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Biotechnological interventions in neurodegenerative disorders: Exploring novel therapeutic strategies

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Abstract
Neurodegenerative diseases such as Alzheimer's and Parkinson's are characterized by the gradual loss of neurons, and diagnoses frequently happen too late because of the shortcomings of current clinical tests. This research emphasizes the importance of developing earlier and more precise diagnostic techniques. Progress in neuroimaging technologies, including MRI and PET scans, has enhanced diagnostic capabilities, and investigations into biomarkers found in blood or cerebrospinal fluid could facilitate early identification. Integrating these approaches with artificial intelligence may lead to the development of predictive models, thereby enhancing diagnosis, treatment, and overall patient outcomes. Psychedelics, specifically serotonergic substances like LSD, psilocybin, and DOI (2,5-dimethoxy-4-iodoamphetamine), are becoming more and more viable therapeutic options for the treatment of mental health issues and neurodegenerative illnesses. Because neurodegenerative diseases progress slowly and have few treatment choices, such as Alzheimer's and Parkinson's, they provide serious obstacles. According to recent studies, psychedelics have an impact on serotonin pathways, particularly those that include 5-HT2A receptors, which are essential for improving neuroplasticity and cognitive performance. Despite their therapeutic potential, challenges remain, including legal restrictions and the need for further research to understand the long-term effects and optimal dosing strategies. As the stigma surrounding psychedelics diminishes, interdisciplinary collaboration among pharmacy, biotechnology, and neuroscience is essential for advancing research and developing personalized treatment approaches. Notwithstanding their potential as therapeutics, there are still issues to be resolved, such as regulatory limitations and the requirement for more study to determine the long-term effects and ideal dose regimens. Interdisciplinary cooperation across pharmacy, biotechnology, and neurology is crucial for expanding research and creating individualized treatment plans as the stigma associated with psychedelics fades. The study on psychedelics and neurodegenerative illnesses is reviewed in this article, with a focus on the drugs' mechanisms of action, clinical evidence, and potential future applications.

Keywords: Neurodegenerative disorders, biotechnological interventions, novel therapeutic strategies, alzheimer's disease, parkinson's disease, huntington's disease, amyotrophic lateral sclerosis (ALS), gene therapy, stem cell therapy, CRISPR-Cas9, biomarkers, protein misfolding, neuroprotection, targeted drug delivery, nanotechnology in neurology, regenerative medicine

Introduction

Neurodegenerative diseases are characterized by aberrant protein accumulation and the progressive loss of certain nervous system cells. Although the type of protein buildup determines the classification of these illnesses, similar features include increased reactive oxygen species, neuroinflammation, neuronal death, and failure of cellular cleaning systems. [1, 2] Several neurodegenerative disorders involve the accumulation of misfolded proteins in different brain regions, such as amyloid-beta in Alzheimer's, tau in Alzheimer's and tauopathies, and α-synuclein in Parkinson's. These proteins have diverse functions, like regulating signaling and maintaining neuronal structures. However, misfolding or modifications lead to their aggregation, forming harmful structures that spread throughout the brain in distinct patterns, as shown in human and animal studies. [3, 4, 5] Cognitive and functional deterioration as well as the onset of neuropsychiatric symptoms are hallmarks of Alzheimer's disease (AD), a progressive neurodegenerative illness. Aggregation of soluble amyloid species into insoluble amyloid plaques is one of the fundamental biological processes of AD. In addition to neuroinflammation, synaptic and circuit failure, mitochondrial and bioenergetic problems, epigenetic modifications, and vascular abnormalities, hyperphosphorylation of tau results in the creation of intracellular neurofibrillary tangles and neuronal death. [6, 7] Cognitive and behavioral symptoms are exacerbated by neurochemical deficiencies caused by neuronal death in transmitter system source nuclei. [8] The goal of personalized medicine is to develop treatments that are specific to each patient by considering their genetic and environmental characteristics. Its goal is to use biotechnological developments to improve patient treatment and increase understanding of diseases. Although the focus of this strategy has been pharmacogenomics and pharmacogenetics, other tactics such as creating biomarkers and figuring out particular drug-targetable pathways are also being investigated. Advances in customized medicine have been further supported by landmark initiatives like the ENCODE project and the Human Genome Project. [9, 10]

Neurodegenerative disease mechanism

Since it has been practiced for more than 2000 years, traditional Chinese medicine (TCM) has provided important insights for the creation of contemporary drugs, particularly for the treatment of symptoms associated with neurodegenerative illnesses like ALS, Parkinson's, Alzheimer's, and Huntington's. Evidence suggests that several TCM substances may have neuroprotective effects

and reduce symptoms by modulating the autophagy-lysosomal pathway, which is a crucial step for removing misfolded proteins, even if there are presently no treatments for these disorders. Autophagy's function in the course of Alzheimer's and Parkinson's disease is highlighted by the connection between its dysregulation and protein aggregation. The advantages of TCM in neurodegenerative disease models through autophagy processes are the main topic of this review. [11]

Neurodegenerative medicine project in personalized medicine

Neurodegenerative diseases indeed present a significant challenge due to their complexity and the lack of effective therapies. Personalized medicine is a promising approach in tackling these diseases by tailoring treatments to individual genetic and environmental profiles. The advances in genomics and proteomics offer new avenues for understanding disease mechanisms and identifying novel biomarkers, which can enhance early diagnosis and therapeutic targeting. Projects like the Human Genome Project and ENCODE have laid the foundation for these developments by mapping genetic elements and their functional roles. Incorporating these insights into clinical practice is key to optimizing patient outcomes. This approach not only focuses on pharmacogenomics but also emphasizes uncovering new druggable targets and mechanisms, pushing the boundaries of current therapeutic strategies. [12, 13] Complex neurodegenerative illnesses such as Parkinson's, Alzheimer's, and multiple sclerosis are currently untreated. There is hope thanks to personalized medicine, which customizes treatments based on environmental and genetic factors. By using systems biology to integrate 'omics' data, medical diagnosis and therapy can be enhanced. SNPs and next-generation sequencing are two examples of the molecular techniques that have advanced and are essential for early detection and therapy. As these diseases become more common, treatment may change as a result of combining genetics and environmental knowledge. [14, 15, 16, 17]

Psychedelic Neurotherapeutics: Innovative Approaches to Neurodegenerative Disease Management

In addition to cognitive deficiencies, positive symptoms of schizophrenia include hallucinations and delusions, and negative symptoms include diminished social engagement and lack of motivation. Schizophrenia is a complicated mental condition. Antipsychotics, which block dopamine type 2 receptors (D2R), are the mainstay of current treatment, mostly addressing positive symptoms with only patchy alleviation for cognitive and negative symptoms. Second-generation antipsychotics, such clozapine, also target 5-HT2A serotonin receptors, although their therapeutic benefit is still up for question. First-generation antipsychotics, including chlorpromazine, have strong D2R antagonistic effects. Even if newer medications are more tolerable, two-thirds of patients experience considerable side effects and treatment resistance, which emphasize the need for alternative therapy. There is continued interest in 5-HT2A receptor regulation for potential future approaches. [18, 19] Serotonin (5-HT) acts on a variety of receptors in the brain, and 5-HT2A receptors (5-HT2ARs) are important in the development of psychosis. Early research using psychedelics such as LSD made the function of 5-HT2AR in controlling behavior and neural activity clearer. Although antipsychotic drugs use 5-HT2AR antagonism to alleviate symptoms, developing genuinely selective medications is still a challenge. Psychedelic-induced neuroplasticity and molecular tools are now being investigated in order to create more precisely tailored treatments for mental disorders such as schizophrenia. [20] Progressive loss of neurons is a hallmark of neurodegenerative illnesses such as Parkinson's and Alzheimer's, which are frequently associated with aberrant protein deposits (such as tau and α-synuclein). Aging is a major factor in the comparable pathophysiology shared by sporadic and hereditary patients. In order to create new treatments, current research attempts to comprehend these pathways. [21, 22]

Current therapies in neurodegenerative medicine

1. Symptomatic treatment

Four cholinesterase inhibitors (ChE-Is) and one NMDA receptor antagonist has received FDA approval to treat Alzh eimer's disease (AD).

Since tacrine is no longer anoption, the ChE Is that are bein g used are donepezil,

rivastigmine, and galantamine, in addition to memantine, an NMDA receptor antagonist.

According to clinical research, these medications stabilize d aily activities and lessen neuropsychiatric symptoms while i mproving cognitive scores by 1.5 to 3 points on the ADAS-cog scale.

The advantages of these medications last for at least a year when compared to a placebo. [23, 24] In order to make up for the loss of presynaptic cholinergic cells in the nucleus basalis, which provides acetylcholine to the cerebral cortex and amygdala, ChE-Is increase post-synaptic cholinergic stimulation. This distinct pre/post-synaptic separation, which is absent from other neurotransmitter systems, is essential to AD pathogenesis. Following ChE-I treatment, advanced imaging methods such as fMRI and FDG PET demonstrate enhanced cortical activity, confirming their function in enhancing cognitive circuit function. These resources might potentially aid in the future development of more potent treatments. [25, 26]

2. Disease modifying therapies

Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disorder characterized by the progressive loss of upper and lower motor neurons in the brain and spinal cord. This leads to muscle weakness, wasting, and pyramidal signs, affecting the limbs, chest, neck, and oropharyngeal muscles. Approximately 50% of patients also experience cognitive impairments, ranging from mild executive dysfunction to frontotemporal dementia (FTD), highlighting a spectrum shared by ALS and FTD. Death typically occurs within 2–5 years due to respiratory failure. [27] Around 15% of ALS cases are familial, often linked to mutations in genes like C9orf72, SOD1, TARDBP, and FUS. The remaining cases are sporadic. Pathologically, ALS is marked by motor neuron degeneration and protein aggregation, notably TDP-43 in 97% of cases, except for SOD1 and FUS mutation carriers. [28] Currently, only two drugs—riluzole and edaravone—are approved for ALS treatment. Riluzole reduces excitotoxicity, while edaravone acts as an antioxidant. However, these drugs offer limited benefits, and there is a pressing need for more effective therapies. Despite over 80 randomized controlled trials

(RCTs) since 1980, most have yielded negative results. Nonetheless, these studies contribute valuable insights for designing future trials aimed at combating this devastating disease. [29]

Personalised medicine in neurodegenerative disease

Neurodegenerative diseases, characterized by progressive neuronal dysfunction and loss, significantly impact health and economics, yet effective treatments remain elusive. [30] Personalized medicine, integrating genetic and environmental factors, offers hope by tailoring therapies to individual patients. Advances in genomics, proteomics, and molecular diagnostics, supported by milestones like the Human Genome Project and ENCODE, have driven progress in identifying biomarkers, understanding disease mechanisms, and optimizing treatments. [31] A systems medicine approach, combining omics data and systems biology, is essential to address disease complexity. Techniques like SNP analysis, next-generation sequencing, and polyomic biomarkers facilitate early diagnosis, therapy selection, and monitoring disease progression. [32] Environmental influences further complicate treatment translation, yet personalized approaches show promise, particularly in neurodegenerative disorders such as Alzheimer's, Parkinson's, and multiple sclerosis, whose prevalence is expected to rise. Integrating modern molecular tools and multidisciplinary research may improve patient outcomes and revolutionize treatment strategies for these challenging conditions. [33, 34]

Innovative approach in neurodegenerative medicine

1. Stem cell therapies

Conventional pharmaceutical treatments for neurodegenerative disorders have disastrous side effects, and as of right now, stem cell therapy is arguably the only possible treatment option that provides a "cure" for these conditions. Animal models are used in the great majority of investigations. As a result, nothing is known about the long-term safety and clinical results of stem cell therapy in people with neurodegenerative illnesses. Before extrapolating the structural and functional gains shown in animals to humans, more research is necessary. Compared to HD and AD, there are comparatively more studies on PD and ALS, two of the four categories of neurodegenerative disorders mentioned above. A number of problems must be resolved before stem cell treatment is used clinically in neurodegenerative illnesses. [35]

2. Gene therapy

Gene therapy, once a taboo subject, is now a reality. Gene therapy was once thought to be incompatible with the extensive pathology of neurodegenerative disorders, but recent developments in vector technology have made it possible to diffusely transfer genes into the central nervous system. [36] Gene-based medicines, when paired with modern genome-manipulation techniques, hold the potential to change the therapeutic management of neurodegenerative diseases in the future. These are debilitating conditions for which there are now few or no disease-modifying treatments. [37] Although there are still many obstacles to overcome, the advent of this new era will depend heavily on the methodical creation of gene-delivery vectors, a thorough assessment of the security of modern gene-manipulation technologies, openness, and collaboration amongst diverse stakeholders. [38]

3. Neuroprotective agents

Kaempferol and its derivatives exhibit neuroprotective effects in neurodegenerative diseases (NDDs) through multiple mechanisms. [39] They inhibit Aβ deposition in Alzheimer's disease (AD) and α-synuclein aggregation in Parkinson's disease (PD), while promoting dopamine release and improving motor dysfunction. Kaempferol regulates inflammation-related pathways (e.g., NF-κB, MAPK, NLRP3 inflammasome) and reduces pro-inflammatory cytokines (IL-1β, IL-6, IL-18, TNF-α). Its antioxidant and anti-apoptotic properties reduce ROS production, enhance SOD and GSH activity, lower MDA levels, and regulate apoptosis-related proteins like Bcl-2. [40] Despite promising potential, clinical application faces challenges, including unclear mechanisms of action, reliance on limited animal and *in vitro* studies, and the need for advanced delivery systems. [41] Green nanomaterials functionalized with targeting ligands or mitochondrial-targeting properties could enhance kaempferol's therapeutic effects. Further research on its pharmacological mechanisms, safety, and efficacy *in vivo* is crucial to unlock its potential in NDD treatment. [42]

4. Targeting misfolding protein

Neurodegenerative diseases are primarily characterized by the misfolding, aggregation, and accumulation of proteins, leading to toxic protein entities that disrupt cellular function, damage synaptic connections, and contribute to brain atrophy. [43] These protein aggregates interfere with neurogenesis and deposit in various cellular compartments, such as the nucleus, cytoplasm, cell membrane, and extracellular spaces. Key proteins involved include β-amyloid, tau, and α-synuclein, which share similar misfolding and aggregation processes despite their distinct functions. [44] The misfolding process typically involves the formation of β-sheet-rich structures through hydrogen bonding and hydrophobic interactions, leading to the creation of oligomers, protofibrils, and fibrils. The aggregation mechanism follows the "seeding-nucleation" model, consisting of two phases: the slow nucleation phase, where oligomeric units form, and the rapid elongation phase, where monomeric proteins are incorporated into the growing polymer. [45] The lag phase is critical, as it generates small amounts of oligomers that act as seeds for the subsequent exponential growth. Excess seeding, often caused by polymer fragmentation, accelerates the elongation phase. Oligomers and fibrils are both important in aggregation, with fibrils being more resistant to clearance, contributing to their persistence in the disease process. [46, 47]

Conclusion

In conclusion, neurodegenerative diseases (NDDs) remain a significant challenge in both clinical and research fields, characterized by the progressive accumulation of misfolded proteins that lead to neuronal dysfunction and cell death. Key proteins like β-amyloid, tau, and α-synuclein play central roles in the pathological processes, undergoing misfolding and aggregation into toxic oligomers, protofibrils, and fibrils. The misfolding cascade follows the "seeding-nucleation" model, where the initial formation of oligomers triggers rapid aggregation, which is crucial for the

development of these diseases. While these molecular mechanisms are well-established, therapeutic strategies to halt or reverse the progression of NDDs remain limited. Current treatments primarily focus on symptomatic management, with no definitive cure available.

The growing body of research emphasizes the need for novel approaches to tackle the root causes of these diseases, including targeting protein misfolding and aggregation. Recent advancements in molecular medicine, particularly in the fields of genomics, proteomics, and systems biology, hold promise for developing more effective, personalized treatments. The integration of genetic information, environmental factors, and disease biomarkers offers the potential to revolutionize patient care, enabling therapies tailored to individual genetic profiles.

Despite progress, significant challenges remain. There is a need for more extensive studies using animal models and clinical trials to better understand the full scope of protein aggregation and its impact on neurodegenerative diseases. Additionally, improving drug delivery systems, such as nanotechnology-based approaches, is crucial for enhancing the efficacy of treatments. In conclusion, while the road to effective therapies for NDDs is still long, ongoing research and innovations in personalized medicine provide hope for better management and potential breakthroughs in the treatment of these debilitating disorders.

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