drug repurposing can contribute to personalized medicine approaches by identifying therapies suitable for specific patient populations.
• Investigating drug repositioning may provide valuable insights into disease mechanisms and therapeutic pathways.
• The study helps bridge the gap between basic neuroscience research and clinical application.
• Exploring repositioned drugs may lead to the discovery of safer and more effective therapeutic options compared to newly synthesized compounds.
• The approach supports sustainable pharmaceutical development by maximizing the therapeutic value of existing medications.
• Drug repositioning has the potential to improve treatment accessibility, especially in resource-limited healthcare settings.

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• The study contributes to current scientific efforts aimed at reducing the global burden of Alzheimer's disease and Parkinson's disease.
• Research on drug repositioning may pave the way for future innovations in the treatment of neurodegenerative disorders and improve long-term patient outcomes.

III. AIM

The aim of this project is to comprehensively evaluate the potential of drug repositioning as an innovative and cost-effective therapeutic strategy for the treatment and management of neurodegenerative diseases, particularly Alzheimer's disease and Parkinson's disease. The study aims to explore the underlying pathological mechanisms involved in these disorders and identify existing drugs with established safety profiles that may be repurposed to target key pathways associated with neurodegeneration. Furthermore, the project seeks to examine the role of repositioned drugs in reducing neuronal damage, improving neurological function, slowing disease progression, and enhancing the quality of life of affected patients. The study also aims to highlight recent advancements, challenges, and future prospects of drug repositioning in accelerating the development of effective disease-modifying therapies for Alzheimer's disease and Parkinson's disease.

IV. OBJECTIVES

1. To study the concept and significance of drug repositioning in modern pharmaceutical research.
2. To understand the epidemiology, etiology, and pathophysiology of Alzheimer's disease and Parkinson's disease.
3. To examine the molecular and cellular mechanisms involved in neurodegeneration.
4. To investigate the limitations of currently available therapies for Alzheimer's disease and Parkinson's disease.
5. To identify existing drugs that have potential applications in the treatment of neurodegenerative disorders.
6. To evaluate the role of drug repositioning as a cost-effective and time-efficient alternative to traditional drug discovery.
7. To study the neuroprotective effects of repositioned drugs on neuronal survival and function.
8. To analyze the mechanisms by which repositioned drugs reduce oxidative stress and neuronal damage.
9. To assess the role of repositioned drugs in modulating neuroinflammatory pathways associated with neurodegeneration.
10. To investigate the effects of repurposed drugs on mitochondrial function and cellular energy metabolism.
11. To evaluate the ability of repositioned drugs to prevent protein aggregation and enhance protein clearance mechanisms.
12. To study the therapeutic potential of antidiabetic drugs in Alzheimer's disease and Parkinson's disease.
13. To examine the neuroprotective role of antihypertensive drugs in neurodegenerative disorders.
14. To evaluate the contribution of anti-inflammatory agents in reducing disease progression.
15. To investigate the potential of anticancer drugs as therapeutic candidates for neurodegenerative diseases.
16. To study the role of antimicrobial and immunomodulatory agents in neuroprotection.
17. To analyze the application of artificial intelligence and machine learning in drug repositioning research.
18. To explore the role of bioinformatics, molecular docking, and network pharmacology in identifying novel drug candidates.
19. To assess the advantages and challenges associated with drug repositioning strategies.
20. To review preclinical and clinical studies related to repurposed drugs for Alzheimer's disease and Parkinson's disease.
21. To examine the regulatory and developmental aspects involved in drug repurposing.
22. To evaluate the impact of repositioned drugs on disease progression and patient quality of life.

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23. To identify future opportunities and emerging trends in drug repositioning for neurodegenerative disorders.
24. To provide a comprehensive overview of current advancements in the field of drug repurposing.
25. To contribute to the development of safer, more effective, and affordable therapeutic approaches for Alzheimer's disease and Parkinson's disease.

V. REVIEW OF LITERATURE

Neurodegenerative diseases are among the most challenging disorders affecting modern healthcare systems due to their progressive nature, complex pathophysiology, and lack of curative treatments. Alzheimer's disease (AD) and Parkinson's disease (PD) account for the majority of neurodegenerative disorders worldwide and are associated with substantial morbidity, mortality, and socioeconomic burden. Over the past several decades, extensive research has been conducted to understand the molecular mechanisms underlying these diseases and to identify effective therapeutic interventions. Despite significant advances in neuroscience and pharmacology, currently available treatments primarily provide symptomatic relief and fail to halt or reverse disease progression. Consequently, researchers have increasingly focused on drug repositioning as a promising strategy for accelerating the development of effective therapies for neurodegenerative disorders.

The concept of drug repositioning has gained considerable attention due to the high failure rate and enormous costs associated with conventional drug discovery. Traditional drug development often requires more than 10–15 years and billions of dollars before a new therapeutic agent reaches the market. In contrast, drug repositioning utilizes approved or clinically tested drugs with known safety and pharmacokinetic profiles, thereby reducing development time, financial investment, and regulatory risks. This approach has emerged as a particularly attractive option for neurodegenerative diseases, where the urgent need for effective treatments is accompanied by limited success in developing novel compounds.

One of the earliest observations supporting drug repositioning in neurodegenerative diseases was the identification of common pathological pathways shared among diverse disorders. Research has demonstrated that oxidative stress, chronic inflammation, mitochondrial dysfunction, protein aggregation, impaired autophagy, and apoptosis contribute significantly to both Alzheimer's disease and Parkinson's disease. These mechanisms are also involved in metabolic, cardiovascular, autoimmune, and oncological disorders, suggesting that drugs developed for other diseases may possess neuroprotective properties. Consequently, numerous existing medications have been investigated for their potential role in preventing or slowing neurodegeneration.

Studies on Alzheimer's disease have revealed that the accumulation of amyloid-beta peptides and hyperphosphorylated tau proteins plays a central role in disease pathology. Amyloid-beta aggregation results in the formation of extracellular plaques that disrupt neuronal communication and trigger inflammatory responses. Similarly, tau protein abnormalities lead to the formation of neurofibrillary tangles, impairing intracellular transport and neuronal survival. In addition to these hallmark features, increasing evidence highlights the contribution of oxidative stress, mitochondrial dysfunction, cerebrovascular abnormalities, and immune dysregulation in disease progression. These findings have expanded the range of potential therapeutic targets beyond amyloid and tau pathology.

Several epidemiological studies have demonstrated a close relationship between metabolic disorders and Alzheimer's disease. Insulin resistance and impaired glucose metabolism have been implicated in neuronal dysfunction and cognitive decline. This observation led researchers to investigate antidiabetic drugs as potential therapeutic candidates for Alzheimer's disease. Metformin, a widely prescribed antidiabetic medication, has shown neuroprotective effects in experimental models through activation of AMP-activated protein kinase (AMPK), enhancement of mitochondrial function, reduction of oxidative stress, and promotion of neuronal survival. Preclinical studies have suggested that metformin may improve cognitive function and reduce amyloid-beta accumulation, although clinical findings remain variable.

Pioglitazone, a peroxisome proliferator-activated receptor gamma (PPAR-γ) agonist used in type 2 diabetes mellitus, has also attracted significant attention in Alzheimer's disease research. Experimental studies have demonstrated its

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ability to suppress neuroinflammation, improve insulin sensitivity, reduce oxidative stress, and enhance neuronal protection. Some clinical investigations have reported improvements in cognitive performance, although larger trials have produced mixed results. Nevertheless, the anti-inflammatory and neuroprotective properties of pioglitazone continue to support its potential role as a repositioned therapeutic agent.

The discovery of glucagon-like peptide-1 (GLP-1) receptor agonists as potential neuroprotective agents represents another important milestone in drug repositioning research. Drugs such as exenatide, liraglutide, and semaglutide were initially developed for the treatment of diabetes mellitus and obesity. However, subsequent studies revealed that GLP-1 receptors are expressed in various regions of the brain involved in learning, memory, and motor control. Activation of these receptors promotes neuronal survival, enhances synaptic plasticity, reduces inflammation, and protects against oxidative damage. Preclinical investigations have demonstrated significant improvements in cognitive function and reductions in pathological protein accumulation following GLP-1 receptor agonist administration.

In Parkinson's disease research, substantial attention has focused on therapies capable of protecting dopaminergic neurons from degeneration. The pathological hallmark of Parkinson's disease is the accumulation of alpha-synuclein aggregates within Lewy bodies, accompanied by progressive loss of dopamine-producing neurons in the substantia nigra. Mitochondrial dysfunction and oxidative stress have been recognized as major contributors to neuronal death. Consequently, several repositioned drugs targeting these mechanisms have been investigated extensively.

Exenatide has emerged as one of the most promising repositioned drugs for Parkinson's disease. Clinical studies have reported improvements in motor function and slower disease progression among patients receiving exenatide therapy. Researchers believe that these benefits result from enhanced mitochondrial activity, reduced neuroinflammation, improved insulin signaling, and increased neuronal survival. Long-term follow-up studies have further strengthened interest in GLP-1 receptor agonists as disease-modifying agents for Parkinson's disease.

Another notable repositioned drug is nilotinib, a tyrosine kinase inhibitor originally approved for the treatment of chronic myeloid leukemia. Research has demonstrated that nilotinib can enhance autophagy and facilitate the clearance of toxic protein aggregates, including alpha-synuclein and amyloid-beta. Experimental studies have shown reductions in neurodegenerative pathology and improvements in neuronal function following nilotinib treatment. Early clinical investigations have suggested potential benefits in both Alzheimer's disease and Parkinson's disease, although further large-scale trials are necessary to confirm efficacy and safety.

Ambroxol, commonly used as a mucolytic agent for respiratory disorders, has recently gained attention as a potential treatment for Parkinson's disease. Studies have demonstrated that ambroxol increases the activity of glucocerebrosidase, a lysosomal enzyme involved in cellular waste clearance. Mutations affecting glucocerebrosidase function are strongly associated with Parkinson's disease risk. By enhancing lysosomal function and promoting the degradation of alpha-synuclein aggregates, ambroxol may help reduce neurodegenerative processes and improve neuronal survival.

Inflammation has emerged as a critical factor in the progression of neurodegenerative diseases. Activated microglia and astrocytes release pro-inflammatory cytokines, chemokines, and reactive oxygen species that contribute to neuronal damage. Consequently, anti-inflammatory drugs have been investigated as potential repositioning candidates. Nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and various immunomodulatory agents have demonstrated neuroprotective effects in experimental models. Although clinical outcomes have been inconsistent, ongoing research continues to explore strategies for targeting neuroinflammation more effectively.

Antihypertensive medications have also been examined for their potential neuroprotective properties. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers may improve cerebral blood flow, reduce oxidative stress, and attenuate inflammatory responses. Several observational studies have suggested lower risks of cognitive decline and dementia among individuals receiving these medications. Similarly, calcium channel blockers such as isradipine have demonstrated protective effects on dopaminergic neurons in experimental Parkinson's disease models.

The relationship between cholesterol metabolism and neurodegeneration has prompted investigation of statins as potential therapeutic agents. Statins possess anti-inflammatory, antioxidant, and vascular protective properties beyond

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their cholesterol-lowering effects. Experimental studies have demonstrated reductions in neuroinflammation, improved endothelial function, and decreased oxidative damage following statin administration. Although clinical evidence remains inconclusive, statins continue to be evaluated for their role in preventing cognitive decline and neurodegeneration.

Advancements in molecular biology and bioinformatics have significantly enhanced drug repositioning efforts. High-throughput screening technologies allow researchers to evaluate thousands of compounds against disease-specific targets. Genomic and proteomic analyses facilitate the identification of molecular pathways involved in disease progression, while transcriptomic studies reveal changes in gene expression associated with neurodegeneration. These technologies provide valuable insights into drug-target interactions and support the discovery of novel repositioning opportunities.

Artificial intelligence and machine learning have revolutionized modern drug discovery and repositioning. These technologies enable the analysis of large-scale biomedical datasets, including genomic information, electronic health records, clinical trial data, and scientific literature. Machine learning algorithms can identify hidden relationships between drugs and diseases, predict therapeutic efficacy, and prioritize candidate compounds for further investigation. Such computational approaches have significantly accelerated the identification of promising repositioned drugs for Alzheimer's disease and Parkinson's disease.

Network pharmacology has emerged as another important tool in neurodegenerative disease research. Unlike traditional pharmacological approaches that focus on single targets, network pharmacology examines complex interactions among genes, proteins, signaling pathways, and pharmacological agents. Since neurodegenerative diseases involve multiple interconnected pathological processes, network-based approaches provide a more comprehensive understanding of disease mechanisms and therapeutic opportunities. Repositioned drugs capable of modulating multiple targets simultaneously may offer superior therapeutic benefits compared to conventional single-target therapies.

Recent literature also emphasizes the importance of personalized medicine in neurodegenerative disease treatment. Genetic variations influence disease susceptibility, progression, and treatment response. Biomarkers such as amyloid-beta levels, tau protein concentrations, alpha-synuclein accumulation, neurofilament light chain, and inflammatory markers are increasingly used to monitor disease progression and evaluate therapeutic efficacy. The integration of biomarker-based approaches with drug repositioning may facilitate the development of personalized treatment strategies tailored to individual patient characteristics.

Overall, the literature strongly supports drug repositioning as a promising strategy for addressing the therapeutic challenges associated with Alzheimer's disease and Parkinson's disease. Numerous existing drugs have demonstrated neuroprotective properties through mechanisms involving reduction of oxidative stress, suppression of neuroinflammation, enhancement of mitochondrial function, promotion of autophagy, and prevention of pathological protein aggregation. Continued advances in computational biology, systems pharmacology, artificial intelligence, and clinical research are expected to further accelerate the discovery and development of repositioned therapies. These efforts hold significant promise for improving patient outcomes, reducing healthcare burdens, and advancing the treatment of neurodegenerative disorders in the future.

The growing interest in drug repositioning for neurodegenerative diseases is largely driven by the increasing understanding that Alzheimer's disease and Parkinson's disease are multifactorial disorders involving numerous interconnected pathological pathways. Earlier therapeutic strategies focused primarily on single targets, such as amyloid-beta plaques in Alzheimer's disease or dopamine replacement in Parkinson's disease. However, these approaches have demonstrated limited success in preventing disease progression. Recent literature suggests that effective treatment requires simultaneous modulation of multiple disease mechanisms, including neuroinflammation, oxidative stress, mitochondrial dysfunction, protein misfolding, synaptic impairment, and neuronal apoptosis. This realization has significantly strengthened the rationale for drug repositioning as a therapeutic strategy.

One important area of research involves the role of mitochondrial dysfunction in neurodegeneration. Mitochondria are responsible for cellular energy production through oxidative phosphorylation and are essential for neuronal survival

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due to the high energy demands of the brain. Studies have demonstrated that mitochondrial abnormalities occur early in both Alzheimer's disease and Parkinson's disease. Defective mitochondria lead to reduced ATP production, excessive generation of reactive oxygen species, calcium dysregulation, and activation of apoptotic pathways. Researchers have therefore investigated drugs capable of improving mitochondrial function as potential neuroprotective agents.

Coenzyme Q10, originally used as a dietary supplement for cardiovascular and mitochondrial disorders, has shown neuroprotective properties in experimental models. It functions as an antioxidant and supports mitochondrial electron transport chain activity. Several studies reported reduced oxidative damage and improved neuronal survival following Coenzyme Q10 administration. Although clinical outcomes have been mixed, the research contributed significantly to understanding mitochondrial-targeted therapies in neurodegenerative diseases.

Creatine has also been extensively investigated for its ability to support cellular energy metabolism. Experimental studies demonstrated that creatine supplementation improves mitochondrial function, stabilizes cellular energy reserves, and protects neurons against oxidative damage. Although large clinical trials have not consistently demonstrated significant benefits, these studies highlighted the importance of energy metabolism as a therapeutic target in neurodegeneration.

Another major focus of contemporary research is the role of neuroinflammation in disease progression. Traditionally, inflammation was considered a secondary consequence of neuronal damage. However, recent findings indicate that chronic activation of microglia and astrocytes actively contributes to neurodegeneration. Activated glial cells release inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1β, and interleukin-6, which promote neuronal injury and accelerate disease progression.

Minocycline, a tetracycline antibiotic, has attracted considerable attention because of its anti-inflammatory and neuroprotective properties. Studies have demonstrated that minocycline suppresses microglial activation, reduces inflammatory mediator production, and inhibits apoptotic pathways. Experimental models of both Alzheimer's disease and Parkinson's disease showed reductions in neuronal degeneration following minocycline treatment. Although clinical findings have been variable, minocycline remains an important example of successful drug repositioning research.

The role of oxidative stress in neurodegeneration has also been extensively documented. Oxidative stress occurs when the production of reactive oxygen species exceeds the capacity of antioxidant defense mechanisms. Neurons are particularly vulnerable to oxidative damage because of their high oxygen consumption, abundant lipid content, and limited regenerative capacity. Oxidative damage affects proteins, lipids, DNA, and cellular organelles, ultimately contributing to neuronal death.

Researchers have investigated numerous antioxidant drugs for repositioning purposes. N-acetylcysteine (NAC), commonly used as a mucolytic agent and antidote for acetaminophen poisoning, has demonstrated significant antioxidant activity by replenishing intracellular glutathione levels. Experimental studies have shown that NAC reduces oxidative stress, improves mitochondrial function, and protects neurons from degeneration. These findings have encouraged further investigation of NAC as a potential therapy for neurodegenerative disorders.

The importance of protein misfolding and aggregation in neurodegenerative diseases has generated substantial interest in drugs capable of enhancing protein clearance mechanisms. Cellular pathways such as autophagy and the ubiquitin-proteasome system are responsible for removing damaged proteins and maintaining cellular homeostasis. Impairment of these pathways contributes to the accumulation of toxic protein aggregates, including amyloid-beta, tau, and alpha-synuclein.

Rapamycin, originally developed as an immunosuppressive agent, has been investigated for its ability to stimulate autophagy through inhibition of the mammalian target of rapamycin (mTOR) pathway. Experimental studies have demonstrated enhanced clearance of pathological protein aggregates, reduced neuronal damage, and improved cognitive function following rapamycin treatment. These findings highlight the potential of targeting cellular quality-control systems in neurodegenerative disease management.

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Recent literature has also emphasized the relationship between cardiovascular health and neurodegeneration. Cerebrovascular dysfunction contributes significantly to cognitive decline by reducing cerebral blood flow, impairing nutrient delivery, and promoting inflammatory responses. Consequently, cardiovascular drugs have emerged as attractive candidates for repositioning.

Research involving angiotensin receptor blockers such as losartan and candesartan has demonstrated reductions in neuroinflammation, oxidative stress, and vascular dysfunction. These drugs may provide neuroprotection by improving cerebral circulation and preserving blood-brain barrier integrity. Similarly, beta-blockers and calcium channel blockers have shown beneficial effects on neuronal function in preclinical studies.

The gut-brain axis has emerged as a novel area of investigation in neurodegenerative disease research. Growing evidence suggests that intestinal microbiota influence brain function through immune, endocrine, and neural pathways. Alterations in gut microbial composition have been observed in patients with Alzheimer's disease and Parkinson's disease, suggesting a potential role in disease pathogenesis.

Certain antimicrobial agents and probiotics have therefore been investigated as repositioning candidates. Research indicates that modulation of gut microbiota may reduce systemic inflammation, improve metabolic regulation, and influence neuroprotective signaling pathways. Although this field remains relatively new, it represents a promising direction for future therapeutic development.

Recent studies have also explored the role of hormonal therapies in neurodegenerative diseases. Estrogen possesses antioxidant, anti-inflammatory, and neuroprotective properties. Epidemiological studies have suggested that estrogen deficiency may contribute to increased susceptibility to neurodegeneration. Similarly, melatonin, commonly used for sleep disorders, has demonstrated antioxidant and mitochondrial protective effects in experimental models. These findings support the potential repositioning of hormonal agents for neurological applications.

The emergence of transcriptomics and genomics has significantly enhanced drug repositioning efforts. Large-scale gene expression studies allow researchers to compare disease-associated molecular signatures with drug-induced genetic changes. Drugs capable of reversing pathological gene expression patterns may possess therapeutic potential. This approach has facilitated the identification of numerous repositioning candidates that would have been difficult to discover through traditional methods.

Artificial intelligence has become one of the most influential technologies in modern drug repositioning research. Machine learning algorithms analyze vast amounts of biomedical data to identify hidden relationships among genes, proteins, diseases, and drugs. These computational approaches significantly reduce the time required for candidate identification and increase the probability of successful therapeutic discovery. AI-based platforms are increasingly used to predict drug efficacy, optimize clinical trial design, and identify patient populations most likely to benefit from specific treatments.

Several studies have highlighted the economic advantages of drug repositioning. Since approved drugs have already undergone extensive safety testing, pharmaceutical companies can avoid many costly stages of drug development. This reduction in development costs is particularly important for neurodegenerative diseases, where traditional drug discovery has historically experienced exceptionally high failure rates. Drug repositioning therefore represents a financially sustainable strategy for accelerating therapeutic innovation.

Clinical trial data have provided valuable insights into the challenges and opportunities associated with drug repositioning. While many repositioned drugs demonstrate promising effects in laboratory studies, successful translation into clinical practice requires careful consideration of dosage optimization, treatment duration, patient selection, and long-term safety monitoring. Nevertheless, the increasing number of ongoing clinical trials reflects growing confidence in the potential of drug repositioning to transform neurodegenerative disease management.

Current literature strongly suggests that future therapeutic success will likely depend on combination approaches targeting multiple disease mechanisms simultaneously. Repositioned drugs may play a crucial role in such strategies due to their diverse pharmacological properties and established safety profiles. Integration of drug repositioning with personalized medicine, biomarker-guided therapy, precision pharmacology, and artificial intelligence-driven drug

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discovery is expected to revolutionize the treatment of Alzheimer's disease and Parkinson's disease in the coming decades.

Thus, the available literature provides compelling evidence that drug repositioning offers a practical, economical, and scientifically robust approach for addressing the unmet therapeutic needs of neurodegenerative diseases. Continued advancements in molecular neuroscience, computational biology, pharmacology, and clinical research are expected to further expand the range of repositioned drugs available for Alzheimer's disease and Parkinson's disease, ultimately improving patient outcomes and reducing the global burden of these devastating disorders.

Recent advances in neurodegenerative disease research have increasingly highlighted the importance of cellular signaling pathways in the progression of Alzheimer's disease and Parkinson's disease. Disruptions in intracellular signaling mechanisms affect neuronal growth, survival, differentiation, and communication. Researchers have therefore investigated drugs capable of modulating these pathways as potential therapeutic candidates. Particular attention has been directed toward pathways involving phosphoinositide 3-kinase (PI3K), Akt, glycogen synthase kinase-3β (GSK-3β), mammalian target of rapamycin (mTOR), and AMP-activated protein kinase (AMPK). Dysregulation of these signaling networks contributes to abnormal protein aggregation, neuronal apoptosis, oxidative stress, and impaired synaptic function. Drug repositioning studies targeting these pathways have shown promising neuroprotective outcomes in experimental models.

Synaptic dysfunction is now recognized as one of the earliest pathological events in Alzheimer's disease and Parkinson's disease. Before extensive neuronal loss becomes evident, abnormalities in synaptic transmission impair communication between neurons, leading to cognitive deficits and motor dysfunction. Studies have demonstrated that synaptic deterioration strongly correlates with disease severity. Consequently, researchers have investigated drugs capable of preserving synaptic integrity and enhancing neurotransmission. Several repositioned compounds have demonstrated the ability to improve synaptic plasticity, increase neurotransmitter release, and promote neuronal connectivity, thereby potentially slowing disease progression.(14,15,16,17)

VI. ROLE AND CLASSIFICATION

- **Role and Classification of Drug Repositioning in Neurodegenerative Diseases**

Drug repositioning, also known as drug repurposing, drug rediscovery, or therapeutic switching, is a modern pharmaceutical strategy that involves identifying new therapeutic applications for existing drugs that have already been approved or investigated for other medical conditions. In recent years, drug repositioning has gained tremendous importance in the field of neurodegenerative diseases, particularly Alzheimer's disease (AD) and Parkinson's disease (PD), due to the limited success of traditional drug discovery approaches. The increasing prevalence of these disorders, combined with the complexity of their pathogenesis and the high failure rates of novel drug candidates, has made drug repositioning an attractive alternative for developing effective therapies.

Role of Drug Repositioning in Neurodegenerative Diseases

1. Accelerating Drug Development

One of the most significant roles of drug repositioning is the reduction in time required for drug development. Conventional drug discovery typically requires 10–15 years of research before a drug reaches the market. Since repositioned drugs have already undergone extensive safety and toxicity evaluations, many stages of development can be bypassed. This enables researchers to focus directly on evaluating therapeutic efficacy for neurodegenerative diseases.

2. Reducing Research and Development Costs

The development of a new drug involves enormous financial investment. Drug repositioning substantially lowers development costs because preclinical toxicity studies, pharmacokinetic evaluations, and manufacturing processes are already established. This economic advantage is particularly important in neurodegenerative diseases, where many investigational compounds fail during clinical trials.

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3. Providing Safer Therapeutic Options

Approved drugs possess well-documented safety profiles, adverse effect information, dosage recommendations, and drug interaction data. Consequently, repositioned drugs generally carry lower safety risks than newly developed compounds. This improves the likelihood of successful clinical application in patients with Alzheimer's disease and Parkinson's disease.

4. Targeting Multiple Disease Mechanisms

Neurodegenerative diseases involve numerous interconnected pathological pathways.

These include:

- Oxidative stress
- Neuroinflammation
- Protein aggregation
- Mitochondrial dysfunction
- Synaptic degeneration
- Impaired autophagy
- Apoptosis
- Insulin resistance
- Vascular dysfunction

Many repositioned drugs possess pleiotropic effects, allowing them to simultaneously influence multiple disease pathways. This multitarget activity may provide greater therapeutic benefits than single-target therapies.

5. Enhancing Neuroprotection

Several repositioned drugs have demonstrated neuroprotective properties by:

- Protecting neurons from oxidative damage
- Improving neuronal survival
- Enhancing synaptic plasticity
- Reducing excitotoxicity
- Promoting neuronal repair mechanisms
- Improving neurotransmitter balance

These effects may contribute to slowing disease progression and preserving neurological function.

6. Reducing Neuroinflammation

Chronic neuroinflammation is a major contributor to neuronal degeneration. Activated microglia and astrocytes release inflammatory mediators that damage neurons. Repositioned anti-inflammatory drugs can suppress these inflammatory responses and reduce neuronal injury.

7. Improving Mitochondrial Function

Mitochondria play a critical role in cellular energy production. Dysfunctional mitochondria contribute to neuronal death through excessive production of reactive oxygen species and energy deficiency. Several repositioned drugs improve mitochondrial efficiency, enhance ATP production, and reduce oxidative stress.

8. Promoting Protein Clearance

The accumulation of abnormal proteins such as amyloid-beta, tau, and alpha-synuclein is a hallmark of neurodegenerative diseases. Certain repositioned drugs stimulate autophagy and lysosomal degradation pathways, facilitating the removal of toxic protein aggregates.

9. Supporting Personalized Medicine

Drug repositioning can contribute to personalized treatment approaches by identifying drugs suitable for specific patient populations based on genetic, molecular, and biomarker profiles.

10. Improving Quality of Life

By slowing disease progression, reducing symptom severity, and preserving cognitive and motor functions, repositioned drugs may improve patient independence, daily functioning, and overall quality of life.

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Classification of Drug Repositioning

A. Classification Based on Discovery Approach

1. Serendipitous Drug Repositioning

This occurs when new therapeutic effects are discovered accidentally during clinical use or research studies.

Characteristics:

• Based on unexpected observations
• Often discovered during post-marketing surveillance
• Requires further validation

Example:

Sildenafil was originally developed for angina but later repositioned for erectile dysfunction.

2. Computational Drug Repositioning

This approach uses bioinformatics, artificial intelligence, machine learning, molecular docking, and network pharmacology to identify new drug-disease relationships.

Characteristics:

• Data-driven approach
• Faster screening of thousands of compounds
• Cost-effective and efficient

3. Experimental Drug Repositioning

This involves laboratory testing using cell cultures, animal models, and biochemical assays.

Characteristics:

• Direct experimental validation
• Identification of novel biological activities
• Provides mechanistic insights

4. Clinical Observation-Based Repositioning

This approach is based on observations of beneficial effects in patients receiving drugs for unrelated diseases.

Characteristics:

• Derived from clinical practice
• Supported by epidemiological studies
• Often leads to new clinical trials

B. Classification Based on Therapeutic Target

1. On-Target Repositioning

The drug acts on the same molecular target but is used for a different disease.

Characteristics:

• Same mechanism of action
• Different clinical indication
• Established pharmacology

2. Off-Target Repositioning

The drug exerts therapeutic effects through a different molecular target than originally intended.

Characteristics:

• Novel mechanism discovered
• Broader therapeutic potential
• Often identified through advanced research techniques

C. Classification Based on Therapeutic Area

1. Disease-Centric Repositioning

The drug is repositioned for a disease closely related to its original indication.

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Examples:

• Drugs used for one neurological disorder being applied to another neurological condition.

2. Target-Centric Repositioning

The drug targets a biological pathway common to multiple diseases.

Examples:

• Anti-inflammatory drugs used in both autoimmune disorders and neurodegenerative diseases.

3. Drug-Centric Repositioning

A drug is investigated for entirely new therapeutic applications based on its pharmacological properties.

Examples:

• Anticancer drugs investigated for Alzheimer's disease and Parkinson's disease.

Classification of Repositioned Drugs Used in Neurodegenerative Diseases

1. Antidiabetic Drugs

Examples:

• Metformin
• Pioglitazone
• Exenatide
• Liraglutide
• Semaglutide

Role:

• Improve insulin signaling
• Reduce neuroinflammation
• Enhance mitochondrial function
• Promote neuronal survival

2. Antihypertensive Drugs

Examples:

• Losartan
• Candesartan
• Telmisartan
• Isradipine

Role:

• Improve cerebral blood flow
• Reduce oxidative stress
• Protect neuronal tissues

3. Anti-Inflammatory Drugs

Examples:

• Ibuprofen
• Celecoxib
• Minocycline

Role:

• Suppress inflammatory mediators
• Reduce microglial activation
• Protect neurons from inflammatory damage

4. Anticancer Drugs

Examples:

• Nilotinib
• Dasatinib

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Role:
• Promote autophagy
• Enhance protein clearance
• Reduce toxic protein accumulation

5. Lipid-Lowering Agents

Examples:
• Atorvastatin
• Simvastatin

Role:
• Improve vascular health
• Reduce oxidative stress
• Modulate inflammatory responses

6. Antimicrobial Agents

Examples:
• Rifampicin
• Doxycycline
• Minocycline

Role:
• Reduce protein aggregation
• Suppress neuroinflammation
• Provide neuroprotection

7. Antioxidant Agents

Examples:
• N-Acetylcysteine
• Coenzyme Q10
• Melatonin

Role:
• Neutralize reactive oxygen species
• Protect neuronal membranes
• Improve mitochondrial function

8. Immunomodulatory Drugs

Examples:
• Fingolimod
• Methotrexate

Role:
• Regulate immune responses
• Reduce chronic neuroinflammation
• Protect neural tissues

9. Hormonal Agents

Examples:
• Estrogen
• Progesterone
• Melatonin

Role:

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- Enhance neuronal survival
- Improve cognitive function
- Provide antioxidant protection

10. Neuroprotective Agents

Examples:

- Rasagiline
- Ambroxol

Role:

- Prevent neuronal degeneration
- Enhance lysosomal activity
- Improve neurotransmission

11. Role in Disease Modification

One of the most important roles of drug repositioning is its potential to develop disease-modifying therapies rather than merely providing symptomatic relief. Most currently available drugs for Alzheimer's disease and Parkinson's disease improve symptoms temporarily without addressing the underlying causes of neurodegeneration. Repositioned drugs have demonstrated the ability to interfere with pathological mechanisms such as amyloid-beta accumulation, tau hyperphosphorylation, alpha-synuclein aggregation, neuroinflammation, and mitochondrial dysfunction. By targeting these fundamental disease processes, repositioned drugs may slow or delay disease progression and preserve neuronal function for longer periods.

12. Role in Early Intervention

Research indicates that neurodegenerative diseases begin years or even decades before clinical symptoms become apparent. Drug repositioning facilitates the identification of compounds that can be administered during early disease stages or even before symptom onset. Early intervention may prevent extensive neuronal loss and improve long-term clinical outcomes. Existing drugs with proven safety profiles are particularly suitable for long-term preventive use in high-risk populations.

13. Role in Biomarker-Based Therapy

Modern neurodegenerative disease management increasingly relies on biomarkers for diagnosis, monitoring, and treatment selection. Repositioned drugs can be evaluated against specific biomarkers such as amyloid-beta levels, tau protein concentrations, alpha-synuclein accumulation, inflammatory markers, and neurofilament proteins. This approach allows more precise assessment of therapeutic responses and facilitates personalized treatment strategies.

14. Role in Precision Medicine

Precision medicine aims to tailor treatment according to an individual's genetic makeup, environmental factors, lifestyle, and disease characteristics. Drug repositioning contributes significantly to this approach by providing multiple therapeutic options that can be matched to specific patient profiles. Genetic studies can identify patient populations most likely to benefit from particular repositioned drugs, thereby improving efficacy and reducing adverse effects.

15. Role in Combination Therapy

Neurodegenerative diseases involve multiple pathological pathways that often cannot be effectively addressed by a single drug. Drug repositioning enables the development of combination therapies involving multiple agents targeting different disease mechanisms simultaneously. For example, one repositioned drug may reduce inflammation while another enhances mitochondrial function or promotes protein clearance. Such combination approaches may provide synergistic therapeutic benefits.

16. Role in Neuroregeneration

Emerging evidence suggests that certain repositioned drugs may stimulate neuronal regeneration and repair mechanisms. These drugs may promote neurogenesis, enhance synaptic plasticity, support axonal growth, and improve neuronal connectivity. Neuroregenerative therapies have the potential to restore lost neurological functions and improve recovery in patients with neurodegenerative disorders.

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17. Role in Improving Blood-Brain Barrier Function

The blood-brain barrier plays a critical role in maintaining central nervous system homeostasis. Disruption of this barrier contributes to neuroinflammation and neuronal injury. Several repositioned drugs have demonstrated the ability to strengthen blood-brain barrier integrity, reduce vascular permeability, and improve cerebral microcirculation, thereby providing indirect neuroprotective effects.

18. Role in Enhancing Cellular Homeostasis

Neurons depend on highly regulated cellular processes to maintain normal function. Repositioned drugs may restore cellular homeostasis by regulating protein synthesis, energy metabolism, calcium signaling, and intracellular transport mechanisms. Maintaining cellular balance is essential for preventing neuronal dysfunction and degeneration.

19. Role in Reducing Healthcare Burden

Neurodegenerative diseases place enormous economic burdens on healthcare systems worldwide. Drug repositioning offers a cost-effective alternative to traditional drug development and may reduce treatment expenses by utilizing readily available medications. Effective repositioned therapies can decrease hospitalization rates, delay institutional care, and reduce long-term healthcare costs.

20. Role in Expanding Therapeutic Options

Many neurodegenerative diseases have limited treatment choices. Drug repositioning significantly expands the pool of potential therapeutic agents by exploring approved drugs from various therapeutic classes. This diversification increases the likelihood of identifying effective treatments for patients with different disease stages and clinical presentations.

Advanced Classification of Drug Repositioning

D. Classification Based on Source of Discovery

1. Knowledge-Based Repositioning

Knowledge-based repositioning relies on existing scientific information regarding disease mechanisms, drug targets, biochemical pathways, and clinical observations. Researchers utilize accumulated biological knowledge to identify new therapeutic applications for known drugs.

Characteristics:

1. Based on scientific literature.
2. Utilizes existing pharmacological data.
3. Supports hypothesis-driven research.
4. Reduces uncertainty during candidate selection.

2. Signature-Based Repositioning

This method compares disease-associated gene expression patterns with drug-induced gene expression profiles. Drugs capable of reversing pathological genetic signatures may possess therapeutic value.

Characteristics:

1. Utilizes genomic databases.
2. Employs transcriptomic analysis.
3. Identifies novel drug-disease relationships.
4. Supports personalized medicine approaches.

3. Pathway-Based Repositioning

Pathway-based repositioning focuses on biological pathways involved in disease progression. Drugs targeting these pathways may be repurposed for new therapeutic indications.

Characteristics:

1. Targets interconnected molecular networks.
2. Addresses multifactorial disease mechanisms.
3. Suitable for complex neurological disorders.

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4. Supports multitarget therapeutic strategies.

E. Classification Based on Molecular Mechanism

1. Anti-Amyloid Agents

These drugs reduce amyloid-beta production, aggregation, or deposition in the brain.

Functions:

1. Prevent plaque formation.
2. Reduce neuronal toxicity.
3. Improve cognitive function.
4. Slow Alzheimer's disease progression.

2. Anti-Tau Agents

These drugs inhibit tau protein hyperphosphorylation and aggregation.

Functions:

1. Prevent neurofibrillary tangle formation.
2. Preserve neuronal structure.
3. Improve intracellular transport.
4. Reduce cognitive decline.

3. Anti-Alpha-Synuclein Agents

These compounds target alpha-synuclein aggregation in Parkinson's disease.

Functions:

1. Reduce Lewy body formation.
2. Improve neuronal survival.
3. Enhance protein clearance.
4. Slow disease progression.

4. Mitochondrial Protective Agents

These drugs improve mitochondrial function and energy production.

Functions:

1. Increase ATP synthesis.
2. Reduce oxidative damage.
3. Improve neuronal metabolism.
4. Enhance cell survival.

5. Autophagy Enhancers

These drugs stimulate intracellular degradation pathways.

Functions:

1. Remove damaged proteins.
2. Eliminate dysfunctional organelles.
3. Maintain cellular homeostasis.
4. Reduce toxic protein accumulation.

F. Classification Based on Therapeutic Outcome

1. Symptom-Modifying Drugs

These drugs primarily improve clinical symptoms without significantly altering disease progression.

Examples:

1. Cognitive enhancers.
2. Dopaminergic agents.
3. Motor symptom regulators.

2. Disease-Modifying Drugs

These drugs target underlying pathological mechanisms.

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Examples:

1. Anti-inflammatory agents.
2. Mitochondrial protectors.
3. Protein aggregation inhibitors.

3. Neuroprotective Drugs

These drugs protect neurons from damage and degeneration.

Examples:

1. Antioxidants.
2. Anti-apoptotic agents.
3. Neurotrophic factor enhancers.

4. Neurorestorative Drugs

These agents promote neuronal repair and regeneration.

Examples:

1. Growth factor stimulators.
2. Synaptic plasticity enhancers.
3. Neurogenesis-promoting compounds.

The expanding role and classification of drug repositioning demonstrate its importance as a transformative strategy in neurodegenerative disease research. By targeting diverse pathological mechanisms and utilizing advanced discovery approaches, drug repositioning provides a promising pathway for developing safer, faster, and more effective therapies for Alzheimer's disease and Parkinson's disease. The continuous integration of molecular biology, pharmacology, bioinformatics, artificial intelligence, and clinical medicine is expected to further enhance the success of drug repositioning in future neurological therapeutics.

5. Materials and Methods :

The present project entitled **"Drug Repositioning for Neurodegenerative Diseases: Alzheimer's Disease and Parkinson's Disease"** was carried out as a review-based and literature-based research study. Since the project focuses on the evaluation of existing drugs for new therapeutic applications in neurodegenerative disorders, no experimental animals, human subjects, or laboratory-based formulations were directly involved. The materials used for this study primarily consisted of scientific literature, research articles, review papers, clinical trial reports, pharmaceutical databases, and online scientific resources.

1. Research Articles

Published research articles related to drug repositioning, Alzheimer's disease, Parkinson's disease, neurodegeneration, neuroprotection, and therapeutic drug development were collected from various scientific journals. These articles provided information regarding disease mechanisms, therapeutic targets, drug efficacy, and clinical outcomes.

2. Review Articles

Comprehensive review articles were utilized to obtain detailed knowledge regarding the pathophysiology of neurodegenerative diseases, current treatment approaches, limitations of existing therapies, and advances in drug repurposing strategies.

3. Clinical Trial Reports

Clinical trial studies investigating the effectiveness and safety of repositioned drugs in Alzheimer's disease and Parkinson's disease were reviewed. These reports provided evidence regarding therapeutic benefits, adverse effects, dosage regimens, and treatment outcomes.

4. Scientific Databases

Various scientific and biomedical databases were used for literature collection and information retrieval, including:

1. PubMed

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2. Google Scholar
3. ScienceDirect
4. SpringerLink
5. Wiley Online Library
6. ResearchGate
7. Scopus
8. MEDLINE
9. NCBI Database

These databases provided access to peer-reviewed scientific literature relevant to the project.

5. Books and Reference Materials

Standard textbooks related to pharmacology, neuropharmacology, medicinal chemistry, neuroscience, pathology, and drug discovery were consulted to obtain fundamental information regarding neurodegenerative diseases and drug repositioning approaches.

6. Online Resources

Reliable online sources including governmental health organizations, research institutes, pharmaceutical organizations, and scientific publications were used for collecting updated information regarding ongoing research and clinical developments.

7. Software and Computational Resources

Various computational tools and bioinformatics resources discussed in scientific literature were reviewed, including:

1. Molecular docking platforms
2. Artificial intelligence-based drug discovery systems
3. Network pharmacology databases
4. Drug-target interaction databases
5. Bioinformatics analysis tools

These resources were studied to understand modern approaches involved in drug repositioning.

Methods

The methodology adopted for this project involved a systematic collection, evaluation, analysis, and interpretation of scientific literature related to drug repositioning in Alzheimer's disease and Parkinson's disease.

1. Literature Survey

A comprehensive literature survey was conducted to collect scientific information related to neurodegenerative diseases and drug repurposing. Research articles published in national and international journals were searched using keywords such as:

- Drug repositioning
- Drug repurposing
- Neurodegenerative diseases
- Alzheimer's disease
- Parkinson's disease
- Neuroprotection
- Neuroinflammation
- Mitochondrial dysfunction
- Protein aggregation
- Neuropharmacology

The collected literature was carefully screened for relevance and scientific validity.

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2. Collection of Scientific Data

Scientific data related to disease pathology, therapeutic targets, mechanisms of neurodegeneration, and repositioned drugs were gathered from peer-reviewed publications and clinical studies. Information regarding approved drugs with potential neuroprotective properties was also collected.

3. Study of Alzheimer's Disease

An extensive review was conducted to understand:

1. Epidemiology
2. Etiology
3. Risk factors
4. Pathophysiology
5. Amyloid-beta pathology
6. Tau protein abnormalities
7. Oxidative stress mechanisms
8. Neuroinflammatory processes
9. Current treatment strategies

This information was used to identify potential therapeutic targets for drug repositioning.

4. Study of Parkinson's Disease

Detailed literature analysis was performed to investigate:

1. Epidemiology
2. Etiology
3. Clinical manifestations
4. Dopaminergic neuronal degeneration
5. Alpha-synuclein aggregation
6. Lewy body formation
7. Mitochondrial dysfunction
8. Oxidative stress
9. Existing therapeutic approaches

The collected information facilitated the identification of pathways suitable for drug repurposing interventions.

5. Identification of Repositioned Drugs

Published studies were reviewed to identify drugs originally developed for other diseases that demonstrated therapeutic potential in neurodegenerative disorders.

Drug categories evaluated included:

1. Antidiabetic drugs
2. Antihypertensive drugs
3. Anti-inflammatory drugs
4. Anticancer drugs
5. Antimicrobial agents
6. Antioxidants
7. Immunomodulators
8. Hormonal agents
9. Neuroprotective compounds

Each drug was assessed based on its mechanism of action and relevance to neurodegenerative disease pathology.

6. Evaluation of Mechanisms of Action

The mechanisms through which repositioned drugs exert neuroprotective effects were studied in detail.

These mechanisms included:

1. Reduction of oxidative stress

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2. Suppression of neuroinflammation
3. Enhancement of mitochondrial function
4. Promotion of autophagy
5. Inhibition of protein aggregation
6. Improvement of synaptic plasticity
7. Regulation of neurotransmitter activity
8. Prevention of neuronal apoptosis

7. Analysis of Preclinical Studies

Experimental studies involving cell cultures and animal models were reviewed to evaluate the efficacy of repositioned drugs.

The following parameters were analyzed:

1. Neuroprotection
2. Behavioral improvement
3. Cognitive enhancement
4. Reduction of pathological protein accumulation
5. Improvement of neuronal survival
6. Biochemical alterations

8. Analysis of Clinical Studies

Human clinical studies were examined to assess:

1. Therapeutic efficacy
2. Safety profile
3. Tolerability
4. Dosage regimens
5. Clinical outcomes
6. Adverse effects
7. Long-term effectiveness

Results from clinical trials were compared and interpreted to determine the potential applicability of repositioned drugs.

9. Comparative Evaluation

Different classes of repositioned drugs were compared based on:

1. Mechanism of action
2. Therapeutic effectiveness
3. Safety profile
4. Clinical evidence
5. Regulatory status
6. Future potential

This comparative analysis enabled identification of the most promising drug candidates.

10. Data Compilation and Interpretation

All collected information was systematically organized, analyzed, and interpreted. Scientific findings from various sources were integrated to develop a comprehensive understanding of drug repositioning strategies for Alzheimer's disease and Parkinson's disease.

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9. Collection and Authentication of Materials :

The materials required for this review-based project were collected from various authentic and reliable scientific sources, including research articles, review papers, clinical trial reports, textbooks, and online biomedical databases such as PubMed, Google Scholar, ScienceDirect, and SpringerLink. Information related to drug repositioning, Alzheimer's disease, and Parkinson's disease was systematically gathered from peer-reviewed publications. The collected literature was carefully screened and authenticated based on publication quality, scientific validity, relevance to the study, and credibility of the source. Only updated and reliable references were included to ensure the accuracy and authenticity of the information used in the project.

10. Evaluation and Formulation

Formulation

In this project, formulation refers to the identification and selection of existing approved drugs with potential therapeutic applications in Alzheimer's disease and Parkinson's disease through drug repositioning approaches. Various classes of drugs such as antidiabetic agents, antihypertensive drugs, anti-inflammatory agents, anticancer drugs, antioxidants, and neuroprotective compounds were reviewed based on their mechanisms of action and neuroprotective potential. Suitable drug candidates were selected by analyzing their ability to target key pathological pathways involved in neurodegeneration.

Evaluation

The selected repositioned drugs were evaluated based on the following parameters:

1. Neuroprotective activity
2. Ability to reduce oxidative stress
3. Anti-inflammatory effects
4. Improvement of mitochondrial function
5. Inhibition of amyloid-beta and alpha-synuclein aggregation
6. Enhancement of autophagy and protein clearance
7. Improvement in cognitive and motor functions
8. Safety and tolerability profiles
9. Clinical efficacy reported in preclinical and clinical studies
10. Potential to slow disease progression

The evaluation was carried out through a detailed review and analysis of published scientific literature, experimental studies, and clinical trial reports related to Alzheimer's disease and Parkinson's disease.

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11. PharmacologicalEvaluation :

Pharmacological evaluation is an essential step in assessing the therapeutic potential of repositioned drugs for the treatment of neurodegenerative diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD). The evaluation focuses on determining the efficacy, safety, mechanism of action, neuroprotective properties, and disease-modifying potential of existing drugs that are being investigated for new neurological applications. Various in vitro studies, animal experiments, and clinical investigations have been conducted to evaluate the pharmacological effects of repositioned drugs on different pathological mechanisms involved in neurodegeneration.

Evaluation of Neuroprotective Activity

Neuroprotective activity is one of the primary parameters assessed during pharmacological evaluation. Repositioned drugs are investigated for their ability to protect neurons from degeneration, apoptosis, and cellular damage. Experimental studies evaluate neuronal survival under conditions of oxidative stress, inflammation, excitotoxicity, and protein aggregation. Drugs demonstrating significant neuroprotective effects are considered promising candidates for further clinical investigation.

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Evaluation of Anti-Inflammatory Activity

Chronic neuroinflammation contributes significantly to the progression of Alzheimer's disease and Parkinson's disease. Pharmacological evaluation involves assessing the ability of repositioned drugs to suppress inflammatory responses within the central nervous system. This includes measurement of inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6). Reduction in microglial activation and inflammatory mediator production indicates potential therapeutic benefit.

Evaluation of Antioxidant Activity

Oxidative stress is a major contributor to neuronal injury and disease progression. Pharmacological studies assess the antioxidant properties of repositioned drugs by measuring their ability to reduce reactive oxygen species (ROS), lipid peroxidation, and oxidative cellular damage. Enhancement of endogenous antioxidant enzymes such as superoxide dismutase, catalase, and glutathione peroxidase is also evaluated. Drugs with strong antioxidant activity may help preserve neuronal integrity and function.

Evaluation of Mitochondrial Function

Mitochondrial dysfunction is commonly observed in neurodegenerative disorders. Pharmacological evaluation includes assessment of mitochondrial membrane potential, ATP production, oxygen consumption rate, and mitochondrial enzyme activity. Repositioned drugs capable of improving mitochondrial function and energy metabolism may reduce neuronal vulnerability and enhance cellular survival.

Evaluation of Protein Aggregation Inhibition

The accumulation of abnormal proteins such as amyloid-beta, tau protein, and alpha-synuclein is a hallmark of neurodegenerative diseases. Pharmacological studies investigate the ability of repositioned drugs to prevent protein aggregation, promote protein clearance, and reduce toxic intracellular deposits. Effective inhibition of protein accumulation may contribute to slowing disease progression.

Evaluation of Autophagy Enhancement

Autophagy is a cellular degradation mechanism responsible for removing damaged proteins and organelles. Pharmacological evaluation examines whether repositioned drugs can activate autophagic pathways and improve lysosomal function. Enhanced autophagy facilitates the clearance of pathological protein aggregates and supports neuronal health.

Evaluation of Cognitive Function

In Alzheimer's disease models, cognitive performance is assessed using behavioral and memory tests. Improvement in learning ability, memory retention, spatial navigation, and cognitive flexibility indicates potential therapeutic efficacy. Repositioned drugs demonstrating cognitive enhancement are considered valuable candidates for dementia treatment.

Evaluation of Motor Function

For Parkinson's disease, pharmacological evaluation includes assessment of motor coordination, locomotor activity, muscle rigidity, balance, and movement control. Improvement in motor performance following drug administration suggests neuroprotective effects on dopaminergic neurons and motor pathways.

Evaluation of Dopaminergic Neuroprotection

In Parkinson's disease studies, the survival of dopaminergic neurons in the substantia nigra is an important parameter. Pharmacological investigations measure dopamine levels, neuronal density, and neurotransmitter function. Drugs capable of preserving dopaminergic neurons may help reduce motor symptoms and disease progression.

Evaluation of Synaptic Plasticity

Synaptic plasticity plays a critical role in learning, memory, and neuronal communication. Pharmacological evaluation includes examination of synaptic protein expression, neurotransmitter release, and neuronal connectivity. Improved synaptic function indicates restoration of neuronal communication and cognitive performance.

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Evaluation of Blood-Brain Barrier Penetration

An effective neuroprotective drug must reach therapeutic concentrations within the brain. Pharmacological studies evaluate the ability of repositioned drugs to cross the blood-brain barrier and achieve adequate distribution within neural tissues. Drugs with favorable brain penetration characteristics have greater potential for clinical success.

Evaluation of Pharmacokinetic Parameters

Pharmacokinetic evaluation assesses the absorption, distribution, metabolism, and excretion of repositioned drugs. Important parameters include bioavailability, half-life, plasma concentration, tissue distribution, and elimination rate. These factors influence therapeutic efficacy and dosing strategies.

Evaluation of Safety and Toxicity

Safety assessment is essential before clinical application. Pharmacological studies evaluate acute toxicity, chronic toxicity, organ toxicity, behavioral abnormalities, and adverse effects associated with drug administration. Since repositioned drugs are generally approved medications, their established safety profiles provide a significant advantage.

Evaluation Through Preclinical Studies

Preclinical evaluation involves laboratory investigations using cell culture models and experimental animals. These studies provide information regarding mechanism of action, therapeutic efficacy, neuroprotection, biochemical changes, and disease-modifying potential before human testing.

Evaluation Through Clinical Studies

Clinical evaluation involves assessment of repositioned drugs in human subjects through controlled clinical trials. Parameters evaluated include therapeutic effectiveness, symptom improvement, disease progression, quality of life, cognitive performance, motor function, and long-term safety. Clinical studies provide critical evidence regarding the practical applicability of repositioned drugs in neurodegenerative disease management.

Evaluation of Disease-Modifying Potential

Unlike conventional symptomatic treatments, repositioned drugs are also evaluated for their ability to alter the underlying course of disease progression. Reduction in neuronal loss, suppression of pathological processes, delayed symptom progression, and preservation of neurological function indicate disease-modifying activity. Such effects are considered highly valuable in Alzheimer's disease and Parkinson's disease.

In addition to the conventional pharmacological parameters, modern drug repositioning studies for Alzheimer's disease and Parkinson's disease involve a comprehensive evaluation of several advanced biological and molecular mechanisms. These evaluations help determine whether a repositioned drug can effectively interfere with disease progression and provide long-term therapeutic benefits. Since neurodegenerative disorders are highly complex and multifactorial, pharmacological assessment extends beyond symptomatic improvement and focuses on disease-modifying effects at cellular, molecular, biochemical, and genetic levels.

Evaluation of Neurotransmitter Regulation

Neurotransmitter imbalance is a major pathological feature of neurodegenerative diseases. Pharmacological evaluation involves assessing the ability of repositioned drugs to regulate neurotransmitter synthesis, release, uptake, and degradation. In Alzheimer's disease, acetylcholine deficiency contributes to memory impairment and cognitive dysfunction, whereas Parkinson's disease is characterized by dopamine depletion due to degeneration of dopaminergic neurons. Repositioned drugs are evaluated for their capacity to restore neurotransmitter balance, improve neuronal communication, and enhance overall brain function. Improved neurotransmitter regulation may lead to better cognitive performance, motor coordination, learning ability, and behavioral outcomes.

Evaluation of Anti-Apoptotic Activity

Programmed cell death, or apoptosis, plays a significant role in neuronal loss during neurodegeneration. Pharmacological studies investigate whether repositioned drugs can inhibit apoptotic signaling pathways and promote neuronal survival. Evaluation includes the measurement of apoptosis-related proteins such as Bax, Bcl-2, Caspases, and cytochrome-C. Drugs that suppress apoptotic mechanisms may prevent excessive neuronal death and preserve brain

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function. The anti-apoptotic potential of repositioned drugs is considered an important indicator of disease-modifying activity.

Evaluation of Neurogenesis Promotion

Recent research has demonstrated that certain regions of the adult brain retain the capacity to generate new neurons. Pharmacological evaluation investigates whether repositioned drugs stimulate neurogenesis within brain areas such as the hippocampus and subventricular zone. Increased neuronal proliferation, differentiation, and maturation indicate regenerative potential. Enhanced neurogenesis may contribute to cognitive improvement, memory restoration, and recovery of neuronal networks damaged by neurodegenerative processes.

Evaluation of Synaptic Protein Expression

Synaptic proteins are essential for communication between neurons. Neurodegenerative diseases often lead to reductions in synaptic density and function. Pharmacological studies evaluate the effects of repositioned drugs on proteins such as synaptophysin, PSD-95, SNAP-25, and other synaptic markers. Increased expression of these proteins suggests improved synaptic integrity and neuronal connectivity. Preservation of synaptic function is strongly associated with better cognitive and motor performance.

Evaluation of Cellular Energy Metabolism

Neurons require large amounts of energy to maintain normal physiological activity. Impaired energy metabolism contributes to neuronal dysfunction and degeneration. Pharmacological evaluation includes assessment of glucose utilization, ATP generation, mitochondrial respiration, and metabolic enzyme activity. Repositioned drugs that improve cellular energy production may enhance neuronal resilience and reduce susceptibility to pathological stress.

Evaluation of Calcium Homeostasis

Calcium ions regulate numerous neuronal functions, including neurotransmitter release, synaptic plasticity, and cellular signaling. Dysregulated calcium homeostasis contributes to excitotoxicity and neuronal death. Pharmacological studies assess the ability of repositioned drugs to stabilize intracellular calcium levels and prevent calcium-mediated toxicity. Improved calcium regulation supports neuronal survival and functional stability.

Evaluation of Endoplasmic Reticulum Stress Reduction

The endoplasmic reticulum is responsible for protein synthesis and folding. Accumulation of misfolded proteins induces endoplasmic reticulum stress, which contributes to neurodegeneration. Pharmacological evaluation examines whether repositioned drugs can reduce protein misfolding, normalize endoplasmic reticulum function, and decrease activation of stress-related signaling pathways. Reduction of cellular stress may enhance neuronal viability and delay disease progression.

Evaluation of Lysosomal Function

Lysosomes are essential for intracellular waste disposal and protein degradation. Impaired lysosomal activity contributes to the accumulation of toxic protein aggregates in Alzheimer's disease and Parkinson's disease. Pharmacological studies evaluate lysosomal enzyme activity, substrate degradation efficiency, and intracellular clearance mechanisms. Drugs that enhance lysosomal function may improve cellular housekeeping processes and reduce neurotoxicity.

Evaluation of Neurovascular Protection

Proper cerebral blood flow is essential for neuronal health and function. Pharmacological evaluation investigates whether repositioned drugs improve vascular integrity, cerebral perfusion, endothelial function, and microcirculation. Enhanced neurovascular protection may reduce ischemic damage, improve nutrient delivery, and support long-term neuronal survival.

Evaluation of Blood-Brain Barrier Stabilization

Disruption of the blood-brain barrier contributes to inflammation and neurodegeneration. Advanced pharmacological assessment includes evaluating the effects of repositioned drugs on tight junction proteins, vascular permeability, and inflammatory infiltration into brain tissues. Improved blood-brain barrier integrity helps maintain a stable neural environment and protects neurons from harmful substances.

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Evaluation of Gene Expression Modulation

Modern pharmacological studies increasingly examine the influence of repositioned drugs on disease-related gene expression. Transcriptomic analyses are used to determine whether a drug can regulate genes involved in inflammation, oxidative stress, apoptosis, protein aggregation, and neuronal survival. Favorable gene expression changes may indicate long-term therapeutic benefits and disease-modifying potential.

Evaluation of Epigenetic Effects

Epigenetic alterations influence the development and progression of neurodegenerative diseases. Pharmacological evaluation investigates whether repositioned drugs affect DNA methylation, histone modification, and non-coding RNA regulation. Modulation of epigenetic mechanisms may restore normal cellular function and provide a novel therapeutic approach for neurodegenerative disorders.

Evaluation of Neurotrophic Factor Production

Neurotrophic factors are proteins that support neuronal growth, maintenance, and survival. Pharmacological studies assess whether repositioned drugs increase the production of brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF), and glial cell-derived neurotrophic factor (GDNF). Elevated neurotrophic factor levels promote neuronal repair, synaptic plasticity, and resistance to neurodegenerative damage.

Evaluation of Behavioral and Cognitive Performance

Comprehensive pharmacological evaluation includes assessment of behavioral and cognitive outcomes. Experimental models are used to evaluate learning ability, memory retention, attention, problem-solving skills, anxiety-related behavior, exploratory activity, and motor coordination. Improvements in these parameters provide functional evidence of therapeutic efficacy.

Evaluation of Long-Term Therapeutic Efficacy

Neurodegenerative diseases progress over many years; therefore, long-term effectiveness is a critical aspect of pharmacological evaluation. Studies assess whether repositioned drugs maintain their beneficial effects over extended treatment periods. Sustained neuroprotection, delayed disease progression, and prolonged preservation of cognitive and motor function indicate successful long-term therapeutic activity.

Evaluation of Drug-Drug Interactions

Patients with neurodegenerative diseases often receive multiple medications simultaneously. Pharmacological evaluation investigates potential interactions between repositioned drugs and standard therapies. Understanding drug-drug interactions is essential for optimizing treatment regimens, minimizing adverse effects, and ensuring patient safety.

Evaluation of Clinical Translation Potential

The ultimate objective of pharmacological evaluation is to determine whether promising preclinical findings can be successfully translated into clinical practice. Factors such as therapeutic efficacy, safety profile, dosing convenience, patient compliance, pharmacokinetics, and regulatory feasibility are carefully analyzed. Drugs demonstrating favorable characteristics across these parameters are considered strong candidates for future clinical application.

Comprehensive Pharmacological Significance

Comprehensive pharmacological evaluation provides critical evidence regarding the therapeutic potential of repositioned drugs for Alzheimer's disease and Parkinson's disease. Through detailed assessment of molecular pathways, cellular functions, biochemical processes, neuronal survival mechanisms, and clinical outcomes, researchers can identify drug candidates capable of slowing neurodegeneration, preserving neurological function, and improving patient quality of life. Such evaluations play a fundamental role in advancing drug repositioning as a promising strategy for the development of effective treatments for neurodegenerative disorders.

VII. RESULTS AND DISCUSSION

The present study was conducted to evaluate the potential of drug repositioning as an innovative therapeutic strategy for the management of neurodegenerative diseases, particularly Alzheimer's disease (AD) and Parkinson's disease (PD).

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A comprehensive review of scientific literature, clinical studies, and experimental investigations revealed that drug repositioning offers a promising alternative to conventional drug discovery by utilizing existing drugs with established safety profiles for new neurological indications.

The findings of the study demonstrated that numerous approved drugs from different therapeutic categories possess significant neuroprotective properties. Antidiabetic drugs such as metformin, pioglitazone, exenatide, and liraglutide showed beneficial effects through improvement of insulin signaling, reduction of neuroinflammation, enhancement of mitochondrial function, and promotion of neuronal survival. These findings support the growing evidence linking metabolic dysfunction with neurodegenerative disease progression.

The review further revealed that anti-inflammatory agents play a crucial role in reducing neurodegeneration. Chronic neuroinflammation has been identified as a major contributor to neuronal injury in both Alzheimer's disease and Parkinson's disease. Several repositioned drugs demonstrated the ability to suppress inflammatory cytokines, reduce microglial activation, and protect neural tissues from inflammatory damage. These observations suggest that modulation of neuroinflammation may represent an effective therapeutic approach for slowing disease progression.

Significant findings were also observed regarding the role of antioxidants and mitochondrial protective agents. Oxidative stress and mitochondrial dysfunction are recognized as central pathological features of neurodegenerative disorders. Repositioned drugs possessing antioxidant properties were found to reduce reactive oxygen species generation, improve cellular energy metabolism, and preserve neuronal integrity. Enhancement of mitochondrial function was associated with improved neuronal survival and reduced susceptibility to neurodegenerative damage.

The study demonstrated that several repositioned drugs exhibited the ability to inhibit pathological protein aggregation. In Alzheimer's disease, reduction of amyloid-beta accumulation and tau pathology was observed, whereas in Parkinson's disease, suppression of alpha-synuclein aggregation was reported. These findings indicate that repositioned drugs may directly target the underlying mechanisms responsible for disease progression rather than merely providing symptomatic relief.

Another important observation was the ability of certain repositioned drugs to stimulate autophagy and lysosomal degradation pathways. Enhanced cellular clearance mechanisms facilitated the removal of damaged proteins and dysfunctional organelles, thereby improving cellular homeostasis and reducing neurotoxicity. This mechanism is particularly important in neurodegenerative disorders where impaired protein clearance contributes significantly to disease pathology.

The analysis of clinical studies indicated that several repositioned drugs demonstrated favorable safety and tolerability profiles. Since these drugs have already undergone extensive toxicological and pharmacokinetic evaluation for their original indications, the risks associated with clinical development are substantially reduced. This advantage contributes to faster translation from laboratory research to clinical application.

The study also highlighted the growing importance of computational approaches, artificial intelligence, machine learning, and bioinformatics in identifying potential repositioning candidates. These advanced technologies have accelerated drug discovery processes by enabling rapid screening of large databases, prediction of drug-target interactions, and identification of novel therapeutic opportunities. Such approaches are expected to further enhance the efficiency and success rate of drug repositioning in the future.

From the collected evidence, it was observed that drug repositioning provides several advantages over traditional drug development. These include reduced research costs, shorter development timelines, lower failure rates, established safety information, and increased accessibility of therapeutic options. Such benefits are particularly valuable in neurodegenerative diseases, where urgent therapeutic needs remain largely unmet.

The discussion of the findings suggests that neurodegenerative diseases involve multiple interconnected pathological pathways, including oxidative stress, neuroinflammation, mitochondrial dysfunction, protein aggregation, apoptosis, synaptic impairment, and vascular abnormalities. Therefore, repositioned drugs capable of targeting multiple mechanisms simultaneously may offer superior therapeutic benefits compared to conventional single-target therapies.

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The reviewed literature further indicates that future treatment strategies will likely involve combination therapies incorporating multiple repositioned drugs with complementary mechanisms of action. Such multimodal approaches may improve treatment efficacy and provide enhanced protection against disease progression. Integration with personalized medicine, biomarker-guided therapy, and precision pharmacology may further optimize therapeutic outcomes.

Overall, the results of this study strongly support drug repositioning as a valuable and scientifically promising strategy for the treatment of Alzheimer's disease and Parkinson's disease. The accumulated evidence suggests that repositioned drugs possess significant potential to provide neuroprotection, improve cognitive and motor functions, slow disease progression, and enhance patient quality of life. Continued research, advanced computational technologies, and well-designed clinical trials are expected to expand the application of drug repositioning and contribute to the development of effective disease-modifying therapies for neurodegenerative disorders.

VIII. CONCLUSION

Drug repositioning has emerged as a highly promising and innovative strategy for the treatment of neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. The present study highlights that repurposing existing drugs offers a faster, safer, and more cost-effective alternative to conventional drug discovery approaches. Various repositioned drugs have demonstrated significant neuroprotective effects through mechanisms including reduction of oxidative stress, suppression of neuroinflammation, improvement of mitochondrial function, enhancement of autophagy, and inhibition of pathological protein aggregation.

The reviewed literature indicates that several approved drugs from different therapeutic classes possess the potential to slow disease progression and improve cognitive as well as motor functions. Furthermore, the availability of established safety and pharmacokinetic data significantly reduces the time and cost required for clinical development. Advances in bioinformatics, artificial intelligence, molecular docking, and network pharmacology have further accelerated the identification of promising drug candidates for neurodegenerative disorders.

Overall, drug repositioning represents a valuable therapeutic approach with the potential to address the growing global burden of Alzheimer's disease and Parkinson's disease. Continued research, clinical validation, and integration of modern computational technologies are expected to facilitate the development of effective disease-modifying therapies and improve the quality of life of affected patients. Thus, drug repositioning holds great promise for the future management of neurodegenerative diseases and may play a crucial role in advancing neurological therapeutics.

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