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MEMORY AND LEARNING DEFICIENCIES IN PARKINSON'S DISEASE: UNDERSTANDING COGNITIVE CHALLENGES AND INTERVENTIONS

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Abstract:

Parkinson's disease (PD) is a progressive neurodegenerative disorder primarily characterized by motor symptoms and cognitive impairments, particularly in memory and learning. These impairments significantly affect the patients' quality of life and people around them. Based on the past studies, aging, genetics and environment are expected to be some of the contributing factors in Parkison's disease. Cognitive impairments in PD commonly caused by neurochemical or structural changes in the brains, which decrease dopaminergic neurons in substantia Nigra pars compacta (SNpc). Furthermore, this paper explores various cognitive domains including memory, learning and other executive functions in order to understand the extent of impairment that is experienced by people with Parkinson's disease. However, despite the extensive study on PD, current interventions demonstrate lack of effectiveness in eradicating the issue of cognitive deficiency especially on learning and memory impairment. Hence, the paper discusses current interventions by addressing and applying the reviewed literature, ranging from pharmacological treatments to lifestyle modifications aimed at mitigating cognitive decline. This study also provides a comprehensive overview of suggestions for future research about PD. Lastly, from Islamic perspective, there is a hadith that encourages seeking medical treatment while integrating it with spiritual practices, such as Qur'anic recitation, to alleviate cognitive impairments. Listening to, reciting, and memorizing the Qur'an have been shown to enhance concentration, promote neuroplasticity, delay cognitive aging and improve memory, which can help mitigate the effects of PD.

Keywords:

Parkinson's Disease, Cognitive Impairment, Memory, Learning, Dopamine, Treatments

Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disorder of aging. The term "progressive" means that the condition gets worse over time, where it begins with mild symptoms and advances to more severe motor and cognitive impairments. "Neurodegenerative" refers to the loss of nerve cells in the brain, and disorder of aging due to PD is more common in older people affecting approximately 1% of individuals over the age of 60 making it the second most common neurodegenerative disorder after Alzheimer's disease (Malaysian Parkinson's Disease Association, 2022). Individuals with PD experience motor symptoms such as shaking (tremors), muscle stiffness (rigidity), slowness of movement (bradykinesia), and balance problems (postural instability). Additionally, PD significantly affects non-motor functions, particularly cognitive impairment, such as memory and learning impairments. Specifically, episodic memory, which is the memories of day-to-day life events, is often impaired in PD patients (Das et al., 2019) and will significantly impact patients' quality of life (Elkurd & Dewey, 2020).

Dysfunctions in the hippocampus, part of the brain structure responsible for memory processing and learning lead to cognitive impairments. Parkinson's disease impacts hippocampal subfields such as CA1, CA2-3, and subiculum, which are highly correlated with learning and recognition (Das et al., 2019). These subfields play crucial roles in episodic memory recollection and processes such as pattern separation, which separates similar events

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into distinct memories, and pattern completion, which aids in recollection even with insufficient input. These impairments disrupt encoding, storing, and retrieving memories, leading to unsuccessful recollection. Episodic memory deficits in PD involve challenges with both recollection (retrieval of previously encountered events with contextual details) and familiarity (sense of recognition that an event has been previously encountered). This severely hindered patients' ability to recall past experiences and learn new information.

The progressive nature of cognitive decline in PD often leads to transitions from mild cognitive impairment to Parkinson's disease dementia (Elkurd & Dewey, 2020). This decline significantly impacts patients' daily activities and quality of life, adding to the difficulties and responsibilities placed on caregivers. Individuals with advanced PD often become highly dependent on their caregivers due to reduced physical and cognitive capacities. There are higher rates of caregivers feeling burdened due to individuals with advanced PD becoming highly reliant on them for support, increased physical dependence, and their ability to complete daily activities have reduced (Jensen et al., 2021). These challenges underscore the urgent need for effective interventions targeting cognitive impairments in PD, not only to improve learning outcomes and memory functions for patients but also to alleviate caregiver burden. This paper aims to explore underlying mechanisms that cause memory and learning impairments for PD while also discussing interventions that may mitigate these effects.

Methodology

A comprehensive literature compilation was executed for this review utilizing the online academic search engine. For the search strategy, a few related keywords were used, including "Parkinson's disease on cognitive impairment," "memory and learning impairment," "cognitive deficits" and "physiological mechanism." Moreover, the academic databases used were Google Scholar, Oxford Academic database, MDPI database and Society for Neuroscience Journal. For specific search results, the Boolean operators (AND, OR) were utilized.

Inclusion and Exclusion Criteria

All papers selected for this review were published between 2020 to 2025 with one article dated back to 2015. To limit the scope of review, only papers with original findings, case studies, meta-analysis and systematic reviews were included in this study. Other literatures were excluded. Additionally, papers published in languages other than the English language were excluded to ensure critical understanding of the topic.

Data Extraction

The data extraction was conducted using a standardized form. This form aimed to synthesize several key importance in each paper namely, the participant details, findings, limitations and future directions. The data was then compared and discussed in a meaningful narrative.

Findings

Across recent reviews and longitudinal cohorts, cognitive impairment is now recognised as a common and clinically meaningful feature of Parkinson's disease (PD), occurring along a continuum from subtle cognitive changes to PD–mild cognitive impairment (PD-MCI) and Parkinson's disease dementia (PDD). Large syntheses consistently place PD-MCI prevalence at roughly a quarter of patients overall, including at or near diagnosis, although estimates vary by cohort characteristics, diagnostic criteria, and disease stage (Aarsland et al., 2021; Jellinger,

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2024; Yu et al., 2022). Ten-year follow-up studies similarly show substantial cumulative dementia risk, but with heterogeneity in timing and trajectories—highlighting that dementia occurs later in some modern cohorts than older reports suggested (Gallagher et al., 2024). These findings support a "multiple pathways" model of cognitive decline in PD, rather than a single uniform mechanism.

Genetic findings show that PD susceptibility and prognosis reflect both rare pathogenic variants (monogenic forms) and numerous risk loci. Contemporary genetic reviews indicate that a sizeable minority of patients carry PD-associated pathogenic variants, and that the genetic background can influence phenotype, progression rate, and non-motor outcomes, including cognition (Trevisan et al., 2024). Among currently known genes, **GBA1** has the clearest and most consistently replicated relationship with earlier and faster cognitive decline and higher dementia risk, compared with non-GBA1 PD (Blazekovic et al., 2024; Fernández-Vidal et al., 2024; Lanore et al., 2025). By contrast, **LRRK2-PD** is often associated with comparatively less cognitive involvement than idiopathic PD or GBA1-PD, although variability remains and subtyping is important (Thaler et al., 2024). Beyond GBA1 and LRRK2, evidence is emerging—yet still mixed—for additional genetic contributors to cognitive outcomes (including APOE-related effects and polygenic influences), suggesting that dementia risk is likely shaped by multi-gene, multi-pathway processes rather than single variants in most patients (Blazekovic et al., 2024; Faouzi et al., 2024). For recessive early-onset genes (e.g., **PARK7/DJ-1**), recent mechanistic and clinical literature supports roles in mitochondrial stress responses, autophagy, and neuroinflammation, but direct, generalisable links to later cognitive decline in typical late-onset PD remain less firmly established and warrant targeted longitudinal profiling (Skou et al., 2024; El Otmani et al., 2025).

Environmental findings increasingly strengthen the view that **modifiable exposures** contribute to PD risk and may interact with biological vulnerability. Critical reviews emphasise associations between PD and long-term exposure to pesticides and industrial solvents (notably trichloroethylene, TCE), while also stressing persistent methodological gaps—especially exposure misclassification, insufficient lag-time modelling, and confounding (Atterling Brolin et al., 2024). More recent work continues to refine exposure–disease links (including ambient TCE exposure analyses), supporting the plausibility of solvent-related neurodegenerative risk and highlighting the need to integrate environmental data into prevention and early-detection strategies (Neurology, 2025; Atterling Brolin et al., 2024). Importantly, these environmental associations align with mechanistic pathways central to PD biology—oxidative stress, mitochondrial dysfunction, lysosomal impairment, neuroinflammation, and α-synuclein aggregation—suggesting that environmental toxicants may accelerate pathology particularly in genetically susceptible individuals (Aarsland et al., 2021; Atterling Brolin et al., 2024; Skou et al., 2024).

Neuropathological findings indicate that cognitive impairment in PD reflects **distributed brain pathology**, not only nigrostriatal dopamine loss. While α-synuclein (Lewy) pathology remains central, the clinical expression of cognitive decline is increasingly linked to (i) the extent and topography of cortical Lewy pathology and (ii) the presence of **Alzheimer's disease (AD) co-pathology** (amyloid-β and tau). A recent systematic review in Lewy body disorders concluded that amyloid and tau co-pathology is associated with worse clinical outcomes, including faster cognitive decline and increased mortality, supporting the clinical observation that "mixed pathology" phenotypes often progress more aggressively (Tan et al., 2024).

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Parallel biomarker work further supports this, with plasma tau markers (e.g., p-tau217) increasingly investigated as indicators of AD co-pathology in PD-MCI/PDD subgroups, reinforcing that cognitive decline in PD may be accelerated by concomitant AD-type processes in a substantial minority of cases (Musso et al., 2025).

Neurochemical findings broaden the mechanistic picture beyond dopamine. Dopaminergic dysfunction remains strongly linked to frontostriatal/executive deficits and can produce task-dependent effects on cognition; however, converging evidence indicates that **cholinergic degeneration**, particularly involving basal forebrain cholinergic nuclei (including Ch4/nucleus basalis of Meynert), is a key substrate for attentional and memory decline and for progression toward dementia (Aarsland et al., 2021). Multi-modal imaging and network studies show that basal forebrain integrity and cholinergic denervation are associated with cognitive impairment and may interact with hippocampal/medial temporal structures, supporting models in which memory outcomes reflect both cholinergic input loss and vulnerability of limbic circuitry (Crowley et al., 2024; Horsager et al., 2022; Sakamaki-Tsukita et al., 2024). Recent work specifically proposes that hippocampal involvement may mediate the relationship between basal forebrain atrophy and memory decline, especially in PD-MCI/PDD trajectories, underscoring the importance of studying "system-level" degeneration rather than isolated regional atrophy (Sakamaki-Tsukita et al., 2024).

Cognitive profile findings align with the notion that **executive dysfunction often appears earlier** than prominent episodic memory impairment, and that memory problems in PD frequently reflect retrieval/strategy and attentional control limitations rather than a primary encoding deficit—at least in earlier stages. This pattern is consistent with broader clinical characterisations of PD cognitive impairment that emphasise executive, attentional, and visuospatial vulnerabilities, with memory deficits often becoming more severe as cholinergic and posterior cortical involvement increases (Aarsland et al., 2021; Jellinger, 2023). Clinically, brief bedside tasks (e.g., clock drawing, serial months backward) remain useful as screening indicators of executive/attentional inefficiency, though interpretation must consider motor and processing-speed constraints and the overlap of cognitive domains (Aarsland et al., 2021).

Therapeutic findings remain mixed, with the most consistent symptomatic evidence supporting cholinesterase inhibitors for cognition-adjacent outcomes (and neuropsychiatric symptoms) in Lewy body spectrum conditions, while acknowledging modest effect sizes and trial design limitations. An individual patient data meta-analysis reported improvements in delusions and hallucinations with cholinesterase inhibitors in Alzheimer's disease and Parkinson's disease cohorts, which is clinically relevant given the frequent coupling of cognitive and neuropsychiatric deterioration in PDD (d'Angremont et al., 2023). Broader clinical-trial reviews of cognition in PD conclude that few interventions show robust, sustained cognitive benefits, and that future studies need sharper phenotyping (e.g., PD-MCI subtypes), better biomarkers (including cholinergic and amyloid/tau measures), and stage-matched outcome measures to avoid masking real but subgroup-specific effects (Bayram et al., 2023).

Discussion

Genetic and Environmental Risk Factors

Cognitive impairment in Parkinson's disease (PD) emerges from a multifactorial risk architecture in which genetic susceptibility interacts with cumulative environmental exposure.

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Among genetic contributors, *GBA1* variants are consistently associated with a higher risk of faster cognitive decline and dementia-related trajectories, with evidence that cognitive phenotypes may involve both posterior-cortical and fronto-striatal domains (Fernández-Vidal et al., 2024; Koros et al., 2024). Contemporary guidance also emphasises that *GBA1* status has practical implications for counselling and prognostication in PD, especially when cognitive decline is a clinical concern (Vieira et al., 2024).

Environmental risks remain highly salient, particularly pesticides, industrial solvents, and air pollution, which may accelerate pathogenic cascades relevant to PD onset and progression (Atterling Brolin et al., 2024; Dorsey et al., 2025). Trichloroethylene (TCE) has received renewed attention as a potentially preventable contributor to PD risk, with recent epidemiological work examining population-level associations with PD (Krzyzanowski et al., 2025). Taken together, current evidence supports a prevention-oriented framing of PD risk alongside the traditional neurodegeneration model, while acknowledging that individual-level causal pathways remain difficult to specify and likely heterogeneous across settings and populations (Atterling Brolin et al., 2024; Dorsey et al., 2025).

Physiological and Neuropathological Mechanisms

The mechanistic pathway toward cognitive decline is best understood as a sequence from neuropathology to neurochemical disruption, then to network-level dysfunction and observable cognitive-behavioural outcomes. PD neuropathology classically involves nigrostriatal dopaminergic degeneration and α-synuclein aggregation (Lewy pathology), but cognitive impairment is often shaped by wider cortical and limbic involvement and by multi-pathology burden (Aarsland et al., 2021). Importantly, concomitant Alzheimer-type pathology (amyloid-β and tau) can modify clinical trajectories in Lewy body–spectrum disorders and plausibly contributes to more rapid or severe cognitive decline in susceptible subgroups (Tan et al., 2024).

At the neurotransmitter level, dopamine depletion contributes strongly to executive dysfunction and fronto-striatal inefficiency, whereas cholinergic dysfunction is closely linked to attentional and memory impairment and is a key substrate for dementia-level decline in PD (Aarsland et al., 2021). This multi-system profile helps explain why "motor-first" models are insufficient for predicting learning and memory outcomes, and why interventions targeting only dopaminergic symptoms often yield inconsistent cognitive benefits (Aarsland et al., 2021).

Cognitive Impairments and Memory Deficits

A prominent clinical implication of the above cascade is that cognitive deficits in PD are rarely unitary. Executive dysfunction (planning, set-shifting, inhibition) often reflects fronto-striatal disruption, while memory problems may arise from impaired retrieval strategies, attentional control during encoding, and disrupted medial temporal connectivity—rather than from pure amnestic encoding failure (Aarsland et al., 2021). This aligns with the view that PD-related memory difficulties frequently improve with cues, consistent with retrieval and strategic deficits rather than the encoding profile typical of Alzheimer's disease (Aarsland et al., 2021). In learning domains, motor learning deficits may reflect altered nigrostriatal function and abnormal motor-network connectivity, yet the precise contributions of substantia nigra substructures and their downstream targets remain difficult to disentangle with standard-resolution imaging (Tzvi et al., 2021). This uncertainty reinforces the need to interpret

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"learning deficiency" in PD as a neurocognitive network problem, not merely as a motor execution limitation (Tzvi et al., 2021).

Interventions for Learning and Memory Deficits

Despite major advances in motor symptom management, disease-modifying treatments for cognitive impairment remain limited. Comprehensive reviews continue to highlight that no therapy has definitively prevented or delayed PD cognitive decline; cholinesterase inhibitors remain the main approved option for Parkinson's disease dementia (PDD), with limited and variable benefits across domains (Aarsland et al., 2021). This gap makes it essential to separate interventions into pharmacological, non-pharmacological, and integrative approaches, and to interpret effectiveness as likely stage-dependent and phenotype-dependent rather than uniform.

Pharmacological Interventions (Symptomatic and Neuroprotective Candidates).

Dopaminergic therapy remains foundational for motor symptoms and may improve certain learning-related outcomes under specific task demands and disease stages; however, cognitive effects can be inconsistent and may differ across executive versus memory processes (Kalia & Lang, 2015; Paul et al., 2020). Beyond symptomatic dopaminergic strategies, neuroprotective candidates are increasingly explored. GLP-1 receptor agonists have been examined as potential disease-modifying or symptom-modifying agents, with a phase 2 trial of lixisenatide in early PD suggesting slowed motor disability progression, though cognitive endpoints require clearer and longer-term evaluation (Meissner et al., 2024). Updated meta-analytic work suggests GLP-1 receptor agonists may improve motor and cognitive performance on some measures, while emphasising that evidence remains limited and heterogeneous (de Albuquerque et al., 2025; Tsai et al., 2025). These findings are promising but do not yet establish robust stage-specific cognitive protection, underscoring the need for longer trials with cognition as a primary endpoint (de Albuquerque et al., 2025; Tsai et al., 2025).

Non-Pharmacological Interventions (Exercise, Cognitive Training, And Task-Based Rehabilitation).

Exercise is among the most consistently supported non-pharmacological approaches, with updated systematic reviews and meta-analyses indicating improvements in global cognition and more modest effects on executive function in PD (Kim et al., 2023). Neuroimaging meta-analytic evidence also suggests exercise may modulate activity within distributed cortical and cerebellar networks implicated in PD functional compensation, which provides a plausible mechanistic basis for cognitive and motor gains (Li et al., 2022).

Computerised cognitive training (CCT) and cognitive rehabilitation are increasingly supported, including when delivered remotely, with evidence that higher training dose and PD-MCI samples may show larger benefits (Gavelin et al., 2022). Recent digital and home-based programmes reinforce the feasibility of telerehabilitation models for cognitive symptoms, although larger trials are still required to clarify durability and which cognitive domains respond most reliably (Tagliente et al., 2025).

Technology-supported approaches are expanding rapidly. Virtual reality (VR) rehabilitation shows significant promise for PD rehabilitation, and recent meta-analytic work supports meaningful gains in core functional outcomes, though protocols vary substantially across studies (Fernandes et al., 2025; Kwon et al., 2023). Importantly, emerging work integrates immersive environments with cognitively demanding tasks, including executive-function-

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oriented paradigms and dual-task training frameworks, which may be especially relevant where cognitive-motor interference drives disability in daily life (Tan, 2024; Nartea et al., 2025).

Integrative And Neuromodulation-Adjacent Approaches.
A growing direction involves combining cognitive rehabilitation with non-invasive brain stimulation (NIBS), with recent comparative evidence suggesting that NIBS combined with cognitive rehabilitation may yield stronger cognitive benefits than single-modality approaches, while motor-cognitive rehabilitation may benefit emotional outcomes (Fan et al., 2025). This points to a pragmatic future in which interventions are selected based on target outcomes (cognition, emotional symptoms, daily function) rather than a one-size-fits-all model (Fan et al., 2025).

Across intervention categories, the main limitations remain consistent: small sample sizes, methodological heterogeneity, variable cognitive phenotyping, and limited long-term follow-up, especially in PD-MCI and early cognitive decline cohorts (Aarsland et al., 2021; Gavelin et al., 2022; Fernandes et al., 2025).

Future Research Directions
Future research should progress from mechanism to implementation. First, higher-resolution imaging and targeted basal ganglia protocols are needed to disentangle substantia nigra subregions and their connectivity contributions to learning impairment (Tzvi et al., 2021). Second, trials should stratify by cognitive phenotype (e.g., executive-dominant vs memory-dominant profiles), genetic risk (e.g., *GBA1* status), and co-pathology likelihood, given evidence that these factors shape cognitive trajectories and treatment response (Aarsland et al., 2021; Fernández-Vidal et al., 2024; Tan et al., 2024). Third, digital interventions (CCT, VR, dual-task training) should adopt common outcome frameworks and report domain-level cognitive endpoints with follow-up intervals that can test maintenance and generalisation to everyday functioning (Gavelin et al., 2022; Tagliente et al., 2025; Tan, 2024). Finally, participatory research that includes patients and caregivers can improve ecological validity and ensure that interventions address real-world priorities such as medication management, wayfinding, memory supports, and independence in instrumental activities of daily living (Aarsland et al., 2021).

Islamic Perspective and Spiritual Interventions
Islam frames illness as a trial accompanied by legitimate means of healing. The Prophetic hadith, narrated by Abu Huraira (RA), states: “There is no disease that Allah has created, except that He also has created its treatment” (Sahih al-Bukhari, Book 71, Hadith 582). Classical and contemporary scholarly explanations emphasise that seeking treatment is permissible and encouraged, while recognising that cure ultimately occurs by Allah’s will; this supports a balanced stance combining evidence-based care, hope, and *tawakkul* (reliance upon Allah) (Pejabat Mufti Wilayah Persekutuan, 2020).

From a scientific standpoint, Qur’anic engagement can be framed as a spiritually grounded practice with plausible neurophysiological correlates relevant to stress regulation and cognitive support. A recent systematic review of EEG studies reports that listening to Qur’anic recitation is commonly associated with increased alpha and theta power, patterns often linked to relaxation and improved emotional regulation, although study quality and heterogeneity remain important limitations (Majidi et al., 2025). Complementing this, a systematic review focusing

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on older adults found evidence supporting a positive association between Qur'an reading and cognitive function, highlighting the potential relevance of religious practices as culturally congruent supports for cognitive health in Muslim populations (Riviati et al., 2024).

In PD, where cognitive decline is intertwined with anxiety, sleep disruption, and chronic stress, such spiritually anchored practices may serve as adjunctive supports—particularly when integrated ethically and clinically (e.g., as part of routine, stress reduction, caregiver-supported engagement), rather than positioned as replacements for medical and rehabilitative care (Aarsland et al., 2021; Majidi et al., 2025; Riviati et al., 2024).

Conclusion

Cognitive impairment in Parkinson's disease arises from a complex interplay of widespread neuropathology and multi-neurotransmitter disruption, with individual trajectories further shaped by genetic susceptibility and environmental exposures (Aarsland et al., 2021; Atterling Brolin et al., 2024; Tan et al., 2024). Pharmacological options remain limited, offering at best modest symptomatic benefit in PDD, whereas exercise, cognitive rehabilitation, and emerging digital or VR-supported programmes appear promising but are still constrained by heterogeneity and limited long-term evidence (Fernandes et al., 2025; Gavelin et al., 2022). Future research should therefore prioritise biomarker- and phenotype-stratified trials with cognition-centred endpoints and meaningful functional outcomes, while also recognising the potential value of culturally congruent adjuncts—such as Qur'anic engagement for stress regulation and daily coping—integrated ethically alongside evidence-based care (Majidi et al., 2025; Pejabat Mufti Wilayah Persekutuan, 2020; Riviati et al., 2024)..

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