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Is neurofeedback A reliable therapy for managing Parkinson's Disease?

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ABSTRACT

Objective: Standard treatments for Parkinson's disease (PD) often lose effectiveness over time, motivating interest in complementary, non-pharmacological approaches. Neurofeedback, which trains patients to consciously modulate brain activity, has shown theoretical promise in PD; however, its clinical value remains uncertain. We conducted a PRISMA-guided systematic review to evaluate the current evidence.

Methods: We included studies of patients with idiopathic PD undergoing any form of neurofeedback training. Case studies were excluded to reduce intersubject variability. Sixteen studies met inclusion criteria; after methodological quality appraisal, twelve were retained for qualitative synthesis.

Results: Neurofeedback modalities included functional magnetic resonance imaging (n = 4), electroencephalography (n = 4), and deep brain stimulation (n = 4). Across modalities, patients generally learned to voluntarily modulate targeted neural signals. However, translation of neural self-regulation into improvement on validated clinical measures or task-specific behavioral outcomes was limited and inconsistent. Interpretation was constrained by small sample sizes and frequent absence of standardized effect size reporting, which weakens statistical robustness and complicates the distinction between true null effects and underpowered findings.

Conclusions: While patients with PD can self-modulate brain activity, current evidence does not demonstrate consistent therapeutic benefit.

Significance: Despite a compelling mechanistic rationale, available data are insufficient to support neurofeedback as an effective treatment for PD.

1. Introduction

Parkinson's disease (PD) is one of the most prevalent neurodegenerative chronic disorders, affecting over 8 million people globally as of 2019, according to the World Health Organization (Ou et al., 2021). Additionally, PD is the fastest-growing neurological disorder, driven by factors such as an aging population, possible changes in environmental or social risk factors, and enhanced detection and diagnosis in routine medical practice (GBD 2016 Neurology Collaborators, 2019). In 2040, the number of PD cases is estimated to be between 12 and 17 million (Dorsey et al., 2018). Therefore, the management and treatment of this disease will pose a significant public health challenge.

PD is due to the progressive depletion of dopaminergic neurons in the substantia nigra pars compacta and in the ventral tegmental area, but it is becoming increasingly evident that other neurotransmitter pathways are also affected (Schapira et al., 2017). PD is characterized by motor manifestations (e.g., bradykinesia, rigidity, tremor, gait and balance disorders) and non-motor symptoms affecting the gastrointestinal system, autonomic nervous system, sleeping patterns, mood, and cognition (Chaudhuri and Schapira, 2009, Schapira et al., 2017). While motor symptoms are consistently the most prominent in PD, there is now a widely accepted agreement that PD is not just a motor disorder, but rather a complex syndrome with multiple facets. Non-motor symptoms play a significant role in determining the quality of life and can lead to significant disability (Chaudhuri and Schapira, 2009, Schapira et al., 2017). Nevertheless, these signs have been underestimated or hardly considered until recently. Dopamine replacement therapy (DRT) is the standard treatment for PD. It consists of the dopamine precursor levodopa (L-dopa), dopamine agonists, and dopamine catabolism inhibitors, such as monoamine oxidase B inhibitors and catechol-O-

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methyltrasferase inhibitors (Olanow et al., 2009, Schapira et al., 2009). This approach achieves optimal control of motor symptoms in the initial stages of PD. However, as PD advances and medication dosages increase, the duration of benefits diminishes, while side effects of DRT, such as motor fluctuations and dyskinesia, begin to appear (Schapira et al., 2009). Neurosurgical interventions, including pallidotomy and deep brain stimulation (DBS) of the subthalamic nucleus (STN) and the internal segment of the globus pallidus, can effectively manage advanced PD by significantly improving motor complications and reducing substantially (20–65%) the need for medication (Benabid, 2003, Benabid

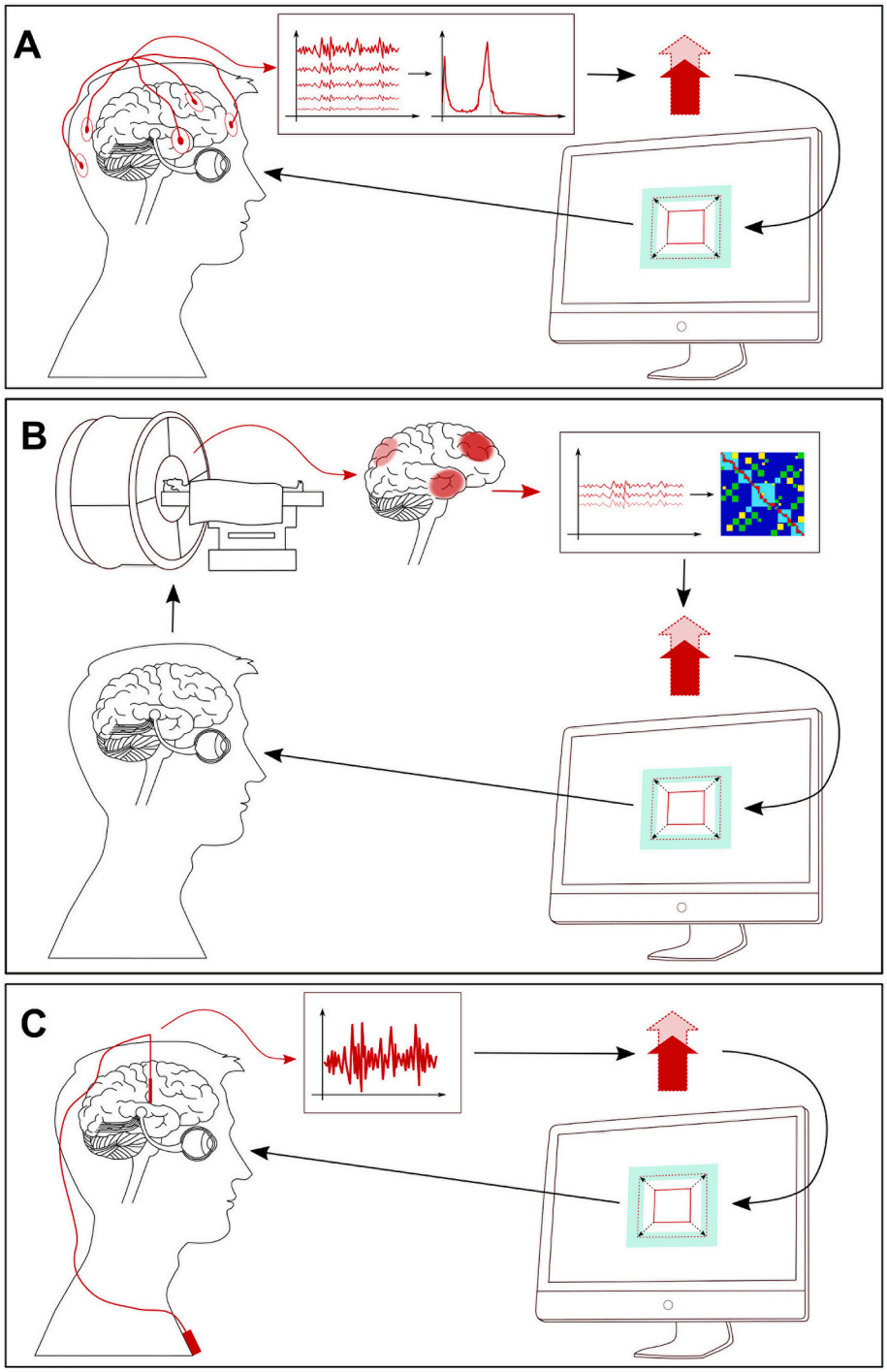

Fig. 1. Neurofeedback training techniques in Parkinson's disease patients. (A) Electroencephalography (EEG)-based neurofeedback: a non-invasive method in which scalp electrodes record cortical activity. Raw signals are transformed into the time–frequency domain (using Fourier or Wavelet transforms) to extract frequency bands of interest. The voltage fluctuations of the EEG can also be time-locked to the processing of specific sensory, cognitive, or motor events and analyzed in terms of event-related potentials (ERP). Changes in neural activity in a selected frequency band or in the ERP control the size of a square on a computer screen. **(B)** Functional-magnetic resonance imaging-based neurofeedback: a non-invasive technique in which brain activity is inferred indirectly from Blood Oxygenation Level–Dependent signal changes that reflect local neuronal activity. **(C)** Local field potential-Deep Brain Stimulation-based neurofeedback: an invasive approach using electrodes implanted in the basal ganglia (typically subthalamic nucleus, less often globus pallidus pars interna). Raw signals are analyzed as in EEG and used for feedback. In all modalities, signals are processed in real time and converted into sensory feedback, enabling patients to learn self-regulation of brain activity.

et al., 2009). Nevertheless, these interventions are costly, involve surgical risks, and may lead to adverse effects such as acute depression (Accolla and Pollo, 2019). Additionally, while standard medical treatments alleviate some non-motor symptoms, others may worsen or even arise as a result of DRT (Schaeffer and Berg, 2017, Schapira et al., 2009). Therefore, currently, two major therapeutic challenges for PD are developing treatments to slow or prevent neurodegeneration and creating effective symptomatic interventions for non-motor features. The significance of this work is crucial since the progression of PD leads to more difficulty in controlling it, both from a medical and social perspective. Patients increasingly rely on both paid and unpaid care, which places a heavier load on caregivers. This increasing tension leads to a steady deterioration in the quality of life of patients (Chaudhuri and Schapira, 2009, Schrag et al., 2000). Although patients exhibit objectively adequate control over motor disturbances, they frequently report a decline in their overall sense of well-being. Furthermore, as the disease advances, the financial burden on patients, caregivers, and society increases considerably. Consequently, there has been a significant rise in efforts to develop complementary therapies that can be integrated with conventional treatment, such as dietary modifications, supplements, physical exercise, mindfulness, and art therapy (Kola and Subramanian, 2023, Rabin et al., 2015). While some therapies, like physical exercise, have documented benefits (Ernst et al., 2024), further randomized controlled trials are necessary. Therefore, other interventions have been proposed, and among them, neurofeedback has received increasing attention. Neurofeedback is a non-pharmacological approach that involves recording, analyzing, and converting participants' brain activity into real-time visual, haptic, or auditory feedback. By engaging with this sensory representation of their brain activity, participants learn to intentionally adjust specific neural patterns based on operant conditioning principles, reinforcing desirable brain activity through positive feedback while minimizing undesirable patterns using negative feedback (Fig. 1). The goal of this training is to enable individuals, through repeated sessions, to progressively acquire the ability to self-regulate their brain activity beyond the experimental environment (Marzbani et al., 2016, Sitaram et al., 2017).

There has been growing interest in utilizing neurofeedback training as a tool to alleviate symptoms in PD patients. Neurofeedback training protocols typically target characteristic pathophysiological alterations of PD brain activity, aiming to teach patients to voluntarily shift those patterns toward profiles seen in healthy individuals. Two main approaches have been pursued.

One stream of research has focused on evidence linking abnormal neural oscillations, particularly in the beta band (12–30 Hz), to the pathophysiology of PD (Hammond et al., 2007, Oswal et al., 2013). In healthy individuals, scalp electrophysiological (EEG) studies showed that self-paced unilateral limb movements, as well as motor imagery, are accompanied by beta event-related desynchronization (ERD) preceding and during the movement, followed by beta event-related synchronization (ERS) after movement offset, primarily in the contralateral sensorimotor cortex (Fry et al., 2016, Pfurtscheller and Lopes da Silva, 1999, Zhang et al., 2020). A similar pattern has been reported in epilepsy patients with preserved motor function, suggesting that these dynamics represent a core feature of motor system physiology (Babiloni et al., 2016, Crone et al., 1998, Miller et al., 2007, Pfurtscheller et al., 2003). Beta ERD has been proposed to reflect enhanced local processing during movement planning and execution, processes that are otherwise gated by beta activity at rest (Joundi et al., 2012, Pogosyan et al., 2009). Several studies have shown that PD patients exhibit abnormally high beta-band synchronization at rest within the sensorimotor cortex (Pollok et al., 2012, Silberstein et al., 2005), the basal ganglia (Kühn et al., 2004, Little et al., 2012) and between these regions (Gilron et al., 2021, Litvak et al., 2011). Notably, resting beta-band levels in PD patients show a direct correlation with the severity of two cardinal motor symptoms, rigidity and bradykinesia (Chen et al., 2010, Kühn et al., 2009, Kühn et al., 2004, Little et al., 2012). Both DRT (Weinberger et al., 2006) and high-frequency DBS (Kühn et al., 2008) reduce pathological beta-band synchronization across the cortico–basal ganglia network, including the STN, thereby improving these symptoms. Together with evidence from healthy subjects showing a causal link between increased beta activity and movement slowness (Joundi et al., 2012, Pogosyan et al., 2009), these findings support the notion that beta oscillatory activity plays a crucial role in PD pathophysiology. The possibility of directly recording local field potentials (LFP) from DBS electrodes implanted in nuclei such as the STN has stimulated neurofeedback approaches based on DBS. Compared with scalp EEG, this approach provides substantially higher spatial specificity and signal-to-noise ratio, enabling real-time monitoring of pathological beta-band oscillations and therefore offering a promising target for neurofeedback interventions (Bichsel et al., 2025). However, DBS-based neurofeedback has the important limitation of requiring an invasive neurosurgical procedure and is therefore applicable only to patients who already undergo DBS implantation for clinical reasons.

The other approach draws on real-time recordings of brain activity in predefined regions using functional magnetic resonance imaging (fMRI). fMRI offers substantially higher spatial resolution than EEG remaining non-invasive, unlike DBS. Its most commonly employed signal, the blood oxygenation level–dependent (BOLD) signal, provides an indirect index of neural activity by capturing changes in blood oxygenation, cerebral blood flow, and blood volume that accompany neural processes reflected in local field potentials (Logothetis, 2002). Most investigations have focused on the supplementary motor complex (SMC), located in the medial region of Brodmann's area 6 (Penfield and Welch, 1951). The SMC comprises two distinct subdivisions, the supplementary motor area (SMA) and the pre-SMA, which differ in both their connectivity and functional roles (Nachev et al., 2008). Both regions, however, are affected in PD. Early studies reported that the SMC shows an overall reduction in activation during complex hand motor tasks compared to healthy individuals (Haslinger et al., 2001, Playford et al., 1992), yet subsequent evidence points to a more nuanced picture. Specifically, pre-SMA activity has consistently been found to be reduced in PD patients during tasks requiring complex hand movements (Mallol et al., 2007), whereas the SMA often shows increased activation during the execution of sequential movements (Caproni et al., 2013, Sabatini et al., 2000). Thus, although there is broad consensus that SMC dysfunction contributes to the characteristic deficits in sequential motor performance in PD, the underlying dynamics remain unclear (Rahimpour et al., 2022). The SMC has also been linked to gait disturbances, one of the most disabling symptoms of PD (Brugger et al., 2020, Martin et al., 2025, Wróbel et al., 2025). Recent evidence indicates that structural and functional alterations in SMCs are associated with gait impairments. Wróbel et al. (2025), using the diffusion-weighted MRI, demonstrated that microstructural changes in the white matter of the right SMA were associated with bilateral deficits in gait control. Martin et al. (2025) reported that changes in gray matter volume of the right SMC could distinguish the PD subtype characterized by postural instability and gait impairments. From a functional perspective, Brugger et al. (2020) showed that the readiness potential over the SMC, a marker of voluntary movement initiation (Fried et al., 2011, Libet, 1985), was reduced in PD patients with freezing of gait compared to those without. In the same group, beta ERD over the SMC was also less attenuated. Moreover, the authors demonstrated a causal link between beta synchronization and gait initiation: enhancing beta synchronization via intermittent theta-burst stimulation impaired gait initiation. Brugger et al. (2020) suggested that the coexistence of a reduced readiness potential and enhanced beta synchronization in the SMC hampers the neural processes necessary for initiating gait. The pivotal role of the SMC in PD stems from its extensive reciprocal anatomical connections with both the basal ganglia and the cerebellum (Akkal et al., 2007). Inputs from the basal ganglia reach the SMC through the internal segment of the globus pallidus, whereas cerebellar inputs arise from the dentate nucleus and are relayed via the thalamus. In turn, projections from the SMC to the STN, one of the input

hubs of the basal ganglia, form the hyperdirect pathway (Nambu et al., 2002), which is thought to enable rapid interruption of behavior and cognition in response to stop signals (Mirabella et al., 2013, Mirabella et al., 2012).

A recent study by Oswal et al. (2021) demonstrated a link between beta-band synchronization in the corticobasal ganglia circuit and the SMC activity. Specifically, they found that coherence between the SMA and STN in the high-beta range (21–30 Hz) was correlated with the fiber density of the hyperdirect pathway. Moreover, directional analyses indicated that SMA activity drives STN activity, suggesting that high-beta oscillations propagate from the cortex to the basal ganglia rather than the reverse.

These findings have prompted the idea that training PD patients to modulate beta rhythms or SMC activity could, in theory, alleviate symptoms such as rigidity, bradykinesia, and gait disturbances. However, evidence from neurofeedback training remains inconclusive, largely due to the absence of robust and well-controlled experimental designs (Anil et al., 2021, Ubeda Matzilevich et al., 2024). Nonetheless, given its potential in symptom management, we revisit the existing literature to evaluate whether neurofeedback could serve as a therapeutic adjunct to current PD treatments. Our analysis differs from previous reviews in two key ways: (a) we excluded single-case studies to minimize intersubject variability, and (b) because most studies involve small sample sizes, we scrutinized the statistical analyses to better judge the robustness of the findings. We included studies employing various methods for recording brain activity, both invasive (DBS) and non-invasive (EEG and fMRI), to assess whether certain neurofeedback approaches may be more effective than others in treating PD symptoms (Fig. 1). The primary aim of this review is to determine whether neurofeedback training improves the severity of PD motor symptoms. A secondary aim is to assess whether specific modalities or protocol parameters, such as training duration and targeted brain activity, play a role in producing clinical improvements.

2. Materials and methods

This *meta*-analysis was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Liberati et al., 2009, Page et al., 2021). The protocol for this review was not registered prior to its conduct.

2.1. Eligibility criteria

The review focused on the analysis of the evidence about the effectiveness of neurofeedback as therapy for managing motor symptoms associated with PD. The authors defined a set of eligibility criteria a priori, and any doubts emerging during the selection process were resolved by consensus.

To be included in this review, studies had to meet the following criteria:

a) Employ adult subjects with idiopathic or familial PD. Studies including subjects with atypical parkinsonism or animals were excluded.
b) Include experimental groups with multiple participants to reduce susceptibility to substantial inter-subject variability; therefore, case studies were excluded.
c) Involve neurofeedback based on brain-derived signals (e.g., EEG, LFP recorded from implanted DBS electrodes, or BOLD signals measured using fMRI). Studies employing biofeedback modalities not directly linked to neural activity were excluded.
d) Include quantitative outcome measures and their statistical analyses.
e) Be written in English and published in international peer-reviewed sources. Narrative or systematic reviews, as well as *meta*-analyses, were excluded.

2.2. Search strategy

Systematic literature searches were conducted on September 16, 2025, querying the Scopus (https://www.scopus.com/) and PubMed (pubmed.ncbi.nlm.nih.gov/) databases. No restrictions were placed on publication date; however, the search was limited to documents written in English. The literature search was conducted using the following search string: TITLE-ABS-KEY (Parkinson*) AND TITLE-ABS-KEY (human* OR patient* OR people OR individual*) AND TITLE-ABS-KEY (neurofeedbac* OR "neur*-feedbac*" OR "neur* feedback*"). The keywords had to appear in the title or abstract. Duplicate removal and selection process were performed using Rayyan (https://www.rayyan.ai, Ouzzani et al., 2016).

2.3. Study selection

Two authors (GM, CA) reviewed the search results independently to ascertain their eligibility. Disagreements about article eligibility were rare, occurring in only 6% of cases. These disagreements were resolved through discussion between the authors.

2.4. Quality appraisal

All selected papers were quasi-experimental studies (QES) and randomized control trials (RCT). Two authors (GM, MG) independently evaluated strengths and weaknesses of the methodological approaches, and investigated how effectively biases were addressed using the Joanna Briggs Institute Critical Appraisal Tools for QESs (Barker et al., 2024) and RCTs (Barker et al., 2023), respectively. This scoring system comprises nine criteria for QESs and thirteen for RCTs, focusing exclusively on methodological aspects of the studies, such as sample selection, reliability of the measurement of key variables, and the appropriateness of statistical analyses (see Tables S1 and S2 in the Supplementary Material for the complete lists of items). To facilitate quantitative comparison, we translated the JBI's categorical responses (yes/no/unclear) into a numerical scoring scheme. Assessors selected one of four options for each criterion: yes (1 point), no (0 points), unclear (0.5 points), or not applicable (N/A). Operational definitions guiding these judgments are provided in the Supplementary Material. Criteria deemed as not applicable were excluded from the calculation of the study's total score. For each study, the scores across criteria were summed and then divided by the maximum possible score, 9 for QESs or 13 for RCTs when all criteria applied. Studies were classified according to the score ranges reported in Table 1, and those not reaching the minimum threshold of 50% were excluded.

2.5. Data Extraction and synthesis of results

A structured proforma was used to collect the information extracted from the selected studies. The proforma synthesizes Exposure and Outcome of each study, and especially keeps track of its main results, as brain changes and UPDRS III or other clinical scales outcomes. Additional information (e.g., unconventional medication conditions, limited number of subjects, peculiar aspects in the protocol) is reported in a Notes area.

Table 1
Score rating applied to the analyzed papers.

Score	Rating
≥80%	high quality and not biased
66–80%	satisfactory quality and not heavily biased
51–65%	medium–low quality and with biases
≤50%	low quality and heavily biased

3. Results

3.1. Search results

The literature search on neurofeedback training in PD yielded 160 articles, which were subsequently reduced to 119 following the elimination of duplicates (Fig. 2). Upon screening titles and abstracts based on predefined inclusion/exclusion criteria, 28 articles remained. Subsequently, the full texts of these articles were examined for compliance with the eligibility criteria, resulting in a final selection of 16 articles.

3.2. Methodological quality assessment

Tables 2 and 3 show the results of the quality assessment of the 16 selected articles for studies classified as Randomized Controlled Trials (RCTs, n = 6) and quasi-experimental studies (n = 10), respectively. Two assessors (GM, MG) initially agreed on 12 of the 16 articles (~75%). After discussion, they reached full consensus on all articles. Among the 16 studies included, four were rated as high quality (25%), seven as satisfactory (43.75%), and one as medium–low quality (6.25%). Detailed justifications for any criterion scored below 1 are provided in the Supplementary Materials.

3.3. Outcomes of selected studies

In this section, we present a summary of the main findings from the 12 selected studies, grouped according to the neurofeedback method employed: (a) non-invasive hemodynamic recordings using fMRI (n = 4), (b) non-invasive electrophysiological recordings using EEG (n = 4), and (c) invasive electrophysiological recordings using DBS (n = 4).

3.3.1. Outcomes of studies using fMRI

fMRI-based neurofeedback studies in PD have generally reported convergent findings (Table 4). Across studies, patients were typically able to modulate targeted brain activity following training. Specifically, three studies demonstrated that patients could either increase SMA

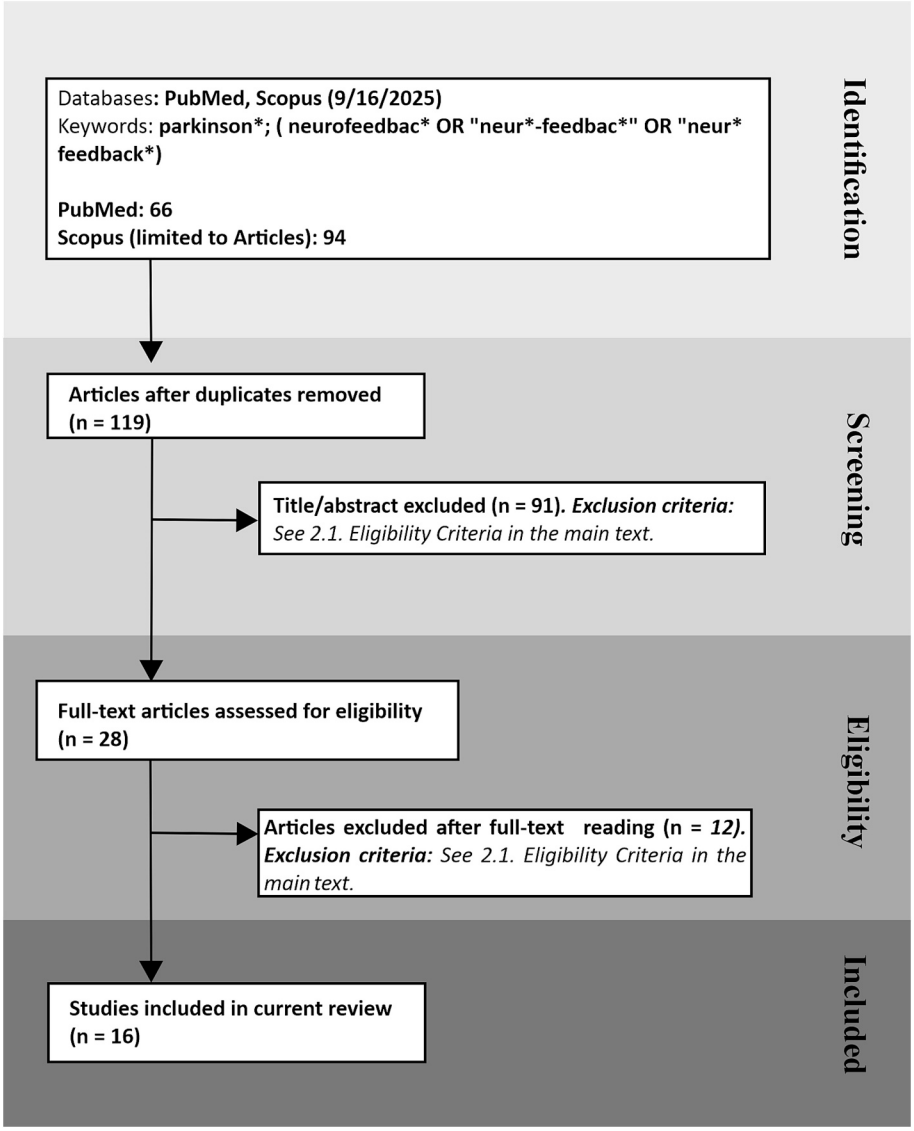

Fig. 2. Flowchart of the search for studies on neurofeedback training in Parkinson's disease in PubMed and Scopus, conducted on September 16, 2025. We restricted the search to publications in which the keywords appeared in the title or abstract, with no date constraints. For the Scopus database, an additional criterion was applied, limiting the search exclusively to documents categorized as articles. The screening process was carried out in two phases. First, titles and abstracts were reviewed; subsequently, full texts were assessed. Studies not meeting the eligibility criteria specified in Section 2.1 were excluded, leaving 16 articles that met the requirements.

Table 2
Summary of the quality assessment for Randomized Controlled Trials.

	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10	Q11	Q12	Q13	Score (%)
Subramanian et al. (2011)	0	0.5	0.5	0.5	0	1	0.5	1	1	1	1	0	1	61.5
Erickson-Davis et al. (2012)	0	0	0	1	0	1	0	1	1	1	0	0	0	**38.5**
Azarpaikan et al. (2014)	1	0	1	1	0	1	1	1	1	1	1	0	1	76.9
Subramanian et al. (2016)	1	0.5	1	0.5	0	1	0.5	1	1	1	1	0	1	73.1
Tinaz et al. (2022)	1	0.5	1	0.5	0.5	1	0.5	1	1	1	1	0.5	1	80.8
Romero-Muñoz et al. (2024)	1	1	0.5	1	0	1	1	1	1	1	1	0.5	1	84.6
Cohen's k	0.67	0.71	1	0.67	1	1	0.75	1	1	1	1	0.67	1	

Notes. Each study was assessed question by question using the JBI evaluation tool for Randomized Controlled Trials. Responses were coded as '1′ (yes), '0′ (no), or '0.5′ (unclear). The final score for each paper was obtained by summing the individual scores, normalizing by the maximum possible score (13), and expressing the result as a percentage. Cohen's k indicates the inter-rater agreement for each question. In bold, excluded studies. Abbreviation: Q = question

Table 3
Summary of the quality assessment for quasi-experimental studies.

	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Score (%)
Fumuro et al. (2013)	1	0	1	0.5	1	0.5	1	1	0	66.7
Khanna et al. (2017)	0	0	0	N/A	N/A	1	0	1	0	**28.6**
Tinaz et al. (2018)	1	1	1	0	1	1	1	1	0	77.8
Fukuma et al. (2018)	1	0	1	N/A	1	1	1	1	0	75.0
He et al. (2019)	1	0	1	N/A	N/A	0	1	0	0	**42.8**
He et al. (2020)	1	0.5	1	N/A	N/A	1	1	1	0	78.6
Bichsel et al. (2021)	1	1	1	N/A	1	1	1	1	0	87.5
Rouzitalab et al. (2023)	1	0	1	N/A	N/A	1	1	1	0	71.4
Rohr-Fukuma et al. (2024)	1	0	1	N/A	1	1	0	0	0	**50.0**
Cooke et al. (2024)	1	0.5	0.5	N/A	1	0.5	1	1	1	81.3
Cohen's k	0.63	0.69	0.63	0.71	1	0.80	1	0.76	0.63	

Notes. Each study was assessed question by question using the JBI evaluation tool for Quasi-experimental studies. Responses were coded as '1′ (yes), '0′ (no), or '0.5′ (unclear). If a criterion was deemed not applicable (N/A) it was excluded from the overall score of the paper. The final score for each paper was obtained by summing the individual scores. normalizing by the maximum possible scores (9 or 7) and expressing the result as a percentage. Cohen's k indicates the inter-rater agreement for each question. In bold, excluded studies. Abbreviation: Q = question.

activation (Subramanian et al., 2011, Subramanian et al., 2016) or enhance functional connectivity between the right insula and the dorsomedial frontal cortex (Tinaz et al., 2018). In contrast, the study with the largest sample size failed to demonstrate reliable neurofeedback-specific modulation of the targeted functional connectivity (Tinaz et al., 2022).

At the clinical level, effects were limited or inconsistent. Findings on task-based measures were similarly mixed and did not consistently translate into clinical benefit. Two studies did not report improvements in UPDRS part III scores or in behavioral motor tasks following training (Tinaz et al., 2022, Tinaz et al., 2018). One RCT reported an improvement in UPDRS part III scores after training, but this effect did not differ significantly from the active control condition, and no improvements were observed across several behavioral tasks (Subramanian et al., 2016). Only the earliest proof-of-concept study reported improvements in both UPDRS part III scores and finger-tapping speed, restricted to the neurofeedback group (Subramanian et al., 2011). However, interpretation of these findings is limited by the very small sample size and the absence of reported effect sizes for both clinical and task-based outcomes.

In summary, fMRI-based neurofeedback reliably affects neural measures of self-regulation, but these changes do not consistently translate into improvements in clinically validated motor symptom scales in PD.

3.3.2. *Outcomes of studies using EEG*

EEG-based neurofeedback studies showed a pattern of results similar to those reported in fMRI-based studies (Table 5). In most cases, patients with PD were able to voluntarily modulate EEG activity during training. However, evidence for clinical benefit was limited. Among these studies, only the one with the smallest sample size (n = 8) reported a significant improvement in balance-related clinical scale scores, without reporting effect sizes and thereby weakening confidence in the strength of the reported findings (Azarpaikan et al., 2014). Fumuro et al. (2013) did not assess clinical or behavioral outcomes. In contrast, both Cooke et al. (2024) and Romero-Muñoz et al. (2024) found no improvement in validated clinical measures following EEG-based neurofeedback alone. With respect to task-based measures, findings were similarly inconsistent. Cooke et al. (2024) reported a reduction in reaction time during a precision handgrip task without improvement in accuracy, whereas Romero-Muñoz et al. (2024) observed no task-level improvements in the neurofeedback-only condition. Importantly, the RCT by Romero-Muñoz et al. (2024) directly compared EEG-guided neurofeedback, bilateral high-frequency repetitive transcranial magnetic stimulation (rTMS) applied over the primary motor cortices, their combination, and no intervention. Improvements were observed only in the combined rTMS–neurofeedback condition, suggesting that EEG-guided neurofeedback may be effective only when preceded by rTMS-induced priming of cortical excitability. Notably, the authors reported effect sizes, provided a detailed characterization of the demographic and clinical features of the study population, and assessed patients' cognitive integrity, supporting the robustness of these findings and strengthening their interpretation.

Taken together, EEG-based neurofeedback studies indicate reliable neural self-regulation during training, but do not provide consistent evidence of meaningful improvement on validated clinical outcomes. Improvements reported on behavioral or task-based measures were limited and did not reliably generalize to clinical benefit when neurofeedback was applied in isolation.

3.3.3. *Outcomes of studies using DBS*

In contrast to EEG and fMRI approaches, LFP-DBS-based neurofeedback studies share two distinctive methodological features: they consistently employ within-subject designs and include small numbers of participants; consequently, none constitutes an RCT, limiting causal inference (Table 6). Furthermore, with the exception of Bichsel et al.

Table 4
Features and key findings of neurofeedback fMRI-based studies.

	NF-PD (n/F)	Age (years)	YSD	H&Y	UPDRS-III	MED ON/OFF	LEDD (mg/day)	Aim of brain activity modulation by NF	CTRL (n)	NF-sessions (duration)	RCT/QE	Clinical/behavioral tests	Outcome: clinical/behavioral tests	Outcome: brain activity
Subramanian et al. (2011)	5 (2F)	58 ± 13	3 ± 1	1	14.2 ± N/A	ON	380 ± 148	Increasing SMA activity via motor imagery	PD performing motor imagery (5)	4 (~6.5 min each) across 2 sessions; 2–6 months apart. All training: ~25–30 min.	RCT	UPDRS-III & finger tapping test	UPDRS-III and finger tapping speed improved in NF-PD with respect to baseline and CTRL	NF-PD learned to increase SMA activity
Subramanian et al. (2016)	13 (1F)	67 ± 9	4.2 ± 3	1.6 ± 0.6	23.3 ± 9.4 (in OFF)	ON	456 ± 219	Increasing SMA activity via motor imagery	PD performing motor exercise on a gaming console (13)	4 (~3 min each) across 3 sessions; 4 weeks apart. All training: ~36 min.	RCT	UPDRS-III & Daily Step Count, Cadence, Step Length, Gait speed	UPDRS-III improved in NF-PD with respect to baseline but no significant difference with CTRL. None of behavioral measure showed an improvement	NF-PD learned to increase SMA activity
Tinaz et al. (2018)	8 (4F)	66 ± 8	3.0 ± 2.5	2 ± 0.0	32.1 ± 6.6 (in OFF likely)	ON	364 ± 292	Increase functional connectivity between right insula and DMFC via motor imagery	N/A	10–12 (~4 min each) across two sessions; 2-weeks apart. All training: ~40–50 min	QE	UPDRS-III & movement imagery	No changes in UPDRS-III. No significant changes in motor imagery abilities	Functional connectivity increased in NF-PD
Tinaz et al. (2022)	22 (10F)	66 ± 8	4.9 ± 3.1	2 ± 0.2	32.3 ± 8.1 (in OFF likely)	ON	380 ± 148	Increase functional connectivity between right insula and DMFC via motor imagery	PD performing visual imagery (22)	10–12 (~4 min each) across two sessions; 2-weeks apart. All training: ~40–50 min	RCT	UPDRS-III & motor function test	No changes in UPDRS-III. Improvements in both groups in motor function score	No differences in functional connectivity

Notes. Demographic and clinical characteristics of the Parkinson's patients (PD) who underwent neurofeedback (NF). No PD patient exhibited cognitive impairment, as assessed using the Montreal Cognitive Assessment (Subramanian et al., 2016, Tinaz et al., 2022, Tinaz et al., 2018), or through clinical screening (Subramanian et al., 2011). Data are reported only for the NF group; no significant differences were found compared with the control group (where available). The column MED ON/OFF indicates whether PD patients performed the NF task while on their usual dopaminergic medication (ON) or after withdrawal (OFF). The column RCT/QE indicates whether the experiment was a randomized controlled trial (RCT) or a quasi-experimental study (QE). Motor symptom severity is reported using the total score of the Movement Disorder Society–Unified Parkinson's Disease Rating Scale, Part III (UPDRS-III) in parenthesis is reported whether it was assessed in MED ON or OFF. Abbreviations: CTRL = control; YSD = years since diagnosis; LEDD = Levodopa equivalent daily dose; DMFC = dorsomedial frontal cortex; SMA = supplementary motor area; F = number of females; n = number of patients included in the final analyses; min = minutes; N/A = not available.

Table 5
Features and key findings of neurofeedback EEG-based studies.

	NF-PD (n/F)	Age (years)	YSD	H&Y	UPDRS-III	MED ON/OFF	LEDD (mg/day)	Aim of brain activity modulation by NF	CTRL (n)	NF-sessions (duration) Total time	RCT/QE	Clinical/behavioral tests	Outcome: clinical/behavioral tests	Outcome: brain activity
Fumuro et al. (2013)	10 (8F) EXCL 3	63 ± 11	N/A	2.85 ± 1	N/A	ON	444 ± 197	Increase negative slow cortical potentials over the motor cortex to restore the early readiness potential	Healthy subjects (11)	2–4 (9–45 min each); 1–6 days apart. All training: ~40 to 180 min	QE	N/A	N/A	50% of CTRL and NF-PD were capable of learning SCP self-regulation
Azarpaikan et al. (2014)	8 (4F)	74.2 ± 3	8.5 ± 2	2.35 ± 0.1	N/A	ON	N/A	Increase low beta (12–15 Hz) and decrease theta (4–7 Hz) rhythm over the visual cortex to enhance static and dynamic balance	PD performing the same task (8)	8 (30 min); Spaced in 2.5 weeks. All training: ~4 h	RCT	Biodex Balance System (BBS) & Berg Balance Scale (BBC)	Both static (BBS) and dynamic balance (BBC) improved in NF-PD with respect to baseline and CTRL	All NF-PD learned to modulate low best and theta rhythms
Cooke et al. (2024)	16 (8F) EXCL 1	67 ± 10	5 ± N/A	N/A	29 ± 14.7 (in OFF)	OFF	N/A	Decrease average spectral power of μ rhythm of M1 before moving	N/A	3 (60 min each) in 3 days; 2 days apart. All training: 3 h	QE	UPDRS-III & precision handgrip task	No changes in UPDRS-III. Decrease in RT but not in accuracy	All NF-PD learned to self-regulate μ ERD
Romero-Muñoz et al. (2024)	11 (4F) NF only 10 (3F) rTMS & NF	62.2 ± 8 59 ± 7.6	5.9 ± 3.7 5.2 ± 3.2	1.9 ± 0.7 1.6 ± 0.7	15.5 ± 7.3 15 ± 6.4	OFF	537 ± 314 521 ± 292	Decrease average spectral power of α (9–12 Hz) & β (18–24 Hz) rhythms over the motor cortex	PD without treatments (9) PD with rTMS only (10)	8 (30 min); Spaced in 2 weeks. All training: ~4 h	RCT	UPDRS-III Finger tapping test (FT); Timed Up and Go (TUG); Limits of Stability (LOS)	Neurofeedback alone did not improve UPDRS-III, RT (FT); Functional mobility (TUG); or Postural stability (LOS).	All NF-PD learned to decrease α & β rhythms

Notes. Demographic and clinical characteristics of the Parkinson's patients (PD) who underwent neurofeedback (NF). Only Romero-Muñoz et al. (2024) assessed cognition, verifying through the Montreal Cognitive Assessment that no participants showed cognitive impairment. The remaining studies did not perform cognitive evaluations. Data (Mean ± SD) are reported only for the NF group; no significant differences were found compared with the Parkinson's disease (PD) control group (where available). The column MED ON/OFF indicates whether PD patients performed the NF task while on their usual dopaminergic medication (ON) or after withdrawal (OFF). The column RCT/QE indicates whether the experiment was a random controlled trial (RCT) or a quasi-experimental study (QE). None of the studies assessed cognitive impairment. Motor symptom severity is reported using the total score of the Movement Disorder Society–Unified Parkinson's Disease Rating Scale, Part III (UPDRS-III) in parenthesis is reported whether it was assessed in MED ON or OFF. Abbreviations: CTRL = control; YSD = years since diagnosis; LEDD = Levodopa equivalent daily dose; rTMS = repetitive Transcranial Magnetic Stimulation; SCP = slow cortical potentials; M1 = motor area; F = number of females; min = minutes; n = number of patients included in the final analyses; EXCL = excluded; N/A = not available.

Table 6
Features and key findings of neurofeedback LFP-DBS-based studies.

	NF-PD (n/F)	Age (years)	YSD	H&Y	UPDRS-III	MED ON/OFF	LEDD (mg/day)	Aim of brain activity modulation by NF	CTRL (n)	NF-sessions (duration) Total time	RCT/QE	Clinical/behavioral tests	Outcome: clinical/behavioral tests	Outcome: brain activity
Fukuma et al. (2018)	8 (5F)	63 ± 6.8	N/A	N/A	31.5 ± 22 (in ON)	ON	N/A	Train patients to change (increase or decrease) STN β-rhythm (13–30 Hz)	N/A	1 (~10 min each) in one day. All training: ~10 min	QE	EMG from forearm muscles to assess tremor-related activity	No changes in tremor-related activity	6 out of 8 NF-PD showed successful modulation of β-rhythm
He et al. (2020)	12 (4F)	62 ± 9	11 ± 5	N/A	45 ± 13.1 (in OFF)	OFF	N/A	Suppress STN β-bursts to reduce pathological β synchrony to improve motor performance and tremor	N/A	4 (~10–15 min each) spaced in 1–2 days. All training: ~40–60 min	QE	Task's RT and tremor, using triaxial accelerometer	RT decreased, but tremor worsened in tremorous patients	All NF-PD suppressed β bursts, reduced STN–cortex coupling, and increased STN γ–power
Bichsel et al. (2021)	10 (4F) 2 EXCL	59.9 ± 8	8.8 ± 5	2.1 ± 0.4	41.8 ± 10 (in OFF)	OFF	1277 ± 666	Train patients to change (increase or decrease) instantaneous β-power estimated over 200-ms windows	N/A	~30 (~1 min each) in one hour session. All training: ~30 min.	QE	Performance in a pronation–supination task measured with an inertial measurement unit.	Faster and more forceful forearm movements. Improvements persisted for 2 days	7 out of 8 NF-PD modulated β-activity. Modulation increased with training and persisted without feedback
Rouzitalab et al. (2023)	9 (4F)	59.8 ± 9	N/A	N/A	45.4 ± 7.5 (in OFF likely)	ON (likely)	Only % improvement	Train patients to change (increase or decrease) instantaneous β-power	N/A	1 (~30–40 min) in one day. All training: ~30–40 min	QE	N/A	N/A	All NF-PD modulated β-activity

Notes. Demographic and clinical characteristics of the Parkinson's patients (PD) who underwent neurofeedback (NF). No cognitive assessment was made in any of the papers. Data (Mean ± SD) are reported only for the NF group. The column MED ON/OFF indicates whether PD patients performed the NF task while on their usual dopaminergic medication (ON) or after withdrawal (OFF). The column RCT/QE indicates whether the experiment was a random controlled trial (RCT) or a quasi-experimental study (QE). None of the studies assessed cognitive impairment. Motor symptom severity is reported using the total score of the Movement Disorder Society–Unified Parkinson's Disease Rating Scale, Part III (UPDRS-III) in parenthesis is reported whether it was assessed in MED ON or OFF. Abbreviations: CTRL = control; YSD = years since diagnosis; LEDD = Levodopa equivalent daily dose; RT = reaction time; STN = subthalamic nucleus; F = number of females; min = minutes; n = number of patients included in the final analyses; EXCL = excluded; N/A = not available.

(2021), the remaining studies (Fukuma et al., 2018, He et al., 2020, Rouzitalab et al., 2023) did not report crucial demographic and clinical characteristics of the samples, substantially limiting replicability. In addition, cognitive integrity was not reported in the original publications included in this review. Collectively, these features pose significant limitations for interpreting the findings. As in fMRI- and EEG-based neurofeedback studies, PD patients were generally able to learn to modulate STN activity. However, assessment of clinical outcomes was limited. Rouzitalab et al. (2023) did not include any clinical or behavioral outcome measures. Fukuma et al. (2018) assessed tremor-related activity and reported no improvement following training. He et al. (2020) reported faster reaction times in a game-like–motor task, but also observed worsening of tremor severity in patients with pre-existing tremor. Only Bichsel et al. (2021) demonstrated faster and more forceful forearm movements at the group level, with motor performance improvements persisting when reassessed two days later. Importantly, in studies reporting task-based improvements, validated clinical endpoints were not assessed. Moreover, the absence of reported effect sizes, together with the small within-subject sample and the lack of control conditions in several studies, limits the interpretation of the magnitude and robustness of these findings.

In summary, LFP-DBS-based neurofeedback studies demonstrate that patients with PD can reliably learn to modulate neural activity. However, evidence for clinically meaningful improvement on validated clinical outcomes is limited, and reported behavioral effects cannot be assumed to reflect clinical benefit.

4. Discussion

The findings of this systematic review, despite being based on a limited body of literature, are nevertheless compelling. With one exception (Tinaz et al., 2022), all included studies indicate that patients with PD can learn to voluntarily modulate neural activity previously associated with disease severity across different neurofeedback modalities. However, the translation of such neural self-regulation into clinically meaningful or behavioral improvements remains limited and inconsistent. Below, we discuss these findings in detail.

4.1. Clinical and behavioral outcomes of neurofeedback interventions

The central issue in neurofeedback interventions in PD is whether training-induced neural modulation translates into durable, clinically meaningful symptom improvement. In this context, it is important to distinguish between clinically validated measures of symptom severity (e.g., UPDRS III, Berg Balance Scale) and behavioral task performance metrics assessed during or around training, which do not necessarily reflect clinically meaningful improvement. Accordingly, the critical question is whether learning to control specific neural substrates has been shown to improve clinically assessed features of PD symptomatology, or whether effects are limited to changes in task-specific behavioral performance. Notably, only 10 of the 12 selected studies included in this review assessed clinical outcomes following neurofeedback training; Rouzitalab et al. (2023) and Fumuro et al. (2013) did not. Moreover, among studies that included clinical assessments, outcomes were limited to motor symptoms, with no evaluation of non-motor symptoms, despite their well-established relevance to patients' quality of life (Chaudhuri and Schapira, 2009). Consequently, a core dimension of PD symptomatology has been systematically overlooked in the neurofeedback literature. Six of the ten studies assessing clinical outcomes employed the UPDRS III, the gold-standard clinician-rated measure of global motor symptom severity in PD (Cooke et al., 2024, Romero-Muñoz et al., 2024, Subramanian et al., 2011, Subramanian et al., 2016, Tinaz et al., 2022, Tinaz et al., 2018). Four of these studies reported no significant post-training improvement in UPDRS III scores following neurofeedback (Cooke et al., 2024, Romero-Muñoz et al., 2024, Tinaz et al., 2022, Tinaz et al., 2018). By contrast, Subramanian et al. (2011, 2016) reported improvements in UPDRS III scores. However, the former study included only five participants and did not report effect sizes, limiting statistical interpretability (Subramanian et al., 2011). The latter study, while including a slightly larger sample ($n = 13$), demonstrated a statistically significant but modest improvement in the neurofeedback group that did not differ significantly from changes observed in the control group (Subramanian et al., 2016). Again, the absence of effect size reporting constrains the interpretation of the clinical relevance of these findings.

Because the UPDRS Part III may not fully capture changes in balance and complex motor performance, all of the above-cited studies included additional motor task measures (see Tables 4, 5, and 6). These measures reflect performance in structured motor tasks rather than global clinical severity. Across these assessments, three studies reported no significant changes following neurofeedback training (Romero-Muñoz et al., 2024, Subramanian et al., 2016, Tinaz et al., 2018), and one study observed motor improvements that did not differ from those seen in the control group (Tinaz et al., 2022). Subramanian et al. (2011) reported faster finger tapping following neurofeedback; however, as discussed above, the interpretability of this finding is limited by the small sample size and lack of effect size reporting. Cooke et al. (2024) reported a significant reduction in reaction time during a precision handgrip task, without concomitant improvement in accuracy. Importantly, the absence of a control group in this study precludes disentangling neurofeedback-related effects from potential task familiarization or practice effects.

The four remaining studies did not assess UPDRS Part III and evaluated outcomes exclusively using motor or balance performance measures. Two focused on tremor, reporting either no change in tremor-related activity (Fukuma et al., 2018) or worsening of tremor in patients with pre-existing tremor (He et al., 2020). Azarpaikan et al. (2014) reported improvement on a validated functional balance scale (Berg Balance Scale), alongside changes in instrumented balance performance (Biodex Limits of Stability test), whereas Bichsel et al. (2021) reported improved performance in a pronation–supination task. However, these findings were derived from small samples and lacked effect-size reporting, substantially limiting their interpretability.

Overall, although patients across all ten studies generally learned to modulate the targeted neural substrates, five studies failed to demonstrate an effect on validated clinical outcome measures attributable to neurofeedback training (Cooke et al., 2024, Romero-Muñoz et al., 2024, Subramanian et al., 2016, Tinaz et al., 2022, Tinaz et al., 2018). In addition, Fukuma et al. (2018) evaluated tremor-related activity using task-based measures and reported no significant change following neurofeedback. One study reported worsening of tremor in a subset of patients, indicating that clinical effects were not uniformly null but included symptom-specific adverse outcomes (He et al., 2020).

Three studies reported positive findings: Azarpaikan et al. (2014) observed improvement on a clinically validated balance scale (Berg Balance Scale) alongside changes in instrumented balance performance, whereas Bichsel et al. (2021) and Subramanian et al. (2011) reported improvements in task-specific motor performance. However, the very small sample sizes and absence of effect size reporting in these studies markedly constrain interpretation, as statistical significance cannot be reliably evaluated, and it remains unclear whether improvements in task-specific performance translate into clinically meaningful symptom reduction.

In addition, among these three studies, only Bichsel et al. (2021) examined persistence beyond the training session, reporting maintenance of motor task performance for two days in seven of eight patients. However, the small sample size and short follow-up interval, substantially limit the ability to draw conclusions regarding durability and clinical relevance.

More recently, additional work has attempted to address some of these methodological limitations. A study by Salzmann et al. (2025), published after the inclusion date of the current review, provides an interesting methodological advance by attempting to bridge neural,

behavioral, and clinical levels of analysis. The authors combined LFP-DBS-based neurofeedback targeting STN beta oscillations with objective movement quality metrics derived from wearable sensors during motor tasks aligned with two MDS-UPDRS III items (foot stomping and hand pronation–supination). This approach enabled the detection of subtle improvements in lower-limb motor performance following beta-power downregulation. However, several limitations should be noted. First, upper-limb motor performance was unaffected by neurofeedback. Second, successful beta-power downregulation was achieved in only eight of the ten participants, highlighting substantial inter-individual variability in responsiveness to neurofeedback. Third, beta-power reduction was not statistically significant during the motor task blocks used for the pre–post behavioral comparison, leaving it uncertain whether the observed behavioral improvements can be directly attributed to the neurofeedback intervention. Finally, clinician-rated MDS-UPDRS III scores were not assessed before and after training, further limiting conclusions regarding clinical efficacy.

Of relevance, the two most rigorously controlled studies, which also included the largest participant samples, failed to demonstrate effects specifically attributable to neurofeedback (Romero-Muñoz et al., 2024, Tinaz et al., 2022). Romero-Muñoz et al. (2024) included three comparison arms (rTMS alone, rTMS plus neurofeedback, and no intervention) and reported the effect sizes, showing that neurofeedback alone did not confer clinical benefits. Notably, Tinaz et al. (2022) compared neurofeedback-guided kinesthetic imagery with a non-motor visual imagery control condition deliberately designed to avoid imagining one's own movements. The authors reported that both interventions equally improved motor functions and training-related whole-brain task-based functional connectivity. Changes in functional connectivity were distributed across visual and motor networks in the kinesthetic and non-motor visual imagery groups, respectively, indicating imagery practice–related network reorganization rather than effects of the neurofeedback training. Furthermore, post hoc analyses (Cherry et al., 2023) showed that whole-brain task-based functional connectivity during visual imagery covaried with imagery quality at the level of specific pairwise connectivity changes, particularly increased coupling between visual and sensorimotor cortical regions. These findings suggest that, within this experimental context, imagery practice may represent a more potent driver of network-level plasticity than neurofeedback itself.

Among the minority of studies reporting positive effects, methodological limitations, including very small sample sizes, lack of effect size reporting, and, in some cases, inadequate control conditions, preclude firm conclusions regarding neurofeedback-specific efficacy. Importantly, the most rigorously controlled and adequately powered studies have consistently failed to identify effects attributable to neurofeedback beyond those associated with imagery practice or other nonspecific training components. Moreover, evidence for the durability of any clinical effects is largely absent. Thus, the preponderance of current evidence suggests that neurofeedback training does not confer clinically meaningful benefits for PD symptoms when evaluated under adequately controlled experimental conditions, and claims of efficacy remain unsupported by robust or durable outcome data.

4.2. Limitations of the current literature

The current literature on neurofeedback interventions has several methodological limitations. Sample sizes are often very small, with an average ($\pm$ SD) of approximately 10 $\pm$ 4 participants undergoing neurofeedback training. Importantly, sample sizes are rarely justified by a priori power analyses, with only two notable exceptions (Cooke et al., 2024, Tinaz et al., 2022). This is problematic because underpowered studies have a low probability of detecting true effects and substantially increase the risk of false-negative and false-positive findings, thereby limiting the reliability and generalizability of reported outcomes.

These limitations are further compounded by incomplete reporting of effect sizes: seven studies did not report effect size measures at all (Azarpaikan et al., 2014, Bichsel et al., 2021, Fukuma et al., 2018, Fumuro et al., 2013, He et al., 2020, Rouzitalab et al., 2023, Subramanian et al., 2011), three reported them only partially, omitting key comparisons (Subramanian et al., 2016, Tinaz et al., 2022, Tinaz et al., 2018), and only two studies reported effect sizes comprehensively (Cooke et al., 2024, Romero-Muñoz et al., 2024). The absence of systematic effect size reporting is a major methodological shortcoming, as it precludes assessment of the magnitude, and thus the practical relevance, of effects that are otherwise described solely by p-values (Lakens, 2013). Moreover, variability in the types of outcome measures employed, ranging from validated clinical scales to task-specific behavioral performance metrics, further complicates interpretation, as improvements in structured motor tasks do not necessarily reflect clinically meaningful reductions in overall PD symptom severity.

Furthermore, in a non-negligible number of studies, key demographic and clinical characteristics of participants, as well as essential statistical parameters, were not reported. Incomplete reporting of participant demographics, clinical characteristics, and statistical details undermines study replicability and limits cumulative knowledge building, a concern widely recognized as a major contributor to the reproducibility crisis in neuroscience and biomedical research (Button et al., 2013, Korbmacher et al., 2023, Munafò et al., 2017).

4.3. Challenges and future directions

Given the above-described state of art, a key requirement for future studies is adherence to the CONSORT guidelines for randomized trials (Moher et al., 2010) and the TREND guidelines for non-randomized intervention studies (Des Jarlais et al., 2004), as appropriate to the study design, to ensure transparency, completeness, and interpretability of quantitative data reporting. In addition, it is highly advisable that future research also report Bayes factors (BF_{10}). The use of BF_{10} makes it possible to distinguish between data that genuinely support the null hypothesis and data that are merely uninformative. This approach is increasingly adopted to complement frequentist inference and to address some of its interpretative limitations, particularly in small-sample studies (Wagenmakers et al., 2018).

Beyond rigorous reporting practices, future investigations must also address several substantive methodological challenges. Chief among these is the need to adequately control for placebo effects when evaluating the therapeutic efficacy of neurofeedback interventions in PD. In neurofeedback paradigms, placebo responses are likely to be amplified by a range of contextual factors, including repeated interactions with clinicians, the use of sophisticated and costly technological equipment, extended multi-session training protocols, and the requirement for sustained attention during training (Thibault et al., 2016). Therefore, the absence of a control group poses a major limitation to data interpretability. From a strict methodological standpoint, the most direct way to isolate neurofeedback-specific effects is through sham-controlled designs, in which non-contingent feedback is provided to the control group. Among the studies included in this review, two implemented such approaches, using either non-contingent EEG signals (Azarpaikan et al., 2014) or a BOLD signal unrelated to participants' own brain activity (Subramanian et al., 2011). However, sham neurofeedback has increasingly been recognized to raise ethical concerns related to participant deception, frustration, and potential symptom exacerbation (Sitaram et al., 2017). As a result, more recent studies have adopted alternative control strategies, including: (a) active control designs, such as visual imagery (Tinaz et al., 2022), or motor tasks (Subramanian et al., 2016); (b) no-intervention designs (Romero-Muñoz et al., 2024), (c) unidirectional designs, requiring participants to volitionally modulate a neural signal in a single direction (Fumuro et al., 2013); (d) bidirectional designs, requiring participants to volitionally modulate the same neural signal in opposite directions under identical feedback conditions (Bichsel et al., 2021, Fukuma et al., 2018, He et al., 2020, Rouzitalab et al., 2023). Among these approaches, within-subject

bidirectional designs offer the strongest control over placebo effects, as they hold expectancy, motivation, and contextual factors constant across conditions. This design was employed by a subset of studies (Bichsel et al., 2021, He et al., 2020, Rouzitalab et al., 2023), in which the same participants received veridical feedback while learning to modulate the same neural signal in opposite directions. Accordingly, this approach currently represents the most methodologically robust and ethically acceptable strategy for isolating neurofeedback-specific effects in clinical populations.

Another critical issue concerns the limited understanding of the mechanisms underlying volitional self-regulation in PD. Among the studies included in this review, four explicitly relied on visual motor (Subramanian et al., 2011, Subramanian et al., 2016) or kinesthetic imagery (Tinaz et al., 2022, Tinaz et al., 2018) as the primary strategy for neural modulation, whereas eight employed reinforcement-based neurofeedback without prescribing imagery strategies (Azarpaikan et al., 2014, Bichsel et al., 2021, Cooke et al., 2024, Fukuma et al., 2018, Fumuro et al., 2013, He et al., 2020, Romero-Muñoz et al., 2024, Rouzitalab et al., 2023). Given that imagery–based neurofeedback depends on explicit, goal-directed cognitive strategies to modulate neural activity, whereas reinforcement-based neurofeedback relies on implicit, trial-and-error learning driven by feedback signals, these methodologies likely engage distinct neural and learning mechanisms. It is currently unknown whether they are equally effective in enabling the acquisition of volitional control over brain activity in PD.

Likewise, it remains unclear whether neurofeedback exposure must exceed a minimum duration or intensity threshold to establish stable learning effects. Across the reviewed studies, neurofeedback training duration varied widely, ranging from as little as 10 min to approximately 240 min of total exposure. Future research should address this issue systematically, as insufficient or inconsistent training dosage represents a plausible contributor to the lack of reliable neurofeedback efficacy.

This consideration leads to a final, fundamental unresolved question: whether neurofeedback-induced neural changes in PD are retained relative to control conditions, generalize beyond the training context, and ultimately support durable, clinically meaningful behavioral improvements. Results across all studies reviewed provide only very limited evidence on this issue. All in all, future investigations should focus on this knowledge gap, as the persistence of benefits is a core feature of therapeutic approaches.

5. Conclusions

While patients with PD can learn to volitionally self-modulate activity in several regions implicated in PD pathophysiology (including the SMA, motor cortices, and STN) across different neurofeedback modalities (BOLD signal, α, β, or μ rhythms) and recording techniques (EEG, LFP-DBS, fMRI), the current evidence does not demonstrate consistent therapeutic benefits. Although caution is warranted when drawing firm conclusions, given the relatively small number of PD neurofeedback studies and their frequent methodological limitations, the present review suggests that neurofeedback, when used as a standalone intervention, does not reliably improve PD symptoms, and evidence for durable clinically meaningful benefit remains lacking.

CRediT authorship contribution statement

Giovanni Mirabella: Supervision, Conceptualization, Data curation, Writing – original draft. **Alberto Borboni:** Supervision, Writing – review & editing. **Michele Guerreschi:** Data curation, Writing – review & editing. **Antonio Suppa:** Writing – review & editing. **Cinzia Amici:** Supervision, Data curation, Writing – review & editing.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

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