1. Introduction

Parkinson’s disease (PD) is one of the most common neurodegenerative conditions, affecting about 1% of individuals over 60 years of age, with standardized incidence rates of 8–18 cases per 100,000 person-years [1,2]. This condition is characterized by the progressive loss of dopaminergic neurons within the substantia nigra pars compacta in the brain, causing a wide range of both motor and non-motor symptoms [3–5]. The motor symptoms constitute the distinctive symptomatic ensemble that forms the basis of the clinical diagnosis of the disease, including tremors, rigidity, bradykinesia, and postural instability [6]. Regarding non-motor symptoms, cognitive deficits affect approximately 30–40% of patients and represent the most debilitating complication of PD [7,8]. These deficits arise from the reduced availability of dopamine in the brain, particularly in the frontal domains and the attentional system, giving rise to a fronto-striatal syndrome [9]. Cognitive deficits can manifest from the early stages of the disease and are primarily characterized by involvement of executive functions, visuospatial abilities, and memory [10,11]. The cognitive dysfunctions of PD encompass various nuances, ranging from mild cognitive impairment in PD (PDMCI) to Parkinson’s disease dementia (PDD) [12,13].
erally considered a safe rehabilitative strategy that offers several advantages, including painless application, few side effects, cost-effectiveness, and the potential for at-home administration with remote expert supervision [32–34]. As a result, tDCS has been adopted with significant success, demonstrating promising results in addressing brain degenerative processes, encompassing both physiological processes related to aging and pathological processes, covering a wide range of neurological disorders [35–38] and neuropsychiatric conditions [39–42]. The application of tDCS has raised new prospects for the treatment of mild cognitive impairments and dementia [43–46]. tDCS appears to be particularly effective in cases of mild cognitive impairment, showing more promising results compared with its application in Alzheimer’s dementia [47]. Therefore, the use of tDCS should be considered during the phase of PD-MCI, when effectiveness seems particularly likely [48]. Considering all this, there is a growing need for randomized placebo-controlled trials that assess both the efficacy and specific neural mechanisms of the exclusive use of tDCS in individuals affected by PD-MCI. Despite the established evidence for tDCS, conflicting results have emerged from studies examining the effects of tDCS in combination with other treatments [49–51]. These studies have reported improvements in various cognitive domains, but whether these improvements can be solely attributed to tDCS or whether there has been adjunct effect—an enhancement of the effects of additional therapies—remains ambiguous [52–55]. This point is of considerable significance and requires further investigation.

1.2 Evaluating Cognitive Functions Through Electroencephalography Parameters in PD

Cognitive deficits in PD encompass various aspects, including executive functions, visuospatial abilities, and memory [10,11]. Event-related potentials (ERPs) represent an objective quantifier of cognitive functions, providing an opportunity to monitor cognitive changes without the apparent influence of motor deficits in PD [56]. ERPs are a valuable tool in cognitive neuroscience that allow researchers to measure and analyze brain activity in response to specific stimuli or events. ERPs are obtained through electroencephalography (EEG), a non-invasive technique that records the electrical activity of the brain over time. EEG involves placing electrodes on the scalp, which detect and record electrical signals generated by neural activity. ERPs are time-locked to a specific event, such as the presentation of a stimulus, and provide a way to assess neural processing associated with sensory, motor, or cognitive functions. In our study, the P300 ERP is of particular importance [57]. The P300 component is a positive deflection in the ERP waveform that typically occurs around 300 ms after the presentation of a rare or unexpected stimulus. P300 is associated with cognitive processes related to attention, memory, and decision-making. The onset latency of the P300 component, which is the time it takes for the P300 response to appear after a stimulus, is an essential measure as it reflects the speed and efficiency of cognitive processing. In addition to ERPs, the present study investigated the role of alpha and beta band rhythms in PD. These neural oscillations, measured through EEG, are critical in understanding the motor and cognitive aspects of PD. The alpha band is associated with the inhibition of sensory information processing. When the brain is at rest or not engaged in a specific cognitive task, alpha rhythms are dominant. They are typically recorded over posterior brain regions and are believed to be involved in inhibiting irrelevant sensory information, thereby promoting focused attention and cognitive stability. Beta rhythms are associated with motor control and motor planning. They are often recorded over motor-related areas of the brain, such as the sensorimotor cortex. These rhythms are implicated in the coordination of muscle movements, and their modulation is linked to changes in motor function [58]. In PD, aberrations in alpha and beta band rhythms, coupled with alterations in P300 oscillatory dynamics, may collectively serve as critical neurophysiological markers. These markers potentially underlie the intricate interplay observed between motor dysfunction and cognitive impairment in individuals with PD [59]. The rationale for their utilization lies in the well-established involvement of alpha and beta band rhythms in motor control, as evidenced by Hammond et al. [59], which is closely intertwined with the cognitive deficits witnessed in PD. Concurrently, abnormalities in P300 oscillations have been linked to cognitive decline in PD [60]. Consequently, we posit that investigating the convergence of alpha and beta band rhythms with P300 oscillations in PD could illuminate the mechanistic underpinnings of both the motor and cognitive manifestations of the disease.

1.3 Study Significance and Hypotheses

Although a body of evidence suggests that ERPs could be a sensitive method to investigate mechanisms of symptomatic improvement due to tDCS [61], and while some studies have investigated the neurophysiological effects of tDCS in samples with mild cognitive decline [62,63], few studies have investigated the effects of tDCS on ERPs in PD [64–68]. The primary objective of this research was to assess the effects of tDCS on the left dorsolateral prefrontal cortex (DLPFC) in patients with PD concerning neurophysiological functions measured through EEG. Our hypothesis posited that tDCS on the left DLPFC would lead to a reduction in the onset latency of the P300 event-related potential, accompanied by an increase in alpha and beta band rhythms within the neural oscillatory activity. This hypothesis is grounded in prior research findings that have demonstrated the modulatory effects of tDCS on cortical excitability and neural oscillations [69–71]. Specifically, studies have shown that tDCS can influence neuronal membrane potentials and enhance cortical synchronization, which are
associated with alterations in event-related potentials and alpha and beta band rhythms. Furthermore, the left DLPFC is a region known to be involved in the cognitive processes of interest, including those related to P300 latency [72,73]. Preliminary studies and research on deep brain stimulation (DBS) in PD have suggested that modulating the left DLPFC could lead to improvements in both motor and cognitive symptoms [74]. These prior findings have justified further investigation into DLPFC stimulation in the context of PD. In fact, the left DLPFC is implicated in high-level decision-making [75]. Cognitive deficits are common in PD [76]. Stimulating the left DLPFC may potentially enhance these compromised cognitive functions. Stimulation of the left DLPFC may have a dual effect, improving both motor and cognitive symptoms because this brain region is involved in both types of functions [14,77]. DLPFC stimulation can influence brain circuits, including those connecting the DLPFC to other brain regions involved in PD symptoms. This modulation may help regulate brain activity and enhance function in affected areas. Furthermore, in this study, we hypothesized that (a) patients undergoing tDCS would exhibit a significant reduction in the onset latency of the P300 event-related potential compared with the group undergoing sham tDCS and (b) patients undergoing tDCS would demonstrate a significant increase in the power spectrum of alpha and beta band rhythms compared with the group undergoing sham tDCS.

### 2. Materials and Methods

#### 2.1 Experimental Design

The study employed an ABA experimental design, consisting of a pre-test, an intervention, and a post-test or both groups. It was double-blinded, on the part of both the researcher and the evaluator. A matching procedure was implemented for the assignment. The tDCS group was initially recruited and, subsequently, the sham group was matched to them based on key demographic and clinical variables, ensuring baseline comparability between the two groups. This approach was chosen to minimize potential confounding factors and enhance the internal validity of our study.

#### 2.2 Participants

Based on the sample size commonly reported in the literature and the estimated sample dimension [78,79], 30 patients diagnosed with PD were recruited from the Madonna della Consolazione Polyclinic Nursing Home in Reggio Calabria, a region in Southern Italy. All participants were aged between 67 and 82 years (mean age [M] = 74.5 years, standard deviation [SD] = 7.2 years). Among these patients, a group of 15 individuals received anodal tDCS, while the other group of 15 age- and sex-matched subjects received simulated tDCS. Inclusion criteria required a diagnosis of PD based on the guidelines of the United Kingdom Parkinson’s Disease Society (UKPDS) and the clinical diagnostic criteria of the Brain Bank [80,81]. Additionally, the diagnosis of PD-MCI aligned with Level 2 (comprehensive) criteria for PD-MCI [82], which involved a performance that was 1.5 SDs below normative data in at least two tests within a single domain or in one or two tests across distinct domains [83]. Selected participants exhibited scores ranging from 22 to 26 points on the Mini-Mental State Examination (MMSE), which was adjusted for the educational level of this population [84,85]. The clinical data related to the Unified Parkinson’s Disease Rating Scale (UPDRS) [86] of patients who underwent anodal tDCS and sham procedure were extracted from medical records. Furthermore, a detailed clinical assessment of the patients was conducted, and they presented motor symptoms ranging from stage 1 to III on the Hoehn and Yahr (H&Y) scale [87]. Right-hand laterality was assessed using the Edinburgh Handedness Inventory (EHI). All patients had been maintained on stable and optimal dopaminergic medication therapy for at least one month prior to the start of the study and were required to maintain this regimen throughout the study’s duration.

Exclusion criteria included a diagnosis or evidence of secondary or atypical parkinsonism, previous clinically significant neurological disorders, prior neurosurgical interventions, head trauma, brain injuries, epilepsy, stroke, or multiple sclerosis. Additionally, patients with a diagnosis of major depressive disorder, psychotic disorders, bipolar disorder, or alcohol and substance use disorders were excluded.

#### 2.3 Procedure

This study was conducted in accordance with the Declaration of Helsinki and received approval from the Ethics Committee of Madonna della Consolazione Polyclinic Nursing Home (protocol approval number 2021-198). Clinically documented data, already available at the healthcare facility and provided by healthcare professionals within a timeframe not exceeding one month (MMSE, UPDRS, DD [Duration of Disease], LEV [Levodopa Equivalent Dose], H&Y score), were utilized for the present study. Before participating, each participant received detailed information about the study’s purpose and data collection procedures. This information was presented clearly and comprehensively, enabling participants to make an informed decision about their involvement. Subsequently, each participant provided written informed consent. Prior to the intervention, participants were thoroughly briefed on the stimulation procedure employed in the study. This explanation included details about the functioning of tDCS, as well as the associated practical procedures. Additionally, a clear illustration of potential side effects related to the intervention was provided. Each participant was informed of their right to withdraw from the study at any time without incurring...
any negative consequences. At the start of the stimulation, participants were asked about their tolerance to the 2 mA or if they experienced any discomfort. Subjects tolerated the treatment well, and no adverse effects associated with tDCS were observed.

2.4 Measurements

In this research, the electroencephalogram was employed as the measuring instrument to explore neurophysiological parameters. More precisely, the analysis focused on the P300 component through an auditory oddball paradigm, while also examining the rhythms within the alpha and beta bands from the DLPFC.

2.4.1 EEG Recording

EEG recordings were captured at a sampling rate of 500 Hz, employing a band-pass filter set between 0.1 Hz and 70 Hz. SCAN software (version 4.3, Neuroscan, Compumedics, El Paso, TX, USA) along with NuAMP amplifiers were used for the recording. Eighteen scalp electrodes (Ag/AgCl) were positioned according to the standard 10/20 system outlined by Jasper [88]. The reference electrode was placed in the midline derivations, such as Fz, FCz, Cz, and Pz, and the ground electrode between Cz and Pz. All positions were referenced to a common Cz reference and later re-referenced offline to average mastoids. Electrode impedances were maintained at or below 10 kΩ. EEG data intended for ERP analysis underwent offline processing. This involved the application of a 20 Hz low-pass filter, baseline correction, and division of waveforms into epochs centered around stimulus presentation. Trials deviating in amplitude beyond ±100 µV were excluded. A minimum of 20 trials for each stimulus were deemed necessary for the inclusion of individual average ERP waveforms. Epochs spanning from 200 ms to 1000 ms were generated offline, centered around low and high tones along with novel noises. The key components of interest, namely P1, N2, and P3a, were automatically detected within specific time intervals (70–110 ms, 210–270 ms, and 270–370 ms, respectively) from midline positions (Fz, FCz, Cz, and Pz) where these peaks exhibit maximum activity [89]. Frequent stimuli immediately preceding each infrequent stimulus were selected for averaging, ensuring comparable signal-to-noise ratios.

2.4.2 Auditory Oddball Paradigm

This study utilized an auditory oddball task, where participants were exposed to auditory stimuli without needing to provide overt responses. The stimuli were presented using specialized presentation software from Neurobehavioral Systems Inc. (Berkeley, CA, USA). Each participant was seated in a partially illuminated and soundproofed room, positioned in front of a computer monitor situated approximately 70 cm away. Adjacent to the monitor, two speakers were set up for audio presentation. The auditory paradigm encompassed three distinct sound categories: frequent pure sinusoidal tones, infrequent pure sinusoidal tones, and novel sounds. Within this framework, 10% of the stimuli consisted of infrequent tones (2 kHz, 200 ms duration, 5 ms rise and fall time, 70 dB sound pressure level [SPL]). Another 10% constituted novel noises, while the remaining 80% were frequent tones (1.5 kHz, 200 ms duration, 5 ms rise and fall time, 70 dB SPL). The duration of each tone or noise was 200 ms, with a stimulus onset asynchrony of 700 ms. The presentation consisted of two blocks, each containing 700 stimuli (560 frequent, 70 infrequent, and 70 novel). The intensity of novel sounds was digitally adjusted to ensure they did not surpass 70 dB SPL, as measured by a Bruel and Kjaer sound pressure meter. Fourteen distinct novel stimuli were employed, each repeated a maximum of five times during the experiment. Overall, the task completion time amounted to approximately 20 minutes.

2.4.3 Alpha and Beta Band Rhythms

Quantitative analysis of EEG data was conducted using customized algorithms developed in MATLAB code (The MathWorks Inc., Natick, MA, USA) [90]. The assessment of power spectral density (PSD) involved converting the signal from the time domain to the frequency domain through the utilization of the Welch method [91]. PSD values were determined for individual epochs, with subsequent computation of their averages. Initial calculations encompassed the absolute power of the entire signal and the absolute power within designated frequency bands for each electrode. PSD was calculated for 5-s windows after rejecting an initial 50 s out of total duration of 200 s for which the rhythmic tones were played. This study specifically focused on the alpha band (8–13 Hz) and beta band (14–29 Hz) from the DLPFC.

2.4.4 UPDRS

The UPDRS was used to integrate elements from existing scales and provide a comprehensive tool for capturing and assessing various aspects of PD, including motor disability, movement impairment, cognitive issues, emotional aspects, and treatment-related complications [87]. The UPDRS is divided into four parts. Part I focuses on non-motor symptoms, such as dementia, depression, and psychosis. Part II assesses the patient’s ability to perform daily activities like dressing, grooming, and using utensils. Part III is evaluated by a physician and measures motor symptoms like speech, facial expression, tremors, muscle tone, speed of hand and leg movements, walking, and balance. Part IV assesses treatment complications. The response scale is five-point, with choices ranging from 0 to 4.

2.5 Intervention

The tDCS device utilized in this study was the BRAINDEE stimulator, manufactured by Omicron-t S.r.l., based in Naples, Italy. Stimulation was conducted using a
pair of sponge electrodes, each with a diameter of 25 mm, previously saturated with a saline solution. A constant current was delivered by a battery-powered stimulator. The decision was made to target the left DLPFC due to its involvement in high-level cognitive functions such as attention, working memory, and decision-making. These cognitive functions are frequently impaired in patients with PD. By modulating the DLPFC, this stimulation may contribute to improvements in both motor and cognitive symptoms. Given the DLPFC’s dual role in motor and cognitive functions, stimulating this region could potentially have a dual effect, enhancing compromised cognitive functions as well [92,93]. To activate the left DLPFC, the anode was positioned over the F7 region, while the cathodic reference electrode was located above the right supraorbital area. The precise electrode placement was determined following the EEG 10–20 system. Stimulation intensity was set at 2 mA (with a current density of 2.5 mA/cm²) for 20 minutes, administered five times a week for two consecutive weeks, both in the study group and the placebo group. The decision to use 2 mA was based on the safety and comfort of our participants. We opted for the 2 mA intensity as it is considered safe and well-tolerated, which is particularly crucial when dealing with individuals who may have various health conditions or sensitivities. Safety is paramount in human research, and a 2 mA intensity level is widely accepted [79,94].

In the simulated sham condition, the electrodes were placed in the same manner as in actual stimulation, but the stimulation automatically ceased after 10 s from the beginning of the session. This time interval is insufficient to induce a significant stimulation effect on the brain. A stimulation ramp was incorporated to ensure that participants would experience the typical tingling sensations near the electrodes, creating the illusion of receiving stimulation even during the simulated sham condition when no actual stimulation was applied. This procedure was implemented to maintain participants’ unawareness of whether they were receiving real stimulation or the sham condition.

### 2.6 Statistical Analysis

All statistical analyses were conducted using SPSS 24.0 software (SPSS Inc., Chicago, IL, USA). The main objective of the analyses was to explore potential differences between the experimental group (i.e., anodal stimulation) and the sham group (i.e., sham stimulation) in neurophysiological parameters such as P300 latency and the alpha and beta band rhythms. To verify the causal relationship between UPDRS as a predictive variable and the P300 latency and the alpha and beta band rhythms, separate linear regressions were conducted. Repeated-measures (RM) analysis of variances (ANOVARs) were performed, employing the experimental groups as a between-subjects factor, and the phases—pre- and post-tests—as within-subjects factors. Furthermore, if the RM ANOVAs revealed significance, a post hoc analysis was conducted to identify which group exhibited changes between the pre- and post-test phases. Post hoc pairwise comparisons were executed using the Student’s paired *t*-test. To address the issue of multiple comparisons, significance was attributed solely to the level *p* < 0.005. Regarding effect size, we employed Cohen’s d for *t*-test comparisons,Eta-squared (η²) for ANOVA, and R-squared (R²) for regression analysis.

### 3. Results

Table 1 summarizes the demographic characteristics of the participants.

Table 2 shows descriptive statistics of the alpha and beta band rhythms and P300 latency. To verify the relationship between the scores pertaining to the UPDRS scores and the P300 latency, the alpha and beta band rhythms, linear regression analyses were conducted. UPDRS scores did not show statistical significance as predictors of the differences between the pre-test and post-test phases in P300 latency (−0.02 ± 0.03, β ± standard error [SE], *p* = 0.06), nor did they significantly predict the differences between the pre-test and post-test phases in the alpha band rhythm (−0.04 ± 0.05, β ± SE, *p* = 0.05) or the beta band rhythm (0.04 ± 0.03, β ± SE, *p* = 0.08).

Fig. 1 shows the pre- and post-test alpha band rhythms for both groups. RM-ANOVA with one between-subject factor group and one within-subject factor phase was conducted. RM-ANOVA showed again that the effect of group was not significant (F (1, 28) = 1.99; *p* = 0.22, η² = 0.08). The phase factor also shows no significant differences (F (1, 28) = 1.34, *p* = 0.34), but the effect of the group x phase interaction was significant (F (1, 28) = 16.51; *p* < 0.01, η² = 0.09). Paired *t*-tests show statistical differences in the alpha band only for PD patients with anodal tDCS. Alpha bands had higher values in the post-test than in the pre-test phase.

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Table 1. Demographic data of participants.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Experimental Group</th>
<th>Sham Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>73.27 (±5.64)</td>
<td>73.73 (±5.20)</td>
</tr>
<tr>
<td>Sex (ratio)</td>
<td>F:M = 2:3</td>
<td>F:M = 2:3</td>
</tr>
<tr>
<td>Education (years)</td>
<td>6.87 (±1.85)</td>
<td>7.13 (±1.64)</td>
</tr>
<tr>
<td>MMSE core</td>
<td>22.87 (±1.85)</td>
<td>23.73 (±1.62)</td>
</tr>
<tr>
<td>UPDRS* Part I score</td>
<td>8.76 (±1.89)</td>
<td>7.46 (±2.11)</td>
</tr>
<tr>
<td>UPDRS Part II score</td>
<td>13.74 (±3.89)</td>
<td>12.74 (±5.13)</td>
</tr>
<tr>
<td>UPDRS Part III score</td>
<td>29.00 (±11.45)</td>
<td>28.40 (±9.88)</td>
</tr>
<tr>
<td>DD (years)</td>
<td>3.67 (±1.05)</td>
<td>3.53 (±0.99)</td>
</tr>
<tr>
<td>LED (mg)</td>
<td>870.45 (±122.12)</td>
<td>899.87 (±134.76)</td>
</tr>
<tr>
<td>H&amp;Y score</td>
<td>2.80 (±0.80)</td>
<td>2.70 (±0.70)</td>
</tr>
</tbody>
</table>

*Higher score means more severe symptoms. SD, standard deviation; F, female; M, male; MMSE, Mini-Mental State Examination; UPDRS, Unified Parkinson’s Disease Rating Scale; DD, disease duration; LED, levodopa equivalent dose; H&Y, Hoehn and Yahr.
Table 2. Alpha and beta band rhythms and P300 latency.

<table>
<thead>
<tr>
<th>Test</th>
<th>Experimental Group Pre-Test Phase</th>
<th>Sham Group Pre-Test Phase</th>
<th>Experimental Group Post-Test Phase</th>
<th>Sham Group Post-Test Phase</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Alpha band power</td>
<td>8.64 (± 0.29)</td>
<td>8.53 (± 0.94)</td>
<td>9.52 (± 0.74)</td>
<td>8.46 (± 0.68)</td>
</tr>
<tr>
<td>Beta band power</td>
<td>15.58 (± 1.06)</td>
<td>15.24 (± 1.42)</td>
<td>17.71 (± 1.07)</td>
<td>15.19 (± 1.40)</td>
</tr>
<tr>
<td>P300 Latency</td>
<td>310.40 (± 3.48)</td>
<td>310.60 (± 3.74)</td>
<td>305.61 (± 2.24)</td>
<td>309.60 (± 3.46)</td>
</tr>
</tbody>
</table>

SD, standard deviation.

(post = 9.52 ± 0.74, pre = 8.64 ± 0.29; t (14) = 4.34, p < 0.001, d = 0.80). An independent t-test was also applied to analyze the post-test means of the two groups (experimental = 9.52 ± 0.74, sham = 8.46 ± 0.68; t (28) = 6.11, p < 0.001, d = 0.78).

Fig. 1. Alpha band rhythms in the pre- and post-test phases for the experimental and sham groups. Plots show mean and whiskers show standard deviation.

Fig. 2 shows the pre- and post-test beta band rhythms for both groups. RM-ANOVA showed again that the effect of group was not significant (F (1, 28) = 1.32; p = 0.27, η² = 0.10) and the phase group was also not significant (F (1, 28) = 0.76; p = 0.56, η²=0.09). The effect of the group x phase interaction was significant (F (1, 28) = 48.94; p < 0.01, η² = 0.09). Paired t-tests show statistical differences in beta band only for PD patients with anodal tDCS. Beta bands had higher values in the post-test than in the pre-test phase (post = 17.71 ± 1.07, pre = 15.58 ± 1.06; t (14) = 5.21, p < 0.001, d = 0.90). An independent t-test was also applied to analyze the post-test means of the two groups (experimental = 17.71 ± 1.07, sham = 15.19 ± 1.40; t (28) = 6.11, p < 0.001, d = 0.86).

Fig. 3 shows the pre- and post-test P300 latency in both groups. RM-ANOVA showed that the group had no significant effect (F (1, 28) = 2.46; p = 0.126, η² = 0.08) and the phase factor also did not show significant differences (F (1, 28) = 2.1; p = 0.09, η² = 0.09). The results revealed again a significant effect of the interaction group x phases (F (1, 28) = 30.56; p < 0.01, η² = 0.09). This significant interaction indicates that P300 latency was lower in the post-test phase than in the pre-test phase only for PD patients with anodal tDCS. Post-hoc analysis was conducted separately for the two groups. With reference to PD patients with anodal tDCS, paired t-tests showed statistical differences in P300 (t (14) = 4.23, p < 0.001, d = 0.84), while this was not seen in the sham group. An independent t-test was also applied to analyze the post-test means of the two groups (t (28) = 4.56, p < 0.001, d = 0.81). These results indicated that sham tDCS did not significantly change measures of P300 latency in the post-test phase.

Fig. 4 shows comparisons of the absolute spectral power in central electrodes between the experimental and sham groups. There were significant differences in the spectral power in the alpha and beta band rhythms between the pre- and post-test phases (respectively, 8–14 Hz, F = 8.23, p < 0.01; 14–30 Hz, F = 6.83, p < 0.01).
4. Discussion

The present study aimed to investigate the effects of anodal tDCS on the left frontotemporal cortex in patients with PD, focusing on the analysis of neurophysiological functions through EEG. The focus was on evaluating ERPs and alpha and beta brain frequencies as indicators of possible changes in brain activity. The overarching goal was to enhance the understanding of how tDCS could influence these aspects in patients with PD-MCI.

Summarizing our results, alpha and beta band rhythms, as well as spectral power, were higher in the post-test phase compared with the pre-test phase for PD patients with anodal tDCS. P300 latency was lower in the post-test phase than in the pre-test phase, specifically for PD patients with anodal tDCS.

The confirmation of the first hypothesis, which posited that patients undergoing tDCS would exhibit a significant reduction in the onset latency of the P300 event-related potential, aligns with the findings of a prior study conducted by Aksu et al. [64]. The consistency between our results and theirs contributes to the consolidation and expansion of current knowledge in this field. The second hypothesis, suggesting that patients undergoing tDCS would show a significant increase in the power spectrum of the alpha and beta bands, was also confirmed.

Through a comprehensive analysis of the neurophysiological functions involved, this research makes a significant contribution to expanding our understanding of the potential effects of tDCS in the context of PD treatment. Furthermore, it provides essential insights for designing new investigative paths aimed at optimizing therapeutic strategies for PD patients with mild cognitive impairment. Numerous studies, including those by Hadoush et al. [65] and Fregni et al. [66], have explored the effects of stimulating the DLPFC in PD, primarily focusing on the cognitive domain. Our findings, which show a reduction in P300 latency and improved cognitive functioning in PD patients following anodal tDCS over the left DLPFC, are consistent with these studies. This suggests a coherent pattern of cognitive improvement associated with DLPFC tDCS. Studies concentrating on stimulating the primary motor cortex (M1), such as those by Lu et al. [67] and Pol et al. [68], have predominantly investigated motor improvements in PD patients. Although our study primarily assessed cognitive outcomes, the observed cognitive enhancements with anodal
tDCS align with the motor improvements reported in these studies. This indicates a potential dual impact of tDCS on both cognitive and motor functions in PD.

With reference to cerebellar tDCS in PD, its role in balance and motor control has been examined in studies conducted by Lu et al. [67] and Pol et al. [68]. Although our study did not specifically concentrate on balance control, the observed increase in alpha and beta band rhythms aligns with findings from studies targeting the cerebellum. This resonance suggests a broader impact of tDCS on neural modulation, further supporting the potential multifaceted effects of tDCS in PD. However, the severity of clinical symptoms measured by the UPDRS were not related to the empowerment of neurophysiological measures; in fact, the UPDRS scores do not predict the differences between the pre and post-test measures.

The observed changes in P300 latency and alpha and beta band rhythms could reflect neuroplasticity [95–99]. If these changes are sustained over time, they might have positive long-term effects on patients’ cognitive and motor abilities. Moreover, the reduction in P300 latency suggests that patients may experience an improvement in cognitive processing speed and responsiveness [100,101]. PD often leads to cognitive deficits, including slowed processing and attention difficulties [102]. By enhancing cognitive processing, patients might find it easier to engage in daily activities, communicate, and maintain mental clarity [103]. Therefore, a shorter P300 latency could indicate an improvement in attention and the ability to identify relevant stimuli more rapidly [104,105]. This improvement could translate into better focus, concentration, and attention control for individuals with PD, thereby aiding in tasks that require sustained attention. Conversely, an increase in alpha and beta band rhythms could be associated with motor symptoms [106,107]. Studies investigating this association have reported conflicting results. While some have suggested that an increase in beta was associated with the presence of motor symptoms, others have proposed that an increase in beta was associated with the improvement of these symptoms [108–110]. Furthermore, it has been emphasized that the positive or negative nature of this association is linked to the specific symptoms considered [111]. While in the advanced stages of PD, excessive activity in the beta band is well-documented and correlated with motor symptoms [112,113], early-stage PD, characterized by mild cognitive deficits, shows baseline levels of activity in the beta band. In fact, our sample showed an increase in baseline beta band levels only after tDCS. Taken together, the results of this research provide interesting insights into the effect of tDCS on alpha and beta band rhythms in the early stages of PD-MCI.

The integrated approach that combined tDCS with the analysis of neurophysiological parameters, such as ERPs and alpha and beta brain rhythms, constitutes one of the significant strengths that emerged from this research. As emphasized, these parameters represent objective measures of cognitive functioning. Other positive aspects include the exclusive use of tDCS, without the combination of other treatments that could influence the attribution of observed improvements. Additionally, a comparison was conducted with a sham group, which received a tDCS simulation, in order to exclude possible placebo-related effects.

It should be noted that this study has some limitations. Among these is the small sample size, the absence of long-term follow-up to assess the persistence of effects over time, and the absence of a post-treatment UPDRS assessment. The assessment of the tolerability of stimulation was carried out through the collection of direct feedback from participants. While this approach provided us with specific and qualitative information, we acknowledge that the use of a standardized scale could have offered more structured and comparative data [114,115]. We acknowledge the importance of examining P300 amplitude as well as P300 latency, and future research should address this issue. It would be advisable for future studies to involve larger clinical trials, following standardized protocols and including long-term assessments, to establish the duration of the observed effects in patients with mild cognitive decline associated with PD.

5. Conclusions

The current research provides evidence of the efficacy of tDCS on crucial neurophysiological parameters, such as P300 brain response latency and alpha and beta frequency rhythms, in individuals exhibiting PD-MCI. Our results can be summarized in the following key points: (a) tDCS demonstrates a significant effect in reducing P300 response latency and (b) tDCS induces a significant increase in alpha and beta band rhythms.

Availability of Data and Materials

The data and materials are available upon request.

Author Contributions

RAF, RS & AG conceived and designed this research. RAF, RS & AG conducted the experiments and led statistical. RAF & RS wrote the manuscript, revised manuscript, and supervised the project. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Madonna della Consolazione Polyclinic Nursing Home (process approval number prot. 2021-198). Prior to participating in the study, each participant was provided
with detailed information about the study’s purpose and data collection procedures. This information was presented clearly and comprehensively so that participants could make an informed decision about their participation. After receiving these explanations, each participant has provided their informed consent in written form.

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**Conflict of Interest**

The authors declare no conflict of interest.

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