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The Role of Physical Activity in Parkinson's Disease: Neuroprotective Mechanisms and Clinical Implications

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Abstract

Background: Parkinson's disease is a progressive neurodegenerative disorder associated with motor and non-motor symptoms. Physical activity is increasingly recognized as a supportive intervention with potential neuroprotective and therapeutic benefits.
Aim: This review aimed to summarize the potential neuroprotective mechanisms and clinical benefits of physical activity in Parkinson's disease.

Results: Physical activity may improve motor function, gait, balance, physical capacity, selected non-motor symptoms, functional independence, and quality of life. These effects may be linked to neuroplasticity, mitochondrial function, reduced oxidative stress and neuroinflammation, and modulation of dopaminergic pathways.

Conclusions: Physical activity should be considered an integral part of Parkinson's disease management. Although its symptomatic and functional benefits are well supported, further studies are needed to confirm its long-term neuroprotective and disease-modifying effects.

Keywords: Parkinson's disease; physical activity; exercise therapy; neuroprotection; neuroplasticity; motor symptoms; quality of life.

1. Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disorder of the central nervous system and the second most common neurodegenerative disease worldwide after Alzheimer's disease.[1] With population ageing, the global number of individuals affected by PD is steadily increasing, leading to substantial health-related, social, and economic consequences.[2] It is estimated that the number of people living with Parkinson's disease has doubled over recent decades, with a further rise in disease prevalence expected in the coming years.[2] The disease is associated with a gradual decline in motor function, progression of non-motor symptoms, and loss of functional independence, thereby significantly reducing the quality of life of both patients and their caregivers.[3]

The pathogenesis of Parkinson's disease is complex and multifactorial.[4] A central pathological feature is the progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta, resulting in dopamine deficiency in the striatum and dysfunction of basal ganglia circuits involved in motor control.[5] Other important mechanisms include α-synuclein misfolding and aggregation and Lewy body formation, chronic neuroinflammation, oxidative stress, mitochondrial dysfunction, and impaired neuroplasticity.[4,6] These processes contribute to the development of characteristic motor manifestations, primarily bradykinesia, resting tremor, muscular rigidity, and, in more advanced stages, gait impairment and postural instability. Parkinson's disease is also associated with numerous non-motor manifestations, including cognitive impairment, depression, sleep disturbances, fatigue, pain, and autonomic dysfunction.[3]

Despite substantial advances in pharmacotherapy and the development of surgical treatment methods, including deep brain stimulation, currently available therapeutic strategies are primarily symptomatic and do not reliably halt the underlying neurodegenerative process.[7] Consequently, increasing attention has been directed toward non-pharmacological interventions that may influence both the clinical course of the disease and, potentially, the mechanisms of neurodegeneration.[8] Physical activity has attracted particular interest and, in recent years, has become the subject of intensive research in the context of neuroprotection and supportive treatment in Parkinson's disease.[8–9]

Current experimental and clinical evidence indicates that regular physical activity may exert multidirectional beneficial effects on nervous system function.[9–10] The proposed mechanisms include stimulation of neuroplasticity, increased expression of neurotrophic growth factors, improvement of mitochondrial function, reduction of oxidative stress and neuroinflammation, and modulation of processes related to α-synuclein aggregation.[9,11] However, it should be emphasized that while the clinical benefits of exercise for motor function and quality of life are relatively well supported, direct evidence confirming long-term neuroprotective or disease-modifying effects in humans remains limited.[10] A growing body of evidence also suggests that appropriately designed exercise programmes may improve motor function, balance, gait, physical capacity, and quality of life in patients with Parkinson's disease.[12] In addition, physical activity appears to have a beneficial effect on non-motor symptoms, including cognitive function, mood, and sleep quality, although the strength of evidence varies across outcomes, disease stages, and exercise modalities.[13]

In recent years, particular interest has focused on various exercise modalities, such as aerobic training, resistance training, high-intensity interval training (HIIT), balance training, treadmill-based exercise, and interventions based on dance or Tai Chi.[12,14] However, important limitations remain regarding the precise determination of the optimal type, intensity, frequency, and duration of physical activity in patients with Parkinson's disease, as well as the assessment of its potential long-term effects on disease progression.[10]

The aim of this review is to present current knowledge on the role of physical activity in Parkinson's disease, with particular emphasis on neurobiological mechanisms relevant to neuroprotection and on the clinical implications of different forms of exercise training in patients affected by this disorder.

2. Pathophysiology of Parkinson's Disease

Parkinson's disease is a chronic, progressive neurodegenerative disorder characterized by the gradual loss of dopaminergic neurons in the substantia nigra pars compacta and the presence of pathological α-synuclein aggregates in the form of Lewy bodies.[4–5] Despite many years of research, the exact mechanisms underlying disease onset and progression have not been fully elucidated, and the pathogenesis of Parkinson's disease is considered multifactorial and complex.[6] Both genetic and environmental factors contribute to disease development and interact with mechanisms such as impaired cellular homeostasis, chronic neuroinflammation, mitochondrial dysfunction, oxidative stress, α-synuclein pathology, and progressive neurodegeneration.[6,15]

2.1. Degeneration of Dopaminergic Neurons

A key mechanism underlying the motor manifestations of Parkinson's disease is the progressive loss of dopaminergic neurons within the substantia nigra pars compacta.[5] The consequence of this process is a reduction in dopamine levels in the striatum, leading to impaired function of the basal ganglia, which are responsible for motor control.[5,16] Motor symptoms are traditionally considered to become clinically apparent only after substantial nigrostriatal degeneration, often estimated at approximately 50–60% loss of dopaminergic neurons and 70–80% depletion of striatal dopamine, reflecting the considerable compensatory capacity of the nervous system in early disease stages.[16]

Dopamine deficiency disrupts the balance between the direct and indirect pathways of the basal ganglia, resulting in excessive inhibition of the thalamus and reduced activation of the motor cortex.[5] Clinically, this manifests primarily as bradykinesia, muscular rigidity, and resting tremor, while gait impairment and postural instability usually become more prominent with disease progression.[3]

However, Parkinson's disease pathology is not limited to the nigrostriatal dopaminergic system. Degeneration and dysfunction of non-dopaminergic pathways, including noradrenergic, serotonergic, cholinergic, and autonomic nervous system circuits, also contribute to the heterogeneous clinical presentation of the disease. These alterations are particularly relevant to non-motor symptoms such as cognitive impairment, depression, sleep disturbances, autonomic dysfunction, and pain, which may occur early in the disease course and substantially affect patients' quality of life.[3]

2.2. α-Synuclein Aggregation and Lewy Body Formation

One of the characteristic neuropathological features of Parkinson's disease is the presence of abnormal α-synuclein aggregates forming Lewy bodies and Lewy neurites.[17] α-Synuclein is a presynaptic protein involved, among other functions, in the regulation of synaptic vesicle trafficking and dopaminergic neurotransmission.[17]

Under pathological conditions, α-synuclein undergoes abnormal misfolding, aggregation, and intracellular deposition within neurons.[17–18] The accumulation of pathological α-synuclein may disrupt mitochondrial function, impair autophagy and proteasomal degradation pathways, and activate neuroinflammatory processes.[18] A growing body of evidence suggests that pathological α-synuclein may propagate between neurons through mechanisms resembling prion-like transmission, although the clinical significance and therapeutic implications of this process continue to be investigated.[19]

2.3. Neuroinflammation and Oxidative Stress

Chronic neuroinflammation plays an important role in the development and progression of Parkinson's disease.[20] Activation of microglia and astrocytes is associated with increased production of pro-inflammatory mediators, including interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumour necrosis factor α (TNF-α).[20]

At the same time, increased oxidative stress is observed, resulting from excessive production of reactive oxygen species (ROS) and impaired antioxidant defence mechanisms.[21] Dopaminergic neurons are particularly vulnerable to oxidative damage due to their high energy demands and intensive oxidative metabolism.[15] Excessive oxidative stress may cause damage to membrane lipids, proteins, and genetic material, thereby intensifying mitochondrial dysfunction and progressive neurodegeneration.[21]

2.4. Mitochondrial Dysfunction

Mitochondrial dysfunction is one of the key pathogenetic mechanisms in Parkinson's disease.[15] Numerous studies have demonstrated impaired function of complex I of the mitochondrial respiratory chain, leading to reduced ATP production and increased generation of reactive oxygen species.[15,22]

The importance of mitochondrial dysfunction is further supported by genetic findings related to mutations in genes involved in mitochondrial quality control, such as PINK1, Parkin, and DJ-1.[22] Disturbances in mitophagy lead to the accumulation of damaged mitochondria and further exacerbation of oxidative stress and neurodegeneration.[22]

2.5. Impaired Neuroplasticity

Parkinson's disease is also associated with disturbances in neuroplasticity mechanisms responsible for the adaptation and reorganization of neuronal connections.[23] Dopaminergic dysfunction may impair corticostriatal synaptic plasticity, which is essential for motor learning, adaptive movement control, and the integration of motor and cognitive processes.[23] Reduced or dysregulated activity of neurotrophic pathways, including those involving brain-derived neurotrophic factor (BDNF), may also contribute to impaired neuronal survival, synaptic plasticity, and compensatory adaptation in Parkinson's disease.[23,24] These abnormalities may be relevant not only to the progression of motor symptoms but also to deterioration of cognitive and functional performance.[24,45] At the same time, mechanisms of neuronal plasticity are regarded as one of the most important potential therapeutic targets for non-pharmacological interventions, including physical activity.[9]

3. Neuroprotective Mechanisms of Physical Activity in Parkinson's Disease

In recent years, physical activity has become the subject of intensive research as a potential modulator of biological processes relevant to neuroprotection in Parkinson's disease (PD). A growing body of experimental and clinical evidence indicates that regular exercise may influence multiple mechanisms involved in the neurodegenerative process, including dopaminergic neuronal function, neuroinflammation, oxidative stress, mitochondrial function, and neuroplasticity.[10,25] Owing to these multidirectional effects, physical activity is currently regarded not only as an element of motor rehabilitation but also as a promising intervention that may support adaptive neurobiological processes in Parkinson's disease. However, direct evidence confirming long-term disease-modifying or neuroprotective effects in humans remains limited and should be interpreted with caution.[10,25]

3.1. Exercise-Induced Neuroplasticity

One of the most important neuroprotective mechanisms of physical activity is the stimulation of neuroplasticity, defined as the capacity of the nervous system for structural and functional reorganization.[9,25] Regular exercise may promote processes related to synaptogenesis, neuronal communication, cortical and subcortical adaptation, and motor learning, although the strength of clinical evidence varies depending on the outcome measure, disease stage, and study design.[9,25]

Particular importance is attributed to increased expression of brain-derived neurotrophic factor (BDNF), which is involved in neuronal survival, synaptic plasticity, and learning and memory processes.[11] In the study by Frazzitta et al., an intensive 4-week motor rehabilitation programme in patients with Parkinson's disease resulted in a significant increase in serum BDNF levels, accompanied by improvement in motor function assessed using UPDRS III.[27] Similar findings were reported by Fisher et al., who demonstrated that 8 weeks of high-intensity treadmill training improved motor performance and corticomotor excitability in people with early Parkinson's disease.[28]

In the randomized SPARX trial, 6 months of high-intensity treadmill exercise in patients with de novo Parkinson's disease was associated with favourable effects on motor outcomes compared with usual care, while also demonstrating feasibility and safety of high-intensity aerobic exercise in this population.[29] The authors suggested that the observed changes may be associated with enhanced neuroplasticity and improved exercise-induced neuronal adaptation.

Physical activity also affects the expression of other neurotrophic factors, such as glial cell line-derived neurotrophic factor (GDNF) and insulin-like growth factor 1 (IGF-1), both of which exert protective effects on dopaminergic neurons.[11,25]

3.2. Effects of Physical Activity on Dopaminergic Neurotransmission

Regular physical activity may beneficially influence the function of the dopaminergic system, whose impairment represents a fundamental component of Parkinson's disease pathophysiology.[10,26] Experimental models have shown that exercise may increase dopamine synthesis and release, improve dopaminergic receptor function, and modulate the expression of the dopamine transporter (DAT).[30]

The clinical relevance of these mechanisms is supported indirectly by studies involving human participants. In the SPARX (Study in Parkinson Disease of Exercise) phase 2 trial, 6 months of high-intensity treadmill exercise was associated with stabilization of motor symptoms, assessed using MDS-UPDRS III, whereas worsening was observed in the usual-care group. The between-group difference was statistically significant ($p = 0.03$).[29]

Importantly, this trial was designed primarily to assess safety, feasibility, and nonfutility, and larger phase 3 studies are needed to confirm whether high-intensity exercise can modify the clinical progression of Parkinson's disease.[29]

In a meta-analysis by Cronin et al., exercise therapy, including aerobic training, was associated with improvements in motor function and gait parameters in patients with Parkinson's disease.[12]

3.3. Anti-Inflammatory Effects of Physical Activity

Chronic neuroinflammation plays an important role in the progression of Parkinson's disease; therefore, modulation of the inflammatory response represents one of the potential neuroprotective mechanisms of physical activity.[31] Experimental studies and mechanistic reviews suggest that regular exercise may reduce microglial activation and downregulate the expression of pro-inflammatory cytokines, including TNF-α, IL-1β, and IL-6, although the magnitude of these effects varies depending on the animal model, exercise protocol, and biomarker assessment method.[31–32]

Furthermore, physical activity may increase the release of anti-inflammatory cytokines, including interleukin-10 (IL-10), which may limit the chronic neurodegenerative process.[31]

3.4. Reduction of Oxidative Stress and Improvement of Mitochondrial Function

Oxidative stress and mitochondrial dysfunction are among the most important mechanisms contributing to dopaminergic neuronal damage in Parkinson's disease.[33] Regular physical activity may improve mitochondrial function and increase antioxidant capacity, thereby potentially reducing vulnerability to oxidative damage.[10,33]

In experimental models of Parkinson's disease, exercise has been associated with enhanced antioxidant defence and reduced oxidative damage, including changes in enzymes such as superoxide dismutase, catalase, and glutathione peroxidase, although these effects depend on the model and exercise protocol.[33–34]

Particular importance is attributed to pathways involved in mitochondrial biogenesis and cellular energy metabolism, including PGC-1α-related signalling.[33]

3.5. Physical Activity and α-Synuclein Pathology

A growing number of studies suggest that physical activity may influence processes related to α-synuclein aggregation and clearance.[35] Preclinical studies suggest that regular exercise may reduce pathological α-synuclein accumulation and limit dopaminergic neuronal damage, partly through mechanisms related to autophagy and protein clearance.[35–36] However, evidence for a direct effect of physical activity on α-synuclein pathology in humans remains limited and should be interpreted with caution.

3.6. Exerkines and Emerging Research Directions

In recent years, particular interest has focused on so-called exerkines, biologically active molecules released during physical exercise that may mediate the beneficial effects of exercise on the nervous system.[37]

Irisin, a myokine secreted by skeletal muscles during exercise, may stimulate BDNF expression and contribute to improved mitochondrial function and neuroplasticity.[37–38] Increasing evidence also suggests that lactate produced during physical exercise may act as a signalling molecule influencing neuronal metabolism and adaptive processes in the brain.[38]

Despite promising findings, further clinical studies are still required to determine which forms of physical activity exert the strongest neuroprotective effects and which training parameters are most effective in patients with Parkinson's disease.[25–26]

Taken together, physical activity may influence several interconnected biological pathways relevant to Parkinson's disease, including neurotrophic signalling, dopaminergic neurotransmission, mitochondrial function, oxidative stress, neuroinflammation, protein clearance, and α-synuclein-related pathology. These mechanisms provide a plausible biological rationale for the clinical benefits of exercise observed in patients with Parkinson's disease. Nevertheless, most direct evidence for neuroprotective effects comes from experimental and mechanistic studies, whereas long-term human data based on imaging, fluid biomarkers, or disease-progression outcomes remain limited.

4. Clinical Effects of Physical Activity in Parkinson's Disease

An increasing number of clinical studies indicate that regular physical activity may have a significant impact on clinical manifestations and functional outcomes in Parkinson's disease, affecting both motor and selected non-motor symptoms.[9,20] In recent years, particular attention has been paid to the potential improvement of motor function, physical capacity, quality of life, and cognitive performance through appropriately designed exercise programmes.[9–10] Importantly, some of the observed effects appear to extend beyond conventional rehabilitation, suggesting a potential influence of physical activity on neurodegenerative processes and neuroplasticity.[11]

4.1. Effects of Physical Activity on Motor Symptoms

Motor symptoms remain the main cause of functional decline in patients with Parkinson's disease. Numerous studies have shown that regular physical activity may improve bradykinesia, balance, gait, and postural control.[39]

In the SPARX phase 2 randomized clinical trial by Schenkman et al., 6 months of high-intensity treadmill exercise in patients with de novo Parkinson's disease was feasible and was associated with a smaller worsening of MDS-UPDRS III scores compared with usual care, supporting further evaluation of high-intensity exercise in larger efficacy trials.[29] However, as a phase 2 nonfutility trial, SPARX should be interpreted as supportive but not definitive evidence for clinical efficacy or disease modification.[29]

Similar findings were reported in a meta-analysis by Cronin et al., which included more than 1100 patients with Parkinson's disease.[12] The authors demonstrated that aerobic training interventions resulted in a mean improvement in UPDRS III scores of approximately 3.5–5.7 points compared with control groups. In addition, improvements were observed in gait speed by approximately 0.08–0.12 m/s and in Timed Up and Go (TUG) performance by a mean of 1.5–2.3 seconds.[12]

Studies on treadmill training have demonstrated improvements in stride length, gait rhythm, and reduction in freezing of gait episodes.[40] In a small open-label pilot study by Herman et al., 6 weeks of intensive treadmill training improved gait speed, gait rhythmicity, functional performance, UPDRS scores, and quality of life in patients with Parkinson's disease.

However, due to the small sample size and uncontrolled design, these findings should be interpreted as preliminary.[40]

Significant benefits have also been observed with resistance training. In a 2-year randomized study by Corcos et al., progressive resistance training improved UPDRS III scores by a mean of 7.3 points compared with the control group ($p < 0.001$).[41] In addition, muscle strength and functional performance improved in the patients undergoing training.

4.2. Effects of Physical Activity on Non-Motor Symptoms

Non-motor symptoms, such as depression, chronic fatigue, sleep disturbances, and autonomic dysfunction, have a substantial impact on the quality of life of patients with Parkinson's disease.[26] A growing body of research suggests that regular physical activity may also have beneficial effects on this group of symptoms.[42]

The most frequently reported benefits concern depressive symptoms, fatigue, sleep quality, and perceived well-being, although the magnitude of these effects varies between studies and depends on the type of intervention, duration of follow-up, disease severity, and baseline symptom burden.[13,42,44]

Physical activity may also have beneficial effects on sleep disturbances and fatigue in selected patients with Parkinson's disease.[44] Randomized studies have associated exercise interventions with improvements in subjective sleep quality and selected objective sleep parameters; however, the results remain heterogeneous, and the optimal type, intensity, timing, and duration of exercise for sleep-related outcomes have not been clearly established.[44]

4.3. Cognitive and Neuropsychological Functions

Cognitive impairment represents an important clinical problem in Parkinson's disease and may affect attention, executive functions, working memory, processing speed, and visuospatial abilities, particularly in the later stages of the disorder.[45] Increasing evidence indicates that physical activity may support cognitive performance by stimulating neuroplasticity, improving cerebral perfusion, modulating neuroinflammatory pathways, and enhancing the function of cortico-subcortical networks.[30]

In a systematic review by da Silva et al., exercise programmes were shown to improve cognitive function and reduce the severity of depressive symptoms.[13] In some of the analysed studies, Montreal Cognitive Assessment (MoCA) scores improved by an average of 1.5–2 points following interventions lasting from 12 to 24 weeks.[13]

In a meta-analysis by Levin et al., regular aerobic and multimodal training were shown to significantly improve executive functions, attention, and information processing speed.[46] The greatest effects were observed in programmes lasting at least 12 weeks and including moderate- to high-intensity exercise.

In the study by Cruise et al., an 8-week aerobic training programme resulted in significant improvement in executive functions and working memory in patients with PD ($p < 0.05$).[43] The authors suggested that these effects may be related to increased BDNF activity and improved cerebral blood flow.

In a randomized study by David et al., patients participating in a cognitive-motor training programme achieved better results in tests assessing executive functions and working memory compared with the control group ($p < 0.05$).[47]

Regular physical activity may support cognitive function through effects on neuroplasticity, cerebral perfusion, and neuroinflammatory pathways; however, evidence that exercise reduces the long-term risk of Parkinson's disease dementia remains insufficient.[11]

4.4. Quality of Life and Functional Performance

The quality of life of patients with Parkinson's disease is significantly limited by both motor and non-motor symptoms.[26] Numerous studies indicate that physical activity may improve daily functioning and increase patients' independence.[9]

In a meta-analysis by Goodwin et al., regular physical exercise led to significant improvement in quality of life assessed using the Parkinson's Disease Questionnaire-39 (PDQ-39).[48] The mean improvement was approximately 6–8 points compared with control groups, which was considered a clinically meaningful therapeutic effect. These improvements are clinically relevant because quality of life in Parkinson's disease is influenced not only by motor disability, but also by fatigue, mood disturbances, sleep problems, cognitive impairment, and reduced participation in daily and social activities.[26,48]

Improvements were also observed in parameters related to activities of daily living (ADL), exercise capacity, and functional independence.[48] Longitudinal and observational data suggest that patients who maintain regular physical activity may experience a slower decline in functional performance than those with a sedentary lifestyle; however, these associations may be influenced by baseline disease severity, general health status, motivation, and access to supervised exercise programmes.[49]

4.5. Reduction of Fall Risk and Improvement of Balance

Balance disturbances and falls are among the most serious complications of Parkinson's disease, especially in patients with more advanced stages of the disorder.[50] Regular physical activity may reduce the risk of falls by improving postural control, muscle strength, and motor coordination.[51] However, fall-prevention strategies should be individualized, particularly in patients with advanced disease, cognitive impairment, freezing of gait, orthostatic hypotension, or a history of recurrent falls, as these factors may limit training safety and effectiveness.

Particularly beneficial effects have been observed with balance-oriented training, Tai Chi, and multimodal exercise programmes.[52] In a randomized study by Li et al., patients participating in a Tai Chi programme demonstrated significant improvement in postural stability and a reduction in the number of falls compared with resistance training and stretching groups ($p < 0.001$).[52]

Meta-analytic data suggest that exercise programmes targeting balance, lower-limb strength, and gait may reduce fall risk in patients with Parkinson's disease, particularly in those with milder disease and preserved ability to participate safely in training.[51] Improvements have also been observed in balance and mobility measures, including the Berg Balance Scale and Timed Up and Go test, although effects are less consistent in more advanced disease.[51]

Overall, current evidence supports physical activity as an important adjunctive intervention in the comprehensive management of Parkinson's disease. Appropriately designed exercise programmes may improve motor symptoms, gait, balance, functional independence, selected non-motor symptoms, cognitive performance, and quality of life. However, the magnitude of benefit depends on disease stage, baseline functional status, exercise modality, intensity, supervision, and adherence. Although exercise-induced neuroplastic adaptations provide a plausible biological explanation for some clinical effects, definitive evidence for long-term disease-modifying or neuroprotective effects in humans remains insufficient.[9,11]

5. Types of Physical Activity and Their Therapeutic Potential in Parkinson's Disease

The choice of physical activity in patients with Parkinson's disease should be guided by the predominant clinical symptoms, disease stage, level of physical function, fall risk, and therapeutic goals. Current evidence indicates that regular, structured exercise may improve motor symptoms, balance, gait, cardiorespiratory fitness, functional performance, and quality of life in patients with Parkinson's disease.[53–54] At the same time, recent meta-analyses suggest that clinical benefits may be achieved through various forms of exercise, and the superiority of one modality over another is not always clearly established.[53] For this reason, increasing emphasis is placed on individualizing exercise programmes and combining several types of physical activity within multimodal therapy.[54–55] It should be noted that most clinical trials have included patients with mild to moderate Parkinson's disease and without major cognitive impairment. Therefore, recommendations derived from these studies should be applied more cautiously in patients with advanced disease, severe postural instability, frequent falls, significant dyskinesia, or clinically relevant cognitive impairment.[53,54] In clinical practice, the selection of exercise modality should be based not only on evidence of efficacy but also on the patient's dominant symptoms, safety profile, personal preferences, access to supervision, and likelihood of long-term adherence.

5.1. Aerobic Training

Aerobic training is one of the best-documented forms of physical activity in Parkinson's disease. It includes walking, brisk walking, Nordic walking, stationary cycling, treadmill training, and other endurance exercises involving large muscle groups.[29,54] Its main aim is to improve cardiorespiratory fitness, exercise tolerance, gait parameters, and overall functional capacity.

In the SPARX trial, Schenkman et al. evaluated the effects of treadmill training in patients with de novo Parkinson's disease who had not yet started dopaminergic treatment. After 6 months of intervention, the group performing high-intensity training, corresponding to 80–85% of maximum heart rate, showed less deterioration in MDS-UPDRS III scores than the control group. The between-group difference was statistically significant ($p = 0.03$), suggesting the potential importance of intensive aerobic training in the early stage of the disease.[29] The same study also demonstrated improvement in physical fitness, supporting the careful implementation and further evaluation of structured endurance exercise in selected patients with early-stage Parkinson's disease.[29]

The importance of aerobic training is also supported by clinical guidelines, in which moderate- to high-intensity exercise is recommended to improve oxygen consumption, reduce the severity of motor symptoms, and enhance functional performance.[54] From a clinical perspective, aerobic training may be particularly useful in patients with early- to moderate-stage disease who are able to safely achieve higher exercise intensity.

5.2. Resistance Training

Resistance training is an important component of therapeutic management because reduced muscle strength, decreased muscle power, and impaired postural control may exacerbate functional limitations in patients with Parkinson's disease.[56] Strength exercises may include body-weight training, resistance bands, free weights, weight machines, or functional exercises such as rising from a chair, squats, or step-ups.

In a 2-year randomized study by Corcos et al., progressive resistance training led to significant improvement in motor symptoms measured using the UPDRS III scale. After 24 months, the difference between the resistance training group and the control group was 7.3 points in favour of resistance training ($p < 0.001$).[41] This is particularly important because the study had a long follow-up period, and the observed effect exceeded the typical short-term improvement seen in many rehabilitation programmes.

Resistance training may be especially valuable in patients with lower-limb muscle weakness, impaired transfers, difficulty rising from a seated position, and increased fall risk.[58] In clinical practice, however, it should be adapted to the patient's functional status, the presence of dyskinesia, orthostatic hypotension, pain, and coexisting cardiovascular or musculoskeletal disorders.

5.3. High-Intensity Interval Training

High-intensity interval training (HIIT) is increasingly being investigated as an intervention that may stimulate cardiorespiratory, metabolic, and potentially neuroplastic adaptations.[57] It consists of alternating short periods of intense exercise with rest phases or low-intensity activity.

A potential advantage of HIIT is the ability to achieve a substantial physiological stimulus in a shorter time compared with continuous moderate-intensity training. Pilot studies suggest that HIIT in selected patients with mild to moderate Parkinson's disease may improve aerobic capacity, gait parameters, and MDS-UPDRS III scores.[57] At the same time, data on this form of training remain less extensive than for conventional aerobic training, and the safety of HIIT in patients with more advanced disease, postural instability, or significant comorbidities requires further investigation.[57]

Therefore, HIIT should be regarded as a promising but selective form of exercise therapy. It is best suited for physically capable, motivated patients with low to moderate fall risk, provided that safety is assessed beforehand and exercise intensity is appropriately monitored.

5.4. Treadmill Training and Gait Training

Gait disturbances are among the most disabling symptoms of Parkinson's disease and include reduced stride length, decreased gait speed, rhythm disturbances, difficulty initiating movement, and freezing of gait.[50] Treadmill training and targeted gait training aim to improve gait automaticity, rhythmicity, stride length, and exercise tolerance.

A Cochrane review on treadmill training showed that this intervention may improve selected gait parameters, particularly gait speed and stride length, whereas effects on walking distance and cadence appear less consistent.[59] Clinically, treadmill training is important because it allows repeated and controlled exposure to the walking task and, when necessary, may be combined with body-weight support, safety harnesses, or rhythmic cues.

Strategies based on external cueing, such as rhythmic auditory stimuli, visual lines on the floor, or metronome cues, are also becoming increasingly important. In patients with freezing of gait, these strategies may improve movement initiation and reduce gait irregularity.[50] In clinical practice, gait training should include not only straight-line walking but also turning, speed changes, obstacle negotiation, stopping, and starting, as these are the situations that most commonly provoke gait disturbances in daily functioning.

5.5. Balance Training and Task-Specific Exercises

Balance disturbances and postural instability are among the most important risk factors for falls in patients with Parkinson's disease. Unlike some motor symptoms, such as rigidity or bradykinesia, postural disturbances often respond less effectively to dopaminergic treatment and therefore require targeted rehabilitation.[50]

Balance training includes exercises focused on trunk stabilization, control of the centre of gravity, balance reactions, proprioception, changes in body position, and dual-task conditions. In a randomized study by Canning et al., an exercise programme targeting fall risk factors showed varying effects depending on disease severity: patients with milder disease experienced greater benefits in fall reduction than those with more advanced disease.[60] This finding highlights the importance of early implementation of balance training before severe postural disturbances become established.

The APTA guidelines indicate that physiotherapists should use balance interventions, task-specific training, and gait training to improve stability, mobility, and functioning in patients with Parkinson's disease.[54] Exercises that reproduce real-life daily situations are particularly useful, such as standing up, turning, reaching for objects, walking on uneven surfaces, or performing two tasks simultaneously.

5.6. Tai Chi, Yoga, and Dance-Based Interventions

Forms of activity that combine movement, rhythm, balance, postural control, and cognitive engagement are particularly interesting in Parkinson's disease because they simultaneously involve the motor, sensory, and executive systems.[61–63] This group includes Tai Chi, yoga, and dance-based interventions.

Tai Chi may improve postural stability, control of the centre of gravity, and gait confidence. Clinical studies have demonstrated beneficial effects of Tai Chi on balance, postural stability, and fall-related outcomes, particularly in patients with mild to moderate Parkinson's disease who are able to safely participate in standing balance exercises.[61] Yoga, in turn, may additionally improve flexibility, muscle tone, anxiety symptoms, sleep quality, and subjective well-being.[62]

Dance-based interventions, especially tango, rhythmic dance, and music-based programmes, may improve gait rhythmicity, turning, coordination, and balance. An important value of dance therapy is also its social and emotional dimension, which may increase adherence to long-term physical activity.[63] Compared with conventional rehabilitation exercises, dance may be more attractive for some patients, which is of practical importance because the effectiveness of exercise depends largely on regular participation.

5.7. Training Using Virtual Reality and Technology

Modern technologies, including virtual reality, exergames, balance platforms, and biofeedback systems, are increasingly used in the rehabilitation of Parkinson's disease. Their potential advantage lies in the ability to combine motor training with visual stimuli, cognitive tasks, and real-time feedback.[64]

Systematic reviews indicate that virtual reality-based training may improve balance, mobility, and gait parameters, although the quality of evidence and heterogeneity of protocols remain important limitations.[64] These interventions may be particularly useful in patients with gait disturbances and difficulties adapting movement to a changing environment. In clinical practice, however, they should complement rather than replace conventional functional training.

5.8. Multimodal Training

For many patients with Parkinson's disease, the most practical model of management is multimodal training, combining elements of aerobic, resistance, balance, stretching, coordination, and task-specific exercises.[53–54] This approach corresponds to the complex clinical presentation of the disease, in which motor and non-motor symptoms, autonomic disturbances, low mood, and reduced functional performance mutually reinforce one another.

Current practical recommendations emphasize that individuals with Parkinson's disease should aim for regular physical activity covering different domains of fitness: endurance, strength, balance, mobility, and stretching.[54,65] Approximately 150 minutes of moderate- to high-intensity activity per week is recommended, provided that the patient's clinical condition allows it.[65] Exercise should be introduced as early as possible after diagnosis, preferably during periods of optimal dopaminergic medication effect, and, in patients at risk of falls, under the supervision of a physiotherapist.[54,65]

In light of the available evidence, the most important clinical factors are not only the choice of a specific type of exercise but also regularity, appropriate intensity, safety, attractiveness of the programme, and long-term adherence.[53–54] Therefore, physical activity should be regarded as a permanent component of comprehensive Parkinson's disease treatment, alongside pharmacotherapy, patient education, physiotherapy, and lifestyle modification. Rather than selecting a single universally superior modality, exercise prescription in Parkinson's disease should be individualized and periodically modified according to disease progression, treatment response, fall risk, cognitive status, comorbidities, and patient preferences.

6. Recommendations for Physical Activity and Practical Clinical Considerations

Physical activity should be regarded as an important component of comprehensive treatment for Parkinson's disease, implemented as early as possible after diagnosis and integrated with pharmacotherapy, patient education, and physiotherapeutic management.[53–55,66] The contemporary approach has moved away from viewing exercise solely as rehabilitation introduced only after significant disability has developed. Increasingly, it is emphasized that physical activity should be "prescribed" in a manner similar to pharmacological treatment — with specification of the type, dose, intensity, frequency, duration, monitoring strategy, safety precautions, and criteria for programme modification.[55,66]

Available evidence indicates that structured exercise programmes may improve motor symptoms, cardiorespiratory fitness, muscle strength, balance, gait, cognitive function, quality of life, and some non-motor symptoms.[53–54,67–68] At the same time, the effectiveness of physical activity depends not only on the selected form of training, but primarily on regularity, appropriate intensity, safety, and the possibility of maintaining the programme in the long term.[53–54,65]

6.1. The FITT Principle in Planning Physical Activity

Practical planning of physical activity in patients with Parkinson's disease should be based on the FITT principle, which includes frequency, intensity, time, and type of exercise.[66–67] This model allows general recommendations for physical activity to be translated into a specific and feasible therapeutic programme.

According to current recommendations, patients with Parkinson's disease should aim to perform moderate- to vigorous-intensity aerobic activity for approximately 150 minutes per week, provided that their clinical condition allows it.[65,67] Aerobic exercise may include walking, brisk walking, stationary cycling, treadmill training, Nordic walking, or other activities involving large muscle groups.[54,65,67]

The programme should also include resistance training at least 2–3 times per week, particularly targeting the muscles of the lower limbs, trunk muscles, and muscle groups responsible for transfers, rising from a chair, and postural stabilization.[54,67] Balance, gait, and task-specific exercises should be performed regularly, especially in patients with postural instability, freezing of gait, shortened stride length, or increased fall risk.[50,54,60] The programme should be complemented by stretching and mobility exercises, which help reduce rigidity, contractures, and secondary pain complaints.[54,65]

In clinical practice, it is particularly important that recommendations are specific. Instead of a general message such as "please move more," the patient should receive a clear and individualized plan: how many times per week to exercise, for how long, at what intensity, which forms of activity to choose, how to monitor tolerance, and in which situations exercise should be stopped or modified.

6.2. Exercise Intensity and Effort Monitoring

Exercise intensity is one of the key factors determining the effectiveness of the intervention. Studies indicate that moderate- and high-intensity training may lead to more pronounced improvements in physical capacity and motor symptoms than very low-intensity activity.[29,66] In the SPARX trial, treadmill training performed for 6 months at 80–85% of maximum heart rate was associated with less deterioration in MDS-UPDRS III scores compared with the control group ($p = 0.03$).[29]

In practice, exercise intensity may be monitored using heart rate, the Borg Rating of Perceived Exertion scale, or a simple "talk test".[67] However, in many patients with Parkinson's disease, heart rate alone may not be sufficiently reliable because of autonomic dysfunction, orthostatic hypotension, chronotropic impairment, medications, or comorbidities. Therefore, the safest approach is to combine several monitoring methods, including observation of clinical symptoms, exercise tolerance, fatigue, dizziness, dyspnoea, and postural stability.[66–67]

Intensity should be increased gradually. In previously inactive or older patients, those with multimorbidity, or those at greater risk of falls, the initial goal may simply be to increase movement regularity and exercise tolerance. Only later should the duration, intensity, or complexity of exercise be progressively increased.[54,66]

6.3. Selection of Activity According to Disease Stage

The physical activity programme should be adapted to the disease stage, predominant symptoms, and degree of patient independence. In patients at an early stage who maintain good functional capacity, the key objectives are to maintain a high level of activity and improve aerobic fitness, muscle strength, coordination, and flexibility.[54–55] In this group, moderate- or high-intensity aerobic training, resistance training, Nordic walking, dance, Tai Chi, stationary cycling, or other activities tailored to the patient's preferences may be safely considered.[54,65]

In the moderate stage, balance exercises, gait training, turning practice, changes of direction, movement initiation, and task-specific exercises become increasingly important.[50,54] The programme should address situations that most commonly cause difficulties in daily life: rising from a chair, initiating gait, walking through doorways, turning, overcoming obstacles, walking on uneven surfaces, and performing two tasks simultaneously.

In patients with advanced disease, the main goals of physical activity are usually to maintain the highest possible level of independence, prevent contractures, reduce complications of immobility, improve transfers, support safe mobility, and decrease fall risk.[54,68] In this group, exercises often need to be performed under the supervision of a physiotherapist or caregiver, with safety support, the use of assistive devices, exercises in seated or lying positions, and adaptation of activity to periods of best motor performance.

6.4. Safety and Contraindications

Physical activity is generally safe for most patients with Parkinson's disease; however, individual risk should be assessed before implementing an exercise programme. This assessment is particularly important before introducing moderate- to high-intensity training, HIIT, treadmill exercise, or unsupervised home-based programmes. Particular attention is required in patients with frequent falls, freezing of gait, postural instability, severe dyskinesia, motor fluctuations, orthostatic hypotension, cognitive impairment, osteoporosis, musculoskeletal pain, or cardiovascular disease.[50,54,66]

In patients at high risk of falls, preferred forms of activity include those that allow safety control, such as stationary cycling, exercises performed near handrails, supervised training, seated exercises, supervised classes, or aquatic exercise.[54,68] In individuals with cognitive impairment, the programme should be simple, repetitive, well structured, and preferably supported by a caregiver.

It is also important to adjust the timing of exercise to the effects of dopaminergic medication. In many patients, exercise is most beneficial during the "on" phase, when motor performance, coordination, and safety are optimal.[65,67] In individuals with severe dyskinesia or motor fluctuations, the optimal time of training during the day may need to be determined individually.

Contraindications to exercise should be assessed similarly to other clinical populations and include, among others, unstable cardiovascular disease, acute infections, recent injuries, uncontrolled metabolic disorders, severe dizziness, or symptomatic orthostatic hypotension. In patients with significant cardiovascular disease, unexplained dyspnoea, chest pain, syncope, or markedly reduced exercise tolerance, medical evaluation should precede the introduction of moderate- or high-intensity exercise. Many of these contraindications are relative or temporary; therefore, physical activity should usually be modified, postponed, reduced in intensity, or undertaken after medical consultation rather than permanently discontinued.[66,67]

6.5. Strategies to Improve Adherence

One of the greatest clinical challenges is not merely recommending physical activity, but maintaining it over the long term. Patients with Parkinson's disease often face numerous barriers, such as fatigue, apathy, depression, fear of falling, pain, bradykinesia, transport limitations, lack of access to physiotherapy, low motivation, or insufficient support from their environment.[69–71]

Adherence may be improved by individualizing the programme, choosing activities consistent with the patient's preferences, setting realistic goals, monitoring progress, providing education about the benefits of exercise, and involving family members or caregivers.[69–70] For some patients, group classes, exercising with a partner, dance, Nordic walking, home-based programmes with telemonitoring, or wearable devices monitoring step count, activity duration, and heart rate may be particularly effective.[70–71]

It is also important to frame the purpose of exercise therapy appropriately. Exercise should not be presented to the patient as short-term rehabilitation, but rather as a long-term strategy supporting physical function, independence, quality of life, and potentially beneficial neuroplastic adaptations, although definitive disease-modifying effects in humans remain unconfirmed.[55,66]

6.6. The Role of the Therapeutic Team

Optimal implementation of physical activity requires interdisciplinary collaboration. The neurologist is responsible for assessing disease stage, optimizing pharmacotherapy, analysing motor fluctuations, dyskinesia, and non-motor symptoms, as well as evaluating the overall safety of exercise. The physiotherapist selects the detailed exercise programme and assesses gait, balance, fall risk, transfers, muscle strength, and functional capacity.[54,68]

In selected cases, an occupational therapist, psychologist, dietitian, speech and language therapist, neurological nurse, and caregiver also play important roles. The occupational therapist may support adaptation of daily activities and the home environment; the psychologist may assist in cases of depression, anxiety, or apathy; and the dietitian may support the maintenance of muscle mass, adequate protein intake, and overall physical capacity.

The APTA guidelines emphasize that physiotherapy should be implemented already in the early stage of Parkinson's disease, rather than only after severe gait or balance disturbances have developed.[54] Early intervention allows for education, fall prevention, maintenance of functional capacity, development of compensatory strategies, and establishment of a long-term habit of physical activity.

6.7. A Practical Model of Recommendations for Patients

In everyday clinical practice, simple, specific, and measurable recommendations are the most useful. An example programme for a patient with early- or moderate-stage Parkinson's disease may include aerobic training 3–5 times per week for 20–40 minutes, resistance training 2–3 times per week, balance and gait exercises 2–3 times per week, and daily stretching and mobility exercises.[54,65,67]

In patients with predominant gait disturbances, particular attention should be paid to training stride length, gait rhythm, movement initiation, turning, and strategies for managing freezing of gait.[50] In patients with predominant muscle weakness and difficulty with transfers, greater emphasis should be placed on resistance training and functional exercises. In patients with fear of falling or postural instability, balance exercises performed under safe conditions should be the priority.[50,54,60]

The effects of exercise should be monitored periodically. Exercise prescriptions should also be periodically reassessed and modified according to disease progression, motor fluctuations, fall history, cognitive status, comorbidities, medication response, patient preferences, and adherence. In practice, simple tools may be used, such as the Timed Up and Go test, the 6-minute walk test, assessment of the number of falls, gait speed, lower-limb strength, fatigue scales, quality of life, and subjective exercise tolerance. This allows the programme to be modified according to disease progression, treatment effectiveness, and the patient's capabilities.[54,68]

However, continuity of activity is of greatest importance. Clinical benefits may diminish after regular exercise is discontinued; therefore, physical activity should be planned as a permanent and adaptable component of the lifestyle of a patient with Parkinson's disease, rather than as a short-term rehabilitation intervention.[53–55,66]

In summary, physical activity in Parkinson's disease should be prescribed early, individualized, monitored, and adapted over time. The most clinically useful programmes combine aerobic, resistance, balance, gait, mobility, and task-specific exercises, while taking into account disease stage, safety, adherence, and patient preferences. Such an approach supports functional independence and quality of life and may enhance neuroplastic adaptations, although its long-term disease-modifying potential remains to be confirmed.

7. Discussion

The available evidence indicates that physical activity is an important component of comprehensive management in patients with Parkinson's disease. Clinical trials, meta-analyses, and guidelines consistently support the beneficial effects of structured exercise on motor symptoms, functional performance, balance, gait, physical capacity, and quality of life.[53–55,68] At the same time, experimental and translational studies suggest that exercise may influence biological processes relevant to Parkinson's disease pathophysiology, including neuroplasticity, mitochondrial function, neuroinflammation, oxidative stress, dopaminergic neurotransmission, and α-synuclein-related mechanisms.[9,55,72–73] However, these two areas of evidence should be clearly distinguished. While the symptomatic and functional benefits of physical activity are relatively well documented, direct confirmation of long-term disease-modifying or neuroprotective effects in humans remains limited.

The strongest clinical evidence concerns improvement in motor symptoms and functional outcomes. Randomized trials and meta-analyses have shown that aerobic training, resistance training, balance training, gait training, Tai Chi, dance, and multimodal programmes may improve MDS-UPDRS III scores, gait speed, Timed Up and Go performance, balance, and quality of life.[29,41,53–54,60] In the SPARX trial, 6 months of high-intensity aerobic training was associated with less deterioration in MDS-UPDRS III scores compared with usual care, suggesting that exercise intensity may be particularly relevant in early-stage Parkinson's disease.[29] Similarly, the 2-year study by Corcos et al. demonstrated the superiority of progressive resistance training over a less intensive control intervention in improving UPDRS III scores, with a between-group difference of 7.3 points after 24 months.[41] These findings support the view that exercise may produce clinically meaningful benefits, especially when it is regular, progressive, appropriately dosed, and adapted to the patient's functional capacity.

An important conclusion emerging from current research is that no single form of physical activity can be considered universally optimal for all patients with Parkinson's disease. Recent network meta-analyses suggest that several exercise modalities may provide benefits in terms of motor symptoms and quality of life, whereas differences between individual interventions are often uncertain because of methodological heterogeneity.[53] From a clinical perspective, this supports an individualized approach. Gait training and cueing strategies may be particularly useful in patients with freezing of gait or impaired movement initiation; balance training and Tai Chi may be preferred in patients with postural instability; resistance training may be especially relevant in individuals with muscle weakness or impaired transfers; and moderate- to high-intensity aerobic training may be appropriate for patients at an early stage of the disease with preserved exercise tolerance.[50,54,60]

The potential neuroprotective role of physical activity remains one of the most promising but also most uncertain areas of research. Preclinical studies indicate that exercise may increase the expression of neurotrophic factors, improve mitochondrial function, reduce oxidative stress, limit microglial activation, and support autophagy and pathological protein clearance.[72–73] Clinical studies have also reported changes in biomarkers such as BDNF after exercise interventions, often accompanied by improvements in motor or functional parameters.[74] Nevertheless, these biomarkers remain difficult to interpret. Their concentrations may be affected by age, sex, medication use, comorbidities, timing of blood sampling, laboratory methodology, and the type and intensity of exercise. Therefore, at present, exercise-related biomarkers should be regarded primarily as research tools rather than as indicators with established routine clinical utility.[74–75]

The concept of "exercise as medicine" is clinically attractive because physical activity has multidimensional effects that extend beyond motor rehabilitation. Unlike pharmacological treatment, which primarily aims to compensate for dopaminergic deficiency, exercise may simultaneously support physical capacity, muscle strength, balance, neuroplasticity, mood, sleep, cognitive function, and independence.[55,68] However, physical activity should not be presented as an alternative to pharmacotherapy or neurological care. Levodopa and other established treatments remain the cornerstone of symptomatic management, whereas exercise should be regarded as a complementary intervention that may increase functional reserve, reduce secondary disability, and support long-term independence.

The clinical relevance of physical activity also extends to non-motor symptoms, which substantially affect quality of life in Parkinson's disease. Exercise programmes may improve executive function, mood, sleep quality, fatigue, and subjective well-being, although these effects are generally more heterogeneous than improvements observed in motor outcomes.[44,46,62] This variability probably reflects differences in exercise modality, intervention duration, disease stage, baseline cognitive or affective status, and the tools used to assess non-motor symptoms. Future studies should therefore include non-motor outcomes more systematically and treat them as clinically important endpoints rather than only secondary observations.

Several limitations of the available literature should be emphasized. Exercise studies in Parkinson's disease differ considerably in terms of intervention type, intensity, frequency, duration, supervision, adherence monitoring, disease stage, medication status, and outcome measures.[53–54] Many trials include relatively small groups, short intervention periods, and limited follow-up after programme completion. These factors make it difficult to determine the optimal "dose" of exercise and the durability of observed benefits. In addition, some studies lack blinded outcome assessment, standardized evaluation in relation to the dopaminergic "on/off" state, and precise monitoring of actual adherence to the exercise protocol.

Patient selection is another important issue. Most clinical trials include individuals with mild to moderate Parkinson's disease, relatively preserved functional capacity, and the ability to participate safely in structured exercise programmes.[54] Less evidence is available for patients with advanced disease, severe freezing of gait, frequent falls, dementia, significant dysautonomia, or multimorbidity. This is clinically important because these patients often have the greatest rehabilitation needs and the highest risk of loss of independence. Further studies should therefore evaluate adapted and supervised exercise programmes in more clinically complex populations.

Long-term adherence remains one of the key determinants of the effectiveness of physical activity. In real-world practice, patients may discontinue exercise because of fatigue, apathy, depression, fear of falling, pain, transport difficulties, limited access to physiotherapy, or insufficient social support.[69–71] For this reason, exercise prescription should not be limited to recommending a specific training modality. It should also include strategies to maintain motivation, adjust the programme to patient preferences, monitor progress, involve caregivers when appropriate, and reduce practical barriers to participation. In many patients, the most effective intervention may be the one that is not only evidence-based but also feasible, safe, enjoyable, and sustainable.

Overall, the current evidence supports the early, individualized, and long-term implementation of physical activity as part of comprehensive Parkinson's disease care. The best documented effects concern motor symptoms, functional performance, balance, gait, physical capacity, and quality of life. The potential influence of exercise on neuroplasticity and neuroprotective mechanisms is biologically plausible and supported by preclinical and translational findings, but it still requires confirmation in long-term randomized trials using reliable biomarkers of disease progression. Future research should focus on defining optimal exercise dose, identifying predictors of response, improving adherence, and developing personalized programmes tailored to disease stage, clinical phenotype, safety profile, and patient preferences.

8. Conclusions

Physical activity is an important component of comprehensive therapeutic management in patients with Parkinson's disease. The available evidence indicates that regular, structured exercise may improve motor symptoms, gait, balance, muscle strength, cardiorespiratory fitness, functional performance, and quality of life.[53–55,68] Current data suggest that benefits may be achieved through several forms of exercise, including aerobic, resistance, balance, gait-oriented, dance-based, Tai Chi, virtual reality-based, and multimodal programmes, although the superiority of one modality over others has not been clearly established.[53]

The role of physical activity in Parkinson's disease extends beyond conventional rehabilitation. Exercise may influence mechanisms relevant to neuroplasticity, mitochondrial function, oxidative stress, neuroinflammation, dopaminergic neurotransmission, and α-synuclein-related pathology.[9,55,72–74] However, the currently available evidence for a direct disease-modifying effect in humans remains indirect. Therefore, physical activity should be recommended primarily because of its proven symptomatic, functional, and quality-of-life benefits, while its potential neuroprotective effects should be regarded as a promising area for further research.[53,75,77]

From a clinical perspective, exercise should be introduced as early as possible after diagnosis and continued as a long-term element of care alongside pharmacotherapy, physiotherapy, patient education, and lifestyle modification.[54–55,66] The programme should be individualized according to disease stage, predominant symptoms, functional capacity, fall risk, comorbidities, patient preferences, and treatment goals. Regularity, safety, appropriate intensity, supervision when needed, and long-term adherence are essential for achieving and maintaining clinical benefits.

In conclusion, physical activity is a safe, accessible, and multidirectionally beneficial intervention that should be considered an integral part of Parkinson's disease management. Future studies should clarify the optimal exercise dose, identify biomarkers and predictors of response, assess long-term effects on disease progression, and develop personalized exercise strategies for patients with different clinical phenotypes and stages of Parkinson's disease.

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References

1. Parkinson's disease Collaborators GBDPsD. Global, regional, and national burden of Parkinson’s disease, 1990-2016: a systematic analysis. Lancet Neurol. 2018;17(11):939-953. doi:https://doi.org/10.1016/S1474-4422(18)30295-3

2. Dorsey ER, Bloem BR. The Parkinson pandemic—a call to action. JAMA Neurol. 2018;75(1):9-10. doi:https://doi.org/10.1001/jamaneurol.2017.3299
3. Poewe W, Seppi K, Tanner CM, et al. Parkinson disease. Nat Rev Dis Primers. 2017;3:17013. doi:https://doi.org/10.1038/nrdp.2017.13
4. Kalia LV, Lang AE. Parkinson's disease. Lancet. 2015;386(9996):896-912. doi:https://doi.org/10.1016/S0140-6736(14)61393-3
5. Obeso JA, Stamelou M, Goetz CG, et al. Past, present, and future of Parkinson's disease: a special essay on the 200th anniversary of the shaking palsy. Mov Disord. 2017;32(9):1264-1310. doi:https://doi.org/10.1002/mds.27115
6. Bloem BR, Okun MS, Klein C. Parkinson's disease. Lancet. 2021;397(10291):2284-2303. doi:https://doi.org/10.1016/S0140-6736(21)00218-X
7. Armstrong MJ, Okun MS. Diagnosis and treatment of Parkinson disease: a review. JAMA. 2020;323(6):548-560. doi:https://doi.org/10.1001/jama.2019.22360
8. Petzinger GM, Holschneider DP, Fisher BE, et al. The effects of exercise on dopamine neurotransmission in Parkinson's disease: targeting neuroplasticity to modulate basal ganglia circuitry. Brain Plast. 2015;1(1):29-39. doi:https://doi.org/10.3233/BPL-150021
9. Petzinger GM, Fisher BE, McEwen S, et al. Exercise-enhanced neuroplasticity targeting motor and cognitive circuitry in Parkinson's disease. Lancet Neurol. 2015;14(7):716-726. doi:https://doi.org/10.1016/S1474-4422(15)00042-8
10. Ahlskog JE. Aerobic exercise: evidence for a direct brain effect to slow Parkinson disease progression. Mayo Clin Proc. 2018;93(3):360-372. doi:https://doi.org/10.1016/j.mayocp.2017.12.015
11. Hirsch MA, Iyer SS, Sanjak M. Exercise-induced neuroplasticity in human Parkinson's disease: what is the evidence telling us? Parkinsonism Relat Disord. 2016;22:S78-S81. doi:https://doi.org/10.1016/j.parkreldis.2015.09.030
12. Cronin NJ, Keogh JWL, Delahunt E, et al. Exercise therapy for motor dysfunction in Parkinson disease: a systematic review and meta-analysis. Neurorehabil Neural Repair. 2021;35(9):775-789. doi:https://doi.org/10.1177/15459683211030979
13. da Silva FC, Iop RDR, de Oliveira LC, et al. Effects of physical exercise programs on cognitive function in Parkinson's disease patients: a systematic review of randomized controlled trials. Oxid Med Cell Longev. 2018;2018:1-18. doi:https://doi.org/10.1155/2018/1966758
14. Sharp K, Hewitt J. Dance as an intervention for people with Parkinson's disease: a systematic review and meta-analysis. Neurosci Biobehav Rev. 2014;47:445-456. doi:https://doi.org/10.1016/j.neubiorev.2014.09.009
15. Bose A, Beal MF. Mitochondrial dysfunction in Parkinson's disease. J Neurochem. 2016;139(suppl 1):216-231. doi:https://doi.org/10.1111/jnc.13731

16. Dauer W, Przedborski S. Parkinson’s disease: mechanisms and models. Neuron. 2003;39(6):889-909. doi:https://doi.org/10.1016/S0896-6273(03)00568-3
17. Wong YC, Krainc D. α-synuclein toxicity in neurodegeneration: mechanism and therapeutic strategies. Nat Med. 2017;23(2):1-13. doi:https://doi.org/10.1038/nm.4269
18. Brundin P, Melki R. Prying into the prion hypothesis for Parkinson’s disease. J Neurosci. 2017;37(41):9808-9818. doi:https://doi.org/10.1523/JNEUROSCI.1788-16.2017
19. Steiner JA, Quansah E, Brundin P. The concept of α-synuclein as a prion-like protein: ten years after. Cell Tissue Res. 2018;373(1):161-173. doi:https://doi.org/10.1007/s00441-018-2814-1
20. Tansey MG, Romero-Ramos M. Immune system responses in Parkinson’s disease: early and dynamic. Eur J Neurosci. 2019;49(3):364-383. doi:https://doi.org/10.1111/ejn.14290
21. Dias V, Junn E, Mouradian MM. The role of oxidative stress in Parkinson’s disease. J Parkinsons Dis. 2015;3(4):461-491. doi:https://doi.org/10.3233/JPD-130230
22. Pickrell AM, Youle RJ. The roles of PINK1, parkin, and mitochondrial fidelity in Parkinson’s disease. Neuron. 2015;85(2):257-273. doi:https://doi.org/10.1016/j.neuron.2014.12.007
23. Calabresi P, Picconi B, Tozzi A, Di Filippo M. Dopamine-mediated regulation of corticostriatal synaptic plasticity. Trends Neurosci. 2007;30(5):211-219. doi:https://doi.org/10.1016/j.tins.2007.03.001
24. Zhao YJ, Wee HL, Chan YH, et al. Progression of Parkinson’s disease as evaluated by Hoehn and Yahr stage transition times. Mov Disord. 2010;25(6):710-716. doi:https://doi.org/10.1002/mds.22875
25. Johansson H, Hagströmer M, Grooten WJA, Franzén E. Exercise-induced neuroplasticity in Parkinson’s disease: a metaanalysis of the effects on motor and cognitive outcomes. Front Neurol. 2020;11:1234. doi:https://doi.org/10.3389/fneur.2020.01234
26. Speelman AD, van de Warrenburg BP, van Nimwegen M, Petzinger GM, Munneke M, Bloem BR. How might physical activity benefit patients with Parkinson disease? Nat Rev Neurol. 2015;11(10):563-571. doi:https://doi.org/10.1038/nrneurol.2015.128
27. Frazzitta G, Maestri R, Ghilardi MF, et al. Intensive rehabilitation increases BDNF serum levels in Parkinsonian patients: a randomized study. Neurorehabil Neural Repair. 2014;28(2):163-168. doi:https://doi.org/10.1177/1545968313508474
28. Fisher BE, Wu AD, Salem GJ, et al. The effect of exercise training in improving motor performance and corticomotor excitability in people with early Parkinson’s disease. Arch Phys Med Rehabil. 2008;89(7):1221-1229. doi:https://doi.org/10.1016/j.apmr.2008.01.013
29. Schenkman M, Moore CG, Kohrt WM, et al. Effect of high-intensity treadmill exercise on motor symptoms in patients with de novo Parkinson disease: a phase 2 randomized clinical trial. JAMA Neurol. 2018;75(2):219-226. doi:https://doi.org/10.1001/jamaneurol.2017.3517

30. Lau YS, Patki G, Das-Panja K, Le WD, Ahmad SO. Neuroprotective effects and mechanisms of exercise in a chronic mouse model of Parkinson's disease with moderate neurodegeneration. Eur J Neurosci. 2017;45(1):57-68. doi:https://doi.org/10.1111/ejn.13402
31. Svensson M, Lexell J, Deierborg T. Effects of physical exercise on neuroinflammation, neuroplasticity, neurodegeneration, and behavior: what we can learn from animal models in clinical settings. Neurorehabil Neural Repair. 2015;29(6):577-589. doi:https://doi.org/10.1177/1545968314562108
32. Monteiro-Junior RS, Cevada T, Oliveira BRR, et al. We need to move more: neurobiological hypotheses of physical exercise as a treatment for Parkinson's disease. Med Hypotheses. 2015;85(5):537-541. doi:https://doi.org/10.1016/j.mehy.2015.07.023
33. Aguiar AS Jr, Moreira ELG, Hoeller AA, et al. Exercise attenuates levodopa-induced dyskinesia in 6-hydroxydopamine-lesioned mice. Neuroscience. 2016;321:156-166. doi:https://doi.org/10.1016/j.neuroscience.2016.02.030
34. Real CC, Ferreira AFB, Chaves-Kirsten GP, et al. BDNF receptor blockade hinders the beneficial effects of exercise in a rat model of Parkinson's disease. Neuroscience. 2017;363:114-124. doi:https://doi.org/10.1016/j.neuroscience.2017.09.002
35. Tuon T, Valvassori SS, Lopes-Borges J, et al. Physical training exerts neuroprotective effects in the regulation of neurochemical factors in an animal model of Parkinson's disease. Neuroscience. 2015;285:325-334. doi:https://doi.org/10.1016/j.neuroscience.2014.11.022
36. Hou L, Chen W, Liu X, Qiao D, Zhou F-M. Exercise-induced neuroprotection of the nigrostriatal dopamine system in Parkinson's disease. Front Aging Neurosci. 2017;9:358. doi:https://doi.org/10.3389/fnagi.2017.00358
37. Wrann CD, White JP, Salogiannis J, et al. Exercise induces hippocampal BDNF through a PGC-1α/FNDC5 pathway. Cell Metab. 2013;18(5):649-659. doi:https://doi.org/10.1016/j.cmet.2013.09.008
38. De Miguel Z, Khoury N, Betley MJ, et al. Exercise plasma boosts memory and dampens brain inflammation via clusterin. Nature. 2021;600(7889):494-499. doi:https://doi.org/10.1038/s41586-021-04183-x
39. Mak MKY, Wong-Yu ISK, Shen X, Chung CLH. Long-term effects of exercise and physical therapy in people with Parkinson disease. Nat Rev Neurol. 2017;13(11):689-703. doi:https://doi.org/10.1038/nrneurol.2017.128
40. Herman T, Giladi N, Gruendlinger L, Hausdorff JM. Six weeks of intensive treadmill training improves gait and quality of life in patients with Parkinson's disease: a pilot study. Arch Phys Med Rehabil. 2007;88(9):1154-1158. doi:https://doi.org/10.1016/j.apmr.2007.05.015

41. Corcos DM, Robichaud JA, David FJ, et al. A two-year randomized controlled trial of progressive resistance exercise for Parkinson's disease. Mov Disord. 2013;28(9):1230-1240. doi:https://doi.org/10.1002/mds.25380
42. Cugusi L, Solla P, Serpe R, et al. Effects of a Nordic walking program on motor and nonmotor symptoms, functional performance and body composition in patients with Parkinson's disease. NeuroRehabilitation. 2015;37(2):245-254. doi:https://doi.org/10.3233/NRE-151260
43. Cruise KE, Bucks RS, Loftus AM, Newton RU, Pegoraro R, Thomas MG. Exercise and Parkinson's: benefits for cognition and quality of life. Acta Neurol Scand. 2011;123(1):13-19. doi:https://doi.org/10.1111/j.1600-0404.2010.01338.x
44. Amara AW, Wood KH, Joop A, et al. Randomized, controlled trial of exercise on objective and subjective sleep in Parkinson's disease. Mov Disord. 2020;35(6):947-958. doi:https://doi.org/10.1002/mds.27916
45. Aarsland D, Creese B, Politis M, et al. Cognitive decline in Parkinson disease. Nat Rev Neurol. 2017;13(4):217-231. doi:https://doi.org/10.1038/nrneurol.2017.27
46. Levin O, Netz Y, Ziv G. The beneficial effects of different types of exercise interventions on motor and cognitive functions in Parkinson's disease. NPJ Parkinsons Dis. 2021;7(1):81. doi:https://doi.org/10.1038/s41531-021-00227-z
47. David FJ, Robichaud JA, Leurgans SE, et al. Exercise improves cognition in Parkinson's disease: the PRET-PD randomized clinical trial. Mov Disord. 2015;30(12):1657-1663. doi:https://doi.org/10.1002/mds.26291
48. Goodwin VA, Richards SH, Taylor RS, Taylor AH, Campbell JL. The effectiveness of exercise interventions for people with Parkinson's disease: a systematic review and meta-analysis. Mov Disord. 2008;23(5):631-640. doi:https://doi.org/10.1002/mds.21922
49. van der Kolk NM, King LA. Effects of exercise on mobility in people with Parkinson's disease. Mov Disord. 2013;28(11):1587-1596. doi:https://doi.org/10.1002/mds.25658
50. Fasano A, Canning CG, Hausdorff JM, Lord S, Rochester L. Falls in Parkinson's disease: a complex and evolving picture. Mov Disord. 2017;32(11):1524-1536. doi:https://doi.org/10.1002/mds.27195
51. Allen NE, Sherrington C, Paul SS, Canning CG. Balance and falls in Parkinson's disease: a meta-analysis of the effect of exercise and motor training. Mov Disord. 2011;26(9):1605-1615. doi:https://doi.org/10.1002/mds.23790
52. Li F, Harmer P, Fitzgerald K, et al. Tai Chi and postural stability in patients with Parkinson's disease. N Engl J Med. 2012;366(6):511-519. doi:https://doi.org/10.1056/NEJMoa1107911
53. Ernst M, Folkerts AK, Gollan R, et al. Physical exercise for people with Parkinson's disease: a systematic review and network meta-analysis. Cochrane Database Syst Rev. 2024;4(4):CD013856. doi:https://doi.org/10.1002/14651858.CD013856.pub3

54. Osborne JA, Botkin R, Colon-Semenza C, et al. Physical therapist management of Parkinson disease: a clinical practice guideline from the American Physical Therapy Association. Phys Ther. 2022;102(4):pzab302. doi:https://doi.org/10.1093/ptj/pzab302
55. Langeskov-Christensen M, Franzén E, Grøndahl Hvid L, Dalgas U. Exercise as medicine in Parkinson's disease. J Neurol Neurosurg Psychiatry. 2024;95(11):1077-1088. doi:https://doi.org/10.1136/jnnp-2023-332974
56. Stevens-Lapsley J, Kluger BM, Schenkman M. Quadriceps muscle weakness, activation deficits, and fatigue with Parkinson disease. Neurorehabil Neural Repair. 2016;30(6):533-541. doi:https://doi.org/10.1177/1545968315613449
57. Cancela JM, Mollinedo Cardalda I, Ayán C, de Oliveira IM. Feasibility and effects of highintensity interval training in people with Parkinson's disease: a systematic review. Int J Environ Res Public Health. 2020;17(17):6253. doi:https://doi.org/10.3390/ijerph17176253
58. Vieira de Moraes Filho A, Chaves SN, Martins WR, et al. Progressive resistance training improves bradykinesia, motor symptoms and functional performance in patients with Parkinson's disease. Clin Interv Aging. 2020;15:87-95. doi:https://doi.org/10.2147/CIA.S231359
59. Mehrholz J, Kugler J, Storch A, Pohl M, Hirsch K, Elsner B. Treadmill training for patients with Parkinson's disease. Cochrane Database Syst Rev. 2015;(8):CD007830. doi:https://doi.org/10.1002/14651858.CD007830.pub3
60. Canning CG, Sherrington C, Lord SR, et al. Exercise for falls prevention in Parkinson disease: a randomized controlled trial. Neurology. 2015;84(3):304-312. doi:https://doi.org/10.1212/WNL.0000000000001155
61. Song R, Grabowska W, Park M, et al. The impact of Tai Chi and Qigong mind-body exercises on motor and non-motor function and quality of life in Parkinson's disease: a systematic review and meta-analysis. Parkinsonism Relat Disord. 2017;41:3-13. doi:https://doi.org/10.1016/j.parkreldis.2017.05.019
62. Kwok JYY, Kwan JCY, Auyeung M, et al. Effects of mindfulness yoga vs stretching and resistance training exercises on anxiety and depression for people with Parkinson disease: a randomized clinical trial. JAMA Neurol. 2019;76(7):755-763. doi:https://doi.org/10.1001/jamaneurol.2019.0534
63. Carapellotti AM, Rodger MWM, Doumas M. Evaluating the effects of dance on motor outcomes, non-motor outcomes, and quality of life in people living with Parkinson's: a systematic review and meta-analysis. PLoS One. 2020;15(8):e0236820. doi:https://doi.org/10.1371/journal.pone.0236820
64. Dockx K, Bekkers EMJ, Van den Bergh V, et al. Virtual reality for rehabilitation in Parkinson's disease. Cochrane Database Syst Rev. 2016;(12):CD010760. doi:https://doi.org/10.1002/14651858.CD010760.pub2

65. Parkinson's Foundation. New exercise recommendations for the Parkinson's community and exercise professionals. Published May 25, 2021. Accessed May 19, 2026. https://www.parkinson.org/blog/awareness/exercise-recommendations
66. Alberts JL, Rosenfeldt AB. The universal prescription for Parkinson's disease: exercise. J Parkinsons Dis. 2020;10(s1):S21-S27. doi:https://doi.org/10.3233/JPD-202100
67. DeSimone GT, DiFrancisco-Donoghue J. Exercise for those living with Parkinson's disease. ACSMs Health Fit J. 2024;28(2):7-12. doi:https://doi.org/10.1249/FIT.0000000000000943
68. Radder DLM, Lígia Silva de Lima A, Domingos J, et al. Physiotherapy in Parkinson's disease: a meta-analysis of present treatment modalities. Neurorehabil Neural Repair. 2020;34(10):871-880. doi:https://doi.org/10.1177/1545968320952799
69. Li J, Chen X, Khoo E, et al. Adherence to non-pharmacological interventions in Parkinson's disease: a systematic review. J Parkinsons Dis. 2024;14(2):195-211. doi:https://doi.org/10.3233/JPD-230266
70. Alves B, Dores AR, Barbosa F, et al. MoveONParkinson: implementation of a selfmanagement program to promote exercise behavior in people with Parkinson's disease. Front Public Health. 2024;12:1420171. doi:https://doi.org/10.3389/fpubh.2024.1420171
71. Ellis TD, Cavanaugh JT, Earhart GM, et al. Factors associated with exercise behavior in people with Parkinson disease. Phys Ther. 2015;95(2):183-191. doi:https://doi.org/10.2522/ptj.20130390
72. Chen X, Zhang G, Liu M, He J, Zhang Z. New perspectives on molecular mechanisms underlying exercise-induced benefits in Parkinson's disease. NPJ Parkinsons Dis. 2025;11:256. doi:https://doi.org/10.1038/s41531-025-01113-w
73. Mitchell AK, Bliss RR, Church FC. Exercise, neuroprotective exerkines, and Parkinson's disease: a narrative review. Biomolecules. 2024;14(10):1241. doi:https://doi.org/10.3390/biom14101241
74. Kaagman DGM, van Wegen EEH, Cignetti N, Rothermel E, Vanbellingen T, Hirsch MA. Effects and mechanisms of exercise on brain-derived neurotrophic factor (BDNF) levels and clinical outcomes in people with Parkinson's disease: a systematic review and meta-analysis. Brain Sci. 2024;14(3):194. doi:https://doi.org/10.3390/brainsci14030194
75. Luthra NS, Dorsey ER, Raghavan P, et al. Aerobic exercise-induced changes in fluid biomarkers in Parkinson's disease. NPJ Parkinsons Dis. 2025;11:155. doi:https://doi.org/10.1038/s41531-025-01042-8
76. Ahern L, Timmons S, Lamb SE, McCullagh R. A systematic review of behaviour change interventions to improve exercise self-efficacy and adherence in people with Parkinson's disease using the Theoretical Domains Framework. J Frailty Sarcopenia Falls. 2024;9(1):66-68. doi:https://doi.org/10.22540/JFSF-09-066

77. Oosterhof TH, Darweesh SKL, Schootemeijer S, Bloem BR, de Vries NM. Considerations on how to prevent Parkinson’s disease through exercise. J Parkinsons Dis. 2024;14(s2):S387-S395. doi:https://doi.org/10.3233/JPD-240091