QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

Cite as: JAKUBCZYK, Jan, KOŁKOWSKI, Wiktor, JANISZEWSKA, Anna, WĄDOŁOWSKA, Monika, ANTOSZEWSKA, Magda, GRABOWSKA, Maria, GOZDERA, Katarzyna, KOSZEWSKI, Jakub and PIELAK, Wiktoria. Physical Activity in Parkinson's Disease: Effects on Motor and Non-Motor Symptoms and Implications for Disease Progression. Quality in Sport. 2026;60:72925. https://doi.org/10.12775/QS.2026.60.72925

ARTICLE TIMELINE
Received: 01.06.2026. Revised: 23.06.2026. Accepted: 23.06.2026. Published: 27.06.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines: Medical Sciences; Health Sciences.
Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Nauki medyczne; Nauki o zdrowiu.

Physical Activity in Parkinson's Disease: Effects on Motor and Non-Motor Symptoms and Implications for Disease Progression

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ABSTRACT

Background:
Parkinson's disease is a progressive condition that affects both movement and non-motor functions, with a growing impact on daily functioning over time. Pharmacological treatment remains central, but it does not address the underlying neurodegeneration.

Objective:
This review examines the role of physical activity in Parkinson's disease, focusing on its effects on symptoms, mechanisms underlying these effects, and its relationship with disease progression.

Methods:
A narrative review was conducted using PubMed, Scopus, and Web of Science. The search covered studies published between January 2015 and February 2026 and focused on systematic reviews, meta-analyses, and relevant clinical studies.

Results:
Exercise improves motor function in Parkinson's disease. This is most evident in walking and balance. Benefits are also reported in cognition, mood, and sleep, although their magnitude varies across studies. Possible mechanisms include changes in neural plasticity and metabolic processes, but these are rarely measured directly in clinical research. The effect on disease progression remains unclear. Some studies suggest a slower functional decline, while others do not show a measurable difference.

Conclusions:
Regular physical activity improves functional outcomes and supports long-term management in Parkinson's disease. Its effect on the underlying disease process remains uncertain, but the available evidence is sufficient to support exercise as part of routine care.

Keywords:
Parkinson's disease; physical activity; exercise; neuroprotection; motor symptoms

1. INTRODUCTION

Parkinson's disease is a progressive neurodegenerative disorder characterized by both motor and non-motor symptoms. The clinical picture includes bradykinesia, rigidity, and tremor, but it also extends beyond movement disorders. Non-motor features such as depression, constipation, and disturbed sleep are part of the disease spectrum and can precede the motor syndrome [1].

Pharmacological treatment remains the foundation of clinical management and can reduce motor symptoms and improve quality of life. At present, however, no available therapy has been shown to slow down or arrest disease progression [1]. This has increased interest in complementary approaches that may support long-term outcomes, including exercise [1,2].

Physical activity is one of the most frequently discussed non-pharmacological strategies in Parkinson's disease. Systematic reviews and meta-analyses show that exercise can improve motor performance, particularly in gait, balance, and mobility [3,4,5]. Aerobic exercise has been associated with improvements in gait velocity, stride length, Berg Balance Scale scores, Timed Up and Go performance, 6-minute walk test results, and UPDRS-III scores [4].

The effects of exercise are not limited to motor symptoms. Exercise interventions have also been associated with better cognitive outcomes [6] and may improve non-motor symptoms more broadly, although the evidence is less consistent than for motor outcomes [7].

Proposed mechanisms include changes in neuroplasticity, increased neurotrophic signaling, and broader metabolic or anti-inflammatory effects [8,9]. Exercise-related increases in BDNF have been reported in people with Parkinson's disease, which supports the idea that at least part of the clinical benefit may be biologically mediated [9]. At the same time, most mechanistic pathways are inferred from indirect evidence rather than measured directly in clinical populations [8,9].

Whether physical activity influences disease progression remains less clear. Recent evidence suggests that higher levels of physical activity may be associated with slower progression, but this relationship is still difficult to interpret and is not equivalent to proof of disease modification [10]

Research Objective. To examine how physical activity affects motor and non-motor symptoms in Parkinson's disease. It also considers the mechanisms that may underlie these effects and discusses whether exercise has any influence on disease progression.

Research Problems. It is not clear how consistent the effects of physical activity on motor symptoms are across studies. The impact on non-motor symptoms is more variable, and underlying mechanisms are mostly inferred rather than directly measured. It also remains uncertain whether exercise influences disease progression or mainly improves functional outcomes.

Research Hypotheses. Physical activity improves both motor and non-motor outcomes in Parkinson's disease, but its effect on disease progression is likely limited and remains uncertain.

2. METHODS

A narrative review methodology was applied in this study. The literature search was conducted using PubMed, Scopus, and Web of Science. Additional relevant studies were identified by screening the reference lists of selected articles.

Keywords related to Parkinson's disease and physical activity were used in different combinations. These included "Parkinson's disease," "physical activity," "exercise," "motor symptoms," "non-motor symptoms," "gait," "balance," "mobility," "cognition," "mood," "sleep," "neuroprotection," "BDNF," and "disease progression."

Studies published between January 2015 and February 2026 were considered to ensure that the review reflects recent evidence. Most of the included publications were systematic reviews, meta-analyses, and randomized clinical trials. Selected narrative reviews were also included when they provided useful clinical or mechanistic context.

The analysis focused on physical activity in Parkinson's disease, with emphasis on motor and non-motor symptoms, proposed mechanisms, and possible links with disease progression. Most studies involved adult patients. Research from related areas, such as geriatric rehabilitation, was used only when it added context, particularly for balance, falls, or long-term function.

Articles were selected based on their relevance and their contribution to the aims of the review. Preference was given to studies reporting clinically meaningful outcomes or providing mechanistic insight.

No formal statistical analysis was performed, as this study represents a narrative review. This allows a broader interpretation of the evidence, but it also introduces a risk of selection bias.

3. EFFECTS OF PHYSICAL ACTIVITY ON MOTOR SYMPTOMS

3.1 Gait

Gait impairment is a common and clinically relevant feature of Parkinson's disease. Patients typically walk more slowly, take shorter steps, and show reduced rhythmicity, which affects walking stability [1].

Exercise improves basic gait parameters. Meta-analyses of aerobic training report increases in gait velocity and step or stride length in patients with Parkinson's disease [4]. Similar effects are described in broader reviews of exercise interventions, although the magnitude of change varies across studies [3,5].

Intervention studies report comparable findings. High-intensity treadmill training improves motor performance in early-stage patients, although gait is not always assessed as a primary outcome [11].

Results are not uniform. Differences in intervention type, intensity, and duration limit direct comparison between studies [4,5]. Most evidence is based on short-term interventions, and data on sustained changes in gait are limited.

Exercise consistently affects selected gait parameters, particularly walking speed and step length, but the overall effect remains moderate.

3.2 Balance

Balance problems are common in Parkinson's disease and become more important as postural control worsens. In practice, this means less stability during standing and walking, greater caution in movement, and a higher risk of falls [1].

Exercise can improve balance, but the effect is not identical across studies or across training types. The strongest support comes from meta-analyses showing better performance on balance-related measures after exercise interventions. In aerobic exercise studies, improvement was reported on the Berg Balance Scale, with a pooled effect size that remained statistically significant [4]. A separate meta-analysis also concluded that exercise training improves balance and gait ability and may help prevent falls in people with Parkinson's disease [12].

What matters here is not only whether balance changes, but how those changes are measured. Many studies use clinical tools such as the Berg Balance Scale or Timed Up and Go. These measures are useful, but they do not capture every aspect of postural control. That is one reason why results are sometimes difficult to compare directly across studies [4].

The overall direction of the evidence is still fairly clear. Exercise-based interventions improve balance in Parkinson's disease, even if the size of the effect is usually moderate rather than dramatic. This matters clinically, because small gains in postural control may reduce fall risk and make daily movement safer [4,12].

3.3 Mobility

Reduced mobility in Parkinson's disease is driven by bradykinesia, rigidity, and impaired coordination. It affects everyday tasks such as standing up, turning, and walking short distances and is commonly assessed using functional tests [1].

Exercise leads to measurable improvements in functional mobility. Meta-analyses report reductions in Timed Up and Go time and increases in walking capacity measured by the 6-minute walk test after aerobic and mixed exercise interventions [4].

These findings are supported by systematic reviews showing improvements in functional performance alongside reductions in motor symptom severity [3,5]. Long-term physiotherapy may also support mobility, although results are less consistent across studies [13].

High-intensity exercise improves motor function in early-stage Parkinson's disease and is associated with better performance in functional tasks [11]. Evidence for sustained effects over longer periods remains limited.

The magnitude of improvement is modest, but even small changes in mobility are clinically relevant.

The key findings from selected studies on the effects of physical activity on motor symptoms are summarized in Table 1.

Study	Study Type	Intervention	Sample	Outcome measures	Key findings
Ernst et al., 2023 [3]	Systematic review & network meta-analysis (Cochrane)	Various exercise interventions	156 studies (n = 7939); NMA: 109 studies (n = 4394)	Motor signs (UPDRS-M), quality of life (PDQ-39), adverse events	Most exercise types produced small to moderate improvements in motor symptoms and QoL; dance and gait/balance training showed moderate effects, but differences between exercise types were small
Zhen et al., 2022 [4]	Systematic review & meta-analysis	Aerobic exercise	20 studies (n = 802)	TUG, BBS, gait velocity, stride/step length, UPDRS-III, 6MWT, PDQ-39	Aerobic exercise improved TUG (SMD −0.41), BBS (0.99), gait velocity (0.49), stride/step length (0.32), UPDRS-III (−0.40), and 6MWT (0.35); no significant effect on step cadence or PDQ-39

Choi et al., 2020 [5]	Systematic review & meta-analysis	Exercise therapies (mixed interventions)	18 studies (n = 1144)	UPDRS (total, II, III), MDS-UPDRS III, BBS, TUG, gait velocity, 6MWT, safety	Exercise therapies significantly improved total UPDRS, UPDRS II, and UPDRS III; compared with nonexercise controls, they also improved BBS, TUG, and preferred walking speed. Interventions were relatively safe, although methodological limitations were noted
Shen et al., 2016 [12]	Meta-analysis (RCTs)	Exercise training	25 RCTs	Balance (BBS, TUG), gait ability, falls (fall rate and number of fallers)	Exercise improved balance and gait (Hedges' g = 0.30 short-term; 0.42 long-term) and reduced fall rate (RR ≈ 0.49 short-term; 0.41 long-term), but did not reduce the number of fallers; facility-based training showed greater effects
Okada et al., 2021 [13]	Systematic review & meta-analysis	Long-term physiotherapy	10 studies (n = 663)	Motor symptoms (off-medication), levodopa-equivalent dose (LED)	Long-term physiotherapy improved motor symptoms (−0.65, 95% CI −1.04 to −0.26) and reduced LED (−0.49, 95% CI −0.89 to −0.09); subgroup effects were observed for aerobic exercise and multidisciplinary rehabilitation, although the quality of evidence was low to very low

Schenkman et al., 2018 [11]	Randomized controlled trial (RCT)	High-intensity treadmill exercise	128 patients with de novo Parkinson's disease	UPDRS-III (6-month change)	High-intensity: +0.3 UPDRS vs control: +3.2 UPDRS (P = .03), indicating slower motor decline

4. EFFECTS OF PHYSICAL ACTIVITY ON NON-MOTOR SYMPTOMS

Non-motor symptoms are an important part of Parkinson's disease and often have a strong influence on how patients function in daily life. They include problems with cognition, mood, and sleep, and in many cases they respond less well to medication than motor symptoms [1]. Current evidence suggests that physical exercise influences both motor and non-motor outcomes in Parkinson's disease [14], which is one of the reasons why physical activity is increasingly considered as an additional form of support.

4.1 Cognitive Function

Cognitive impairment is common in Parkinson's disease and affects domains such as attention and executive function, which are important for everyday functioning [1].

Exercise appears to influence cognition, although the effects are not consistent across all domains. A meta-analysis of randomized controlled trials reported improvements in global cognitive function and executive function following exercise interventions [6]. In contrast, no consistent effects were found for memory, visuospatial abilities, or language.

These differences suggest that the impact of exercise is selective rather than general. The observed effects also depend on how cognition is assessed and which outcomes are used.

Overall, physical activity may improve specific aspects of cognitive performance, particularly executive function, but current evidence does not support a uniform effect across all cognitive domains [6].

4.2 Mood and Mental Health

Mood symptoms also occur in Parkinson's disease, and depression is one of the most clinically relevant non-motor problems. It is associated with reduced quality of life and increased disability.

The review by Wu et al. [15] examined the effects of physical activity on depression in Parkinson's disease. Exercise, particularly aerobic training, was associated with a reduction in depressive symptoms and improvements in quality of life. In the analyzed studies, aerobic training led to significant improvements in depression scores measured with the Beck Depression Inventory. The effects varied depending on the type and intensity of the intervention, and not all exercise protocols produced the same results.

More general evidence suggests that physical activity affects both motor and non-motor outcomes in Parkinson's disease [14], although these findings are not specific to mood and do not allow conclusions about individual symptom domains.

Physical activity may reduce depressive symptoms in Parkinson's disease. The most consistent effects are reported for aerobic exercise, while the evidence for other forms of training is less clear. The available data are still limited, and the results depend on the study design and the type of intervention used.

4.3 Sleep Quality

Sleep disturbance is a frequent non-motor problem in Parkinson's disease. It includes difficulty falling asleep, fragmented sleep, and excessive daytime sleepiness, all of which can worsen daily functioning [1].

A meta-analysis by Li and Hu [16] reported that exercise interventions were associated with an improvement in sleep quality in patients with Parkinson's disease. Similar effects were reported across different exercise modalities, including aerobic training, resistance exercise, and mind–body interventions.

Sleep improvements were reported alongside changes in motor function, balance, gait performance, and quality of life. These outcomes were often observed within the same intervention studies.

The included studies were heterogeneous, and intervention protocols varied in duration, intensity, and type. Exercise appears to have a positive effect on sleep quality in Parkinson's disease, although the magnitude of this effect differs between studies and depends on the characteristics of the intervention [16].

Table 2. Effects of Physical Activity on Non-Motor Symptoms in Parkinson's Disease

Study	Study Type	Intervention	Sample	Outcome Measures	Key Findings
Kim et al., 2023 [6]	Systematic review & meta-analysis (RCTs)	Physical exercise interventions (combined, aerobic, strength, flexibility)	21 RCTs (n = 761)	Global cognition, executive function, other cognitive domains (attention/working memory, language, memory, visuospatial function)	Exercise improved global cognitive function (SMD = 0.69) and executive function (SMD = 0.94); no significant effects for attention/working memory, language, memory, or visuospatial function; benefits were observed mainly in combined exercise programs
Wu et al., 2017 [15]	Systematic review	Physical activity interventions (aerobic training, Qigong, Tai Chi, balance training, combined exercise)	11 studies (n = 342)	Depressive symptoms (e.g., BDI, HADS, HDRS), UPDRS/physical function, anxiety, quality of life	Across 11 studies, effects on depression were mixed: some aerobic and combined programs reduced depression scores, while others showed no significant change. Qigong improved non-motor symptoms and depression, and Tai Chi improved balance and selected quality-of-life domains

Li and Hu, 2025 [16]	Systematic review & meta-analysis	Exercise interventions (aerobic, resistance, mind–body, etc.)	62 studies (n = 3274)	Sleep quality, quality of life (PDQ-39)	Exercise improved sleep quality (SMD = −0.55) and quality of life (SMD = −0.38). Subgroup analyses showed benefits for aerobic and traditional Chinese exercise in sleep outcomes, and for aquatic therapy and resistance training in quality of life

5. NEUROPROTECTIVE MECHANISMS

The clinical benefits of physical activity in Parkinson's disease are well documented, but the biological basis of these effects is less clearly defined. Rather than acting through a single pathway, exercise is thought to involve several overlapping mechanisms. Their relative importance likely depends on the type, intensity, and duration of training.

5.1 Neuroplasticity and motor network adaptation

Neuroplasticity is often used to describe how physical activity may influence Parkinson's disease at a mechanistic level. In this context, it refers to functional changes within existing neural networks rather than regeneration of dopaminergic neurons. Some studies report changes in markers linked to neural reorganization, although the available human evidence remains limited and of low methodological quality [17].

Motor impairment in Parkinson's disease is not explained solely by neuronal loss. Altered coordination within motor networks also plays a role. Repeated, task-specific movement practice can lead to more stable patterns of activation in these circuits, typically interpreted as compensatory rather than restorative.

Findings from the review by Johansson et al. are consistent with this interpretation. Different forms of training were associated with changes in markers of neuroplasticity, but the overall level of evidence was rated as very low [17], which limits the strength of these conclusions.

Changes in neurotrophic signaling, including BDNF, have also been observed following exercise, although these findings remain indirect [9].

Taken together, physical activity may support adaptive changes within motor networks in Parkinson's disease. However, current human evidence does not indicate restoration of normal circuitry or reversal of neurodegeneration [9,17].

5.2 Neurotrophic signaling and BDNF

Neurotrophic signaling is considered one of the mechanisms linking physical activity with changes in brain function in Parkinson's disease. Among the factors studied, brain-derived neurotrophic factor (BDNF) is the most consistently reported, given its role in synaptic plasticity and neuronal signaling.

Clinical studies indicate that exercise can increase circulating BDNF levels in patients with Parkinson's disease. Kaagman et al. reported that exercise therapy raises serum BDNF and is associated with improvements in motor symptoms [9], although the magnitude of this effect varies across studies and exercise modalities.

Interpreting these findings is not straightforward. Serum BDNF does not reflect activity within specific brain regions, and methodological differences between studies make direct comparisons difficult. As a result, the relationship between circulating BDNF and functional neural adaptation remains uncertain.

Exercise-induced signaling is also broader than a single molecule. Mitchell et al. describe a group of exercise-responsive factors, referred to as exerkines, which may mediate communication between peripheral tissues and the brain [8]. In this context, BDNF should be viewed as one component of a more complex response.

Overall, physical activity appears to influence neurotrophic signaling in Parkinson's disease, but the link between circulating BDNF and changes in brain function remains indirect [8,9].

5.3 Oxidative Stress, Inflammation, and Mitochondrial Function

Oxidative stress and mitochondrial dysfunction are closely linked to the pathophysiology of Parkinson's disease. Impaired mitochondrial activity disrupts cellular energy balance and promotes the production of reactive oxygen species, contributing to neuronal damage. These processes are also associated with changes in inflammatory signaling [18].

Physical activity may influence these mechanisms. Magaña et al. describe exercise-induced mitochondrial remodeling, including changes in mitochondrial dynamics, oxidative phosphorylation, and redox balance [18]. Such adaptations are associated with improved mitochondrial efficiency and reduced cellular stress.

These effects extend beyond intracellular processes. Exercise also triggers the release of signaling molecules, known as exerkines, which may influence communication between peripheral tissues and the brain and contribute to the regulation of inflammatory pathways [8].

Much of the available evidence comes from indirect markers or experimental models, and its relevance for clinical outcomes remains uncertain.

Overall, physical activity may affect oxidative stress, inflammation, and mitochondrial function in Parkinson's disease, but a direct link to disease modification has not been established [18].

5.4 Systemic Circulation and Brain Support

Physical activity also influences systemic circulation, which may affect brain function in Parkinson's disease. Increased blood flow during exercise can enhance the delivery of oxygen and metabolic substrates to neural tissue.

Mitchell et al. describe exercise-induced signaling molecules, referred to as exerkines, which may support communication between peripheral tissues and the brain and influence inflammatory and metabolic processes [8]. In addition, broader cardiovascular adaptations associated with regular activity may contribute to these effects [2].

The role of cerebral perfusion in Parkinson's disease remains difficult to define. Most available findings are indirect, and evidence linking changes in circulation to disease progression is limited.

Physical activity may therefore support brain function through improved circulation and substrate delivery, although current data do not indicate a direct effect on neurodegeneration [2,8].

5.5 Integration of mechanisms

The mechanisms described above do not act in isolation. Changes in neural networks, neurotrophic signaling, mitochondrial function, and systemic circulation are likely interconnected and may occur in parallel in response to physical activity.

Exercise does not operate through a single pathway but through combined effects across different levels of biological organization. This makes it difficult to separate individual mechanisms and may partly explain the variability reported between studies.

These processes are consistent with observed functional improvements, while their relevance for disease progression remains unclear.

6. PHYSICAL ACTIVITY AND DISEASE PROGRESSION

6.1 Evidence from clinical studies

Clinical studies on physical activity in Parkinson's disease report improvements in standard clinical measures. These outcomes, however, are not intended to assess disease progression.

Measures such as motor performance or functional mobility reflect changes in symptoms rather than the underlying neurodegenerative process. Higher scores therefore indicate improved performance or compensation, not necessarily slower progression.

In some cases, benefits persist beyond the intervention period, particularly in structured and longer-term programs [19]. Such observations remain limited and do not establish a disease-modifying effect.

Only a small number of studies address progression directly, and their findings are inconsistent. Variation in study design, intervention type, and outcome measures further limits comparability, while progression-specific markers are rarely used [20].

Taken together, available data point to a role of physical activity in symptom management, while its effect on disease progression remains unclear.

6.2 Functional improvement vs disease modification

Improvements following exercise interventions are often interpreted as clinically meaningful, yet they do not necessarily reflect changes in the disease process.

Common outcome measures, including motor scales and functional tests, capture performance rather than neurodegeneration. Better results may arise from more efficient movement or compensatory strategies without altering the underlying pathology.

This distinction is particularly relevant in Parkinson's disease. Adaptive changes within neural networks can obscure ongoing degeneration, so functional gains may occur alongside continued disease progression.

Reports of longer-term effects exist, but they do not provide evidence of disease modification. Without progression-specific markers, it remains difficult to determine whether physical activity alters the course of the disease or primarily affects its clinical expression.

In this context, exercise is best understood as a symptomatic intervention, with no confirmed effect on disease progression.

6.3 Long-term effects and progression-related uncertainty

The long-term impact of physical activity in Parkinson's disease remains uncertain. Most studies examine relatively short intervention periods, typically lasting weeks or months, which do not correspond to the time scale of disease progression [6,16].

Some data suggest that benefits may persist after the intervention, particularly in structured programs [19]. These findings are not consistent and are difficult to compare across studies.

Parkinson's disease develops over years. Short-term changes in clinical outcomes therefore provide limited insight into long-term progression. Whether repeated or sustained physical activity can influence the trajectory of the disease is still unclear.

Evidence with extended follow-up is limited, and results vary across studies. Differences in design, intervention type, and outcome measures further complicate interpretation [20].

Current data describe functional changes within a restricted time frame rather than clear effects on disease progression.

6.4 Summary of current evidence

Available evidence does not demonstrate a disease-modifying effect of physical activity in Parkinson's disease. Observed changes in clinical outcomes are more consistent with improvements in function than with altered progression.

At the same time, similar findings across multiple studies indicate that physical activity has a meaningful role in clinical management. Reported benefits include improved motor performance, balance, gait, and selected non-motor symptoms, all of which affect daily functioning and quality of life.

Interpretation is constrained by the short duration of most studies and the lack of progression-specific markers.

Physical activity therefore contributes to symptom control and functional capacity, while its influence on disease progression remains uncertain

7. Discussion

Physical activity is consistently linked with better motor and non-motor functioning in Parkinson's disease. These effects appear across different exercise models and across different outcome measures, which supports its role in clinical care.

The biological basis of these effects is less clear. Neuroplastic adaptation, neurotrophic signaling, mitochondrial remodeling, and systemic physiological responses have all been proposed as contributing mechanisms. They are unlikely to act separately. More probably, they overlap and interact during repeated training.

The connection between these biological changes and clinical outcomes remains indirect. Much of the available evidence comes from surrogate markers, short interventions, or mechanistic studies that do not show how molecular changes translate into sustained clinical benefit.

This becomes more important when disease progression is considered. Better results in motor scales, gait measures, or functional tests show that patients function better, but they do not show that the neurodegenerative process has slowed. In Parkinson's disease, compensation can improve performance while degeneration continues.

Study duration is another problem. The disease develops over years, whereas many exercise studies last only weeks or months. Some benefits persist after the intervention, but that alone is not enough to argue for disease modification.

Physical activity has a clear place in symptom management and functional support in Parkinson's disease. Its effect on disease progression remains uncertain.

8. Limitations

The available literature on physical activity in Parkinson's disease is heterogeneous. Exercise protocols differ in type, intensity, and duration, while outcome measures are not consistent across studies. This limits direct comparison and reduces the strength of pooled interpretations.

Follow-up periods are often short relative to the time course of the disease. Most interventions are evaluated over weeks or months, which restricts conclusions about long-term effects and disease progression.

Assessment of progression remains a major limitation. Commonly used clinical scales, including the Unified Parkinson's Disease Rating Scale, reflect symptom severity and functional status rather than underlying neurodegeneration. As a result, it is difficult to distinguish symptomatic improvement from true modification of the disease process.

Another issue concerns study design. Sample sizes are frequently small, and intervention protocols are not standardized. Differences in patient characteristics, disease stage, and baseline activity levels further complicate interpretation of results.

As a narrative review, this work is also subject to selection and interpretation bias. The included studies reflect the available literature, which may not capture the full scope of evidence.

10. CONCLUSIONS

Exercise improves functional outcomes in Parkinson's disease, including mobility, daily functioning, and selected non-motor symptoms. These effects are consistent and clinically relevant.

Current evidence does not demonstrate an effect on disease progression. Improvements are best understood as changes in function rather than modification of the neurodegenerative process.

In clinical practice, physical activity should be part of long-term management. Combined exercise programs appear to provide the most consistent benefits.

Future research should focus on long-term outcomes and progression-specific measures to clarify the role of exercise across different stages of the disease.

Disclosures

Author's contribution:

Conceptualization: JJ, AJ, WK
Methodology: MW, KG, WP
Formal analysis: MG, JJ, WP
Investigation: MA, JK, KO
Resources: AJ, WK
Data curation: MW, KG
Writing – original draft: MA, MG

Writing – review & editing: KO, JK

Visualization: JJ, MW, MG
Supervision: AJ
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All authors have read and approved the published version of the manuscript.

Funding: The study did not receive external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Acknowledgements: Not applicable.

Conflicts of Interest: The authors declare no conflicts of interest.

Declaration of the use of generative AI and AI-assisted technologies in the writing process:

During the preparation of this work, the authors used ChatGPT for the purpose of language editing and stylistic refinement to improve clarity and readability of the manuscript. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the accuracy, integrity and conclusions of the publication.

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