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The Many Faces of Parkinson's: A Holistic Scientific Review

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Abstract

Introduction and Definition: Parkinson's disease (PD) is a "neurodegenerative disorder characterized by early and marked loss of dopaminergic neurons," leading to abnormal motor presentation, including bradykinesia, muscular rigidity, rest tremor, and postural and gait impairment[1]. The primary cause of these Parkinsonian symptoms is the insufficient production of dopamine, predominantly resulting from the degeneration of dopamine-producing neurons in the basal ganglia.

Body: First systematically documented by James Parkinson in 1817 and later refined by Jean-Martin Charcot; this condition affects approximately 10 million people worldwide, and about 90,000 new cases are diagnosed annually in the United States. PD manifests itself in both classical forms with Parkinsonian motor impairments, and in atypical variants with diverse symptom profiles. The study of protein aggregation in PD began with Friedrich Lewy's identification of Lewy bodies in 1912. This protein aggregation was linked to inherited genetic mutations in genes synuclein alpha (*SNCA*) and leucine-rich repeat kinase 2, which 15% of PD cases are attributed to. Most cases, however, have no identifiable cause and are believed to result from a combination of genetic susceptibility, environmental factors, and pathological protein misfolding and aggregation. These aggregates cause oxidative stress, mitochondrial dysfunction, and neuronal death. Recent research highlights the spread of α-synuclein (α-syn) aggregates across neural networks, emphasizing genetic predispositions, environmental toxins, and impaired protein clearance mechanisms as critical contributors to disease progression. Small-molecule inhibitors, immunotherapies targeting α-syn, and gene-editing technologies such as clustered regularly interspaced short palindromic repeats-associated protein 9 (CRISPR-Cas9) are being researched as potential treatments and therapies for PD. Advancements in neuroimaging and biomarker discovery also represent crucial steps for earlier detection and improved patient management. Other ongoing research aims to detail aggregation pathways, identify reliable biomarkers, and validate novel therapeutic strategies in clinical trials, ultimately striving to improve and transform disease management to significantly enhance patient outcomes.

Keywords: α-synuclein; bradykinesia; dopamine; gait; Lewy bodies; Parkinson's disease; substantia nigra pars compacta; tremor

Introduction and Definition

Parkinson's disease (PD) is a "neurodegenerative disorder characterized by early and marked loss of dopaminergic neurons," with hallmark symptoms such as muscular rigidity, rest tremor, and postural and gait impairment [1]. Beyond its motor symptoms, PD is also associated with a wide spectrum of non-motor manifestations, including cognitive decline and autonomic dysfunction. Classified under the broader category of neurodegenerative diseases that include Alzheimer's disease and amyotrophic lateral sclerosis , these disorders are characterized by the gradual structural and functional loss of neurons in the brain, processes increasingly linked to protein misfolding, mitochondrial dysfunction, and oxidative stress [2]. Although PD is classified as an idiopathic condition, its strong association with degeneration in the substantia nigra pars compacta (SNpc), a midbrain region that provides dopamine to the basal ganglia, was first described by Edward Brissaud in 1893. Subsequent research revealed that dopamine depletion in the basal ganglia correlated with disturbances in cognitive function [3]. In 1912, Fredric Lewy identified abnormal protein aggregates, later termed Lewy bodies, in the brainstems of Parkinson's patients [4]. Building on that discovery, Maria Grazia Spillantini demonstrated that Lewy bodies were primarily composed of α-synuclein (α-syn), illuminating the underlying neuropathological process of the disease [5].

Body

Public Health Burden of PD

PD is currently one of the fastest-growing neurological disorders. In 2021, approximately 11.8 million individuals worldwide were living with PD, a stark increase from 3.2 million in 1990, with projections estimating a continuous

upward trend as age and exposure to environmental risk factors continue [6]. This economic burden of PD encompasses direct medical costs and indirect and non-medical costs. In the United States alone, the annual cost of PD in 2017 was $25.4 billion in medical costs and $26.5 billion in indirect and non-medical costs [7]. Even without accounting for productivity lost by individuals with PD, family caregivers, and volunteers, the burden of PD is globally substantial. Most significantly, this burden is not distributed equally: individuals in low- and middle-income countries and communities often lack access to timely diagnosis and treatment, exacerbating disparities in disease management and outcomes [8].

Current public health initiatives focus on awareness, legislative regulation of known neurotoxicants, and early screening in those with a family history of PD.

Discovery of PD

While PD was formally defined in 1817 by James Parkinson in *An Essay on the Shaking Palsy*, symptoms such as tremor and shuffling gait have long been described in ancient medical texts across various cultures [9–11].

Around 1000 Before Common Era (BCE), Indian Ayurvedic texts described *Kampavata*, a condition marked by tremor, stiffness, and drowsiness. Similar descriptions of involuntary tremors and abnormal muscle movements appear in the *Yellow Emperor's Internal Classics* (425-221 BCE) and the *Akkadian Diagnostic Handbook (1068-1046 BCE)* [12]. Although these early writings captured key features of PD, Parkinson provided the "first systematic clinical definition, describing it as:

"Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace: the senses and intellects being uninjured [9]."

In 1872, Jean-Martin Charcot expanded upon Parkinson's work by emphasizing tremor, rigidity, and bradykinesia as core symptoms. Charcot distinguished PD from other neurological disorders, such as multiple sclerosis, and introduced the eponym "Parkinson's disease."

Significant anatomical and pathological insights emerged after 1899, when Édouard Brissaud first proposed that PD originated from damage to the SNpc region [13]. This was later confirmed by the identification of Lewy bodies–abnormal protein aggregates in SNpc–by Frederick Lewy (1912) and named by Konstantin Tretiakoff (1919)[6, 14]. In 1960, Oleh Hornykiewicz discovered that dopamine loss in the striatum, rather than serotonin or noradrenaline loss, was the primary neurochemical cause of PD. This finding laid the groundwork for dopamine replacement therapies [15]. Likewise, over the past two centuries, advances in the understanding of PD have shaped current efforts to treat and potentially cure one of the most prevalent neurodegenerative disorders.

Symptoms and Progression

Since PD lacks a definitive diagnostic test, diagnosis relies on recognizing key motor symptoms: bradykinesia, tremor, and rigidity. Bradykinesia involves depression in spontaneous movement, difficulty with fine motor skills, monotone speech, decreased facial expression, blinking, and shuffling gait. Tremors and rigidity appear as resistance to passive movement and stiffness, causing jerky motion [16, 17]. Although PD is primarily defined by its motor symptoms, it frequently presents with non-motor deficits as well, including sleep disturbances, psychosis, dementia, and orthostatic hypotension [18].

In 1967, Hoehn and Yahr introduced a simple five-stage scale to describe the progression of PD [19, 20]:

Stage 1: unilateral involvement
Stage 2: bilateral involvement with balance impairment
Stage 3: Postural instability
Stage 4: Severe disability; still physically independent
Stage 5: Wheelchair-bound or bedridden

While the scale emphasizes posture instability and overlooks other motor symptoms and non-motor features, it remains a widely used standard for staging and monitoring PD [21]. A more frequently used diagnostic criterion for PD is the Unified Parkinson's Disease Rating Scale (UPDRS), encompassing 4 main criteria [22]:

Mentation, Behavior, and Mood: a clinician-rated assessment of cognitive impairment, thought disorder, depression, and motivation.

Activities of Daily Life: a self- or informant-rated assessment of daily functions (e.g., cutting food, hygiene, dressing).

Motor Examination: a clinician-rated assessment of motor signs (e.g., tremors, postural stability, gait).

Complications of Therapy: general assessment of adverse effects of long-term dopaminergic treatments.

Specific items of assessment are scored on a scale of 0 (normal) to 4 (severe impairment or disability). The UPDRS has undergone many revisions, and the Movement Disorder Society's version is most widely used now, retaining the strengths of the original version, while resolving critiqued elements, such as the lack of emphasis on non-motor features of PD [23].

Pathogenesis

PD is primarily characterized by the progressive loss of dopaminergic neurons in the SNpc, the brain region responsible for motor control [24]. On the molecular level, PD is most associated with Lewy bodies observed in the brain, which are α-syn protein aggregates clumping at the SNpc region [25].

Although most PD cases are idiopathic, approximately 5–10% are attributed to monogenic mutations. The first genetic link was identified as a point mutation in the synuclein alpha (SNCA) gene, which encodes α-syn [26]. Normally, α-syn regulates neurotransmitter release at

presynaptic terminals, but it will misfold into β-sheet-rich oligomers under pathological conditions. These aggregates, which form the core of Lewy bodies, are linked to the cellular damage and death in PD [26]. Genetic forms of PD involve mutations that disrupt key processes such as mitochondrial quality control (e.g., Parkin RBR E3 ubiquitin protein ligase, PTEN-induced kinase, Parkinsonism-associated deglycase), protein handling (e.g., SNCA, leucine-rich repeat kinase 2, glucosylceramidase beta 1), and lysosomal function, ultimately promoting oxidative stress and α-syn aggregation. In parallel, neuroinflammation driven by activated glia amplifies neuronal injury, highlighting the multifactorial nature of PD pathogenesis [25, 27].

Causes and Risk Factors

While the PD remains idiopathic, altogether, PD can no longer be viewed merely as a dopamine deficiency disorder. It represents a convergent cascade of pathologies involving protein misfolding, bioenergetic failure, cellular stress, and immune dysregulation across the nervous system. Although several genes have been correlated with PD, many cases arise without identifiable genetic mutations. This suggests that environmental factors may also contribute to the disease etiology, either independently or in combination with genetic influences. PD incidence increases steadily after age 45 and accelerates with each decade, making age the most significant risk factor[28, 29]. Traumatic brain injury and exposure to toxicants such as herbicides and pesticides are also associated with increased risk [28].

Therapeutics and Future Perspectives

Even though there is no cure for PD, there are several treatment methods that aim to slow the progression of the disease and lessen the symptoms. The most prescribed frontline drug for PD is Levodopa, a drug that crosses the blood-brain barrier and works in the striatum to be converted into dopamine and alleviate dopamine deficiency. Furthermore, inhibitors to block enzymes that break down dopamine are also often prescribed in conjunction with Levodopa [30]. Symptom management can also be done with anticholinergic drugs that are used to reduce tremors and dystonia.

Alternatively, several invasive and non-invasive treatment options are emerging. Among the invasive techniques, deep brain stimulation (DBS) involves implanting electrodes into the brain, connected to a pulse generator, to stimulate either the subthalamic nucleus or the globus pallidus internus [31]. DBS has been shown to significantly reduce tremors and motor fluctuations [32]. Among non-invasive techniques, magnetic resonance–guided high-intensity focused ultrasound ablates targeted brain tissue and has shown potential as a treatment option [33]. In contrast, low-intensity focused ultrasound uses ultrasound waves to modulate brain activity and is currently under investigation [34].

Another promising approach is the use of induced pluripotent stem cells (iPSCs), reprogrammed from adult somatic cells and differentiated to dopaminergic neurons, offering a patient-specific therapy. iPSC-derived neurons have demonstrated long-term survival and motor function restoration in preclinical models [35]. Clustered regularly interspaced short palindromic repeats-associated protein 9 (CRISPR-Cas9) gene editing has also opened new potential for disease-modifying therapies, successfully correcting PD-related mutations in genes when used in conjunction with stem cell therapy in various animal models [36]. PD presents in subtypes—tremor-dominant, akinetic-rigid, and postural-instability/gait-difficulty—that differ in symptoms, progression, and neurobiology. Neuroimaging studies show distinct circuit alterations across these forms, underscoring the need for such personalized therapies and clinical trials that integrate multimodal imaging and genetic profiling to guide precision treatment [37].

Artificial Intelligence, Machine Learning, AlphaFold, and Parkinson's disease

Recent advancements in Artificial Intelligence (AI)-driven protein 3D structure prediction technologies, such as AlphaFold, as well as Machine Learning (ML) directed DBS, are promising towards the advancement of technologies that could treat PD. However, ML still has its limitations. Figure 1 is the known tertiary structure of human micelle-bound α-syn imaged by nuclear magnetic resonance (NMR). When AlphaFold was provided the sequence of human micelle-bound α-syn, it predicted a structure with one elongated helix, rather than two helices connected by a turn (Figure 2). These evident disparities in predicting wild-type tertiary structure are indicative of the limitations still present in AI structure-prediction technology.

Moreover, ML is experimentally employed to enhance surgical targeting for DBS and to predict patient outcomes pre- and post-operation [38]. ML directed DBS enables adaptive, closed-loop systems that adjust stimulation in real time based on neural signals, aiming to improve efficiency and reduce side effects.

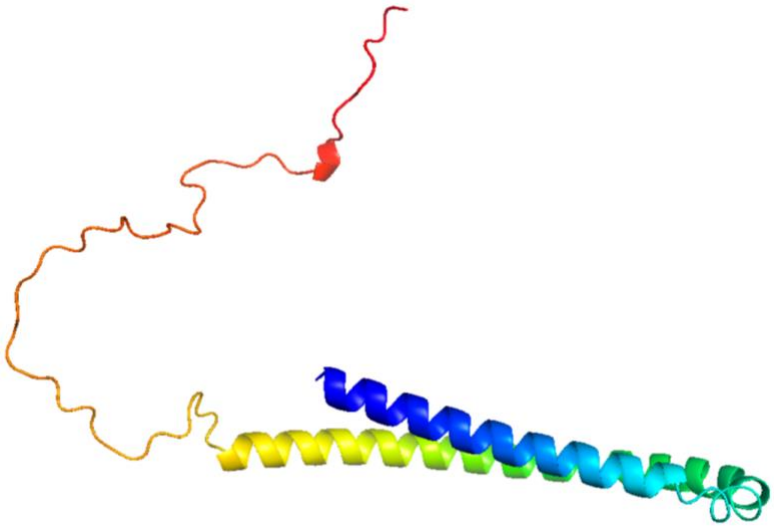

Figure 1. The Tertiary Structure of Human micelle-bound α-syn (PDB 1XQ8) modeled on PyMol, using known sequences and NMR structures of native (wild-type) α-syn [39].

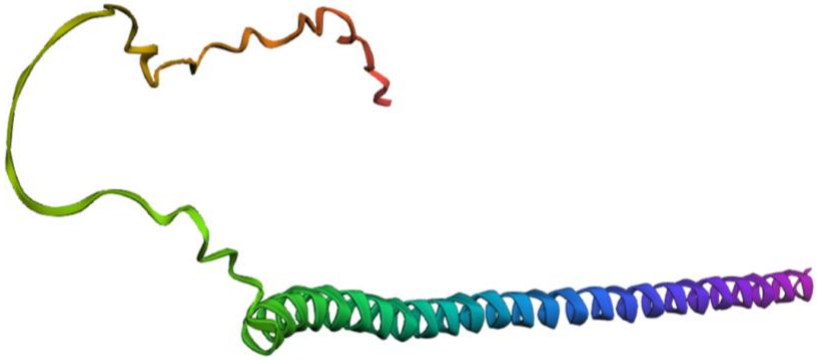

Figure 2. The predicted Tertiary Structure of Human micelle-bound α-syn modeled by AlphaFold, using known sequences and structures of native (wild-type) α-syn [40].

Conclusion

PD remains a fast-growing, multifaceted disease that challenges the scientific community and poses a significant burden on public health. From its early clinical descriptions to the discovery of PD's linkage to dopamine deficiency and genetic mutations, researchers have revealed PD to be more than a simple deficit. The modern-day view of PD encompasses a complex interplay of protein aggregation, mitochondrial dysfunction, genetic predisposition, and environmental exposures that highlights the need for continuous interdisciplinary research. While traditional therapies center around symptom relief, promising new technologies, including stem cell therapy, gene editing, and AI-driven neurostimulation, offer the potential for disease-modifying and even restorative interventions. The integration of neuroscience, biotechnology, and AI signals a transformative shift toward personalized, preventive, and regenerative care, redefining how PD could be managed in the future.

List of Abbreviations

AI: artificial intelligence
BCE: before common era
CRISPR-Cas9: clustered regularly interspaced short palindromic repeats - associated protein 9
DBS: deep brain stimulation
iPSCs: induced pluripotent stem cells
ML: machine learning
NMR: nuclear magnetic resonance
PD: Parkinson's disease
PDB: protein data bank
SNCA: synuclein alpha
SNpc: substantia nigra pars compacta
UPDRS: unified parkinson's disease rating scale
α-syn: α-synuclein

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Authors' Contributions
HH, JJP, and RES: substantially contributed to the study's design, conducted secondary research, performed fact-checking, and drafted the manuscript.
RES: produced the AlphaFold figures.
JJP: critically revised the manuscript and organized citations.
HH: critically revised the manuscript and provided final approval of the version to be published.

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