

## Research Article

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# Impact of Biophoton Treatment on Functional Brain Age in Patients with Parkinson's Disease Based on EEG-Derived Metrics

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### ABSTRACT

**Background:** Parkinson's disease (PD) is associated with progressive neurophysiological decline that is incompletely captured by chronological age or clinical scales alone. Functional brain age derived from electroencephalography (EEG) provides a quantitative marker of neural aging and functional integrity.

**Objective:** To evaluate the effect of biophoton-based treatment on EEG-derived functional brain age in patients with Parkinson's disease compared with untreated controls.

**Methods:** Functional brain age was estimated by EEG metrics at baseline and last follow up in 16 participants (10 treatment, 6 control). The primary outcome was change in functional age. Between-group comparisons were performed using Welch's t-test, and an analysis of covariance (ANCOVA) was conducted adjusting for baseline functional age.

**Results:** Treated patients demonstrated a significant reduction in functional brain age (mean 5.1 years) at the last follow up, whereas controls showed no change. After adjustment for baseline functional age, the treatment effect remained significant, with a large effect size.

**Conclusion:** Biophoton treatment was associated with a clinically meaningful and statistically significant improvement in EEG-derived functional brain age in patients with Parkinson's disease ( $P < 0.01$ ). These findings support further investigation of biophoton-based interventions as a potential neurofunctional modulator in neurodegenerative disease.

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### Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized not only by dopaminergic neuronal loss but also by widespread systemic dysfunction, including impaired microcirculation, mitochondrial insufficiency, oxidative stress, and chronic inflammation. These pathological processes interact to exacerbate neuronal vulnerability and accelerate neurodegeneration as the disease progresses [1-3]. Microvascular dysfunction and impaired oxygen delivery have increasingly been recognized as contributors to PD pathology and disease progression [1].

Traditional clinical assessments primarily focus on motor symptoms and functional disability but do not directly quantify underlying neurophysiological aging or systemic contributors to neuronal dysfunction. Quantitative electroencephalography (qEEG) offers a non-invasive approach to measuring large-scale neural dynamics and has been increasingly applied to estimate functional or biological brain age, reflecting neurophysiological

integrity beyond chronological age [4-11].

Recent exploration of biophoton-based interventions has introduced a novel, non-invasive approach aimed at restoring systemic coherence and cellular bioenergetics. Biophotons, ultra-weak photon emissions generated by living cells, have been proposed to regulate mitochondrial cytochrome c oxidase activity, modulate redox balance, and promote biological coherence at the cellular and systemic levels [12-15]. Emerging clinical evidence suggests that such interventions may influence disease-related systemic dysfunctions relevant to neurodegenerative disorders [16-18].

In a recently published 12-day longitudinal case study, continuous biophoton therapy in a patient with advanced PD was associated with progressive normalization of hemorheology, red blood cell morphology, plasma clarity, and microvascular flow, culminating in a physiologically optimal blood terrain by Day 12 [18]. These findings indicate improved oxygen transport, reduced inflammatory burden, and enhanced microcirculatory dynamics—biological processes closely linked to neuronal metabolism and electrophysiological function.

Complementary clinical studies have further reported symptomatic improvement and favorable safety profiles of biophoton therapy in patients with Parkinson's disease and other neurodegenerative conditions [16, 17]. Despite these observations, objective neurophysiological outcome measures capable of quantifying treatment-associated changes in brain function remain limited.

The present study therefore evaluates EEG-derived functional brain age as a quantitative biomarker to assess whether biophoton treatment is associated with measurable improvements in neurophysiological aging in patients with Parkinson's disease, compared with untreated controls.

## Methods

### Study Design and Participants

This observational comparative study enrolled patients with Parkinson's disease who underwent quantitative EEG assessments at baseline and at 2- and/or 4-week follow up. Participants were assigned to either a biophoton intervention group or a control group without biophoton exposure. All included participants had complete EEG-derived functional age data available at baseline and at the final follow up timepoint. The study protocol was approved by an independent Institutional Review Board and registered at ClinicalTrials.gov (Identifier: NCT06147999).

### Active and Non-Active Devices

Patients in the biophoton treatment group slept on a bed equipped with four active biophoton generators, positioned around the bed, and were exposed to the devices during nighttime sleep and any additional time spent in bed during the day. Participants were instructed to sleep at least 8 hours per night. Daytime bed use was not restricted and varied according to individual routine.

Patients in the control group slept on an identical bed setup containing four sham devices matched in appearance and weight but without biophoton emission.

### Functional Brain Age Estimation

Functional brain age was estimated using a predefined rubric integrating EEG-derived metrics, including spectral features and task-related components. Functional brain age is expressed in years to facilitate comparison with chronological age.

Rubric of quantitative EEG-derived functional age: 50% resting EEG rhythms (alpha peak frequency, theta/beta balance, coherence); 30% ERP attention / working-memory latency (P300, vigilance, information processing); 20% motor / reaction-time indices.

The same weighting scheme and computational procedure were applied uniformly across all participants and time points to ensure numerical consistency.

### Statistical Methods

All statistical analyses were performed using standard parametric methods. Functional age was assessed at baseline and at the last

follow up visit for each participant. The primary outcome measure was the change in functional age ( $\Delta$  functional age), calculated as the difference between the last follow up value and the baseline value for each subject, with negative values indicating functional improvement.

Descriptive statistics are reported as mean  $\pm$  standard deviation (SD). Initial between-group comparisons of change in functional age between the treatment and control groups were conducted using a two-sample Welch's t-test to account for unequal variances and unequal sample sizes. A two-sided p-value  $< 0.05$  was considered statistically significant.

To further account for potential baseline differences and regression-to-the-mean effects, an analysis of covariance (ANCOVA) was performed with the last follow up functional age as the dependent variable, treatment group as the fixed effect, and baseline functional age as a covariate. This approach allows estimation of the treatment effect while adjusting for baseline functional age and improves statistical efficiency in small samples. Model assumptions, including linearity between the covariate and outcome and homogeneity of regression slopes, were assessed and deemed acceptable.

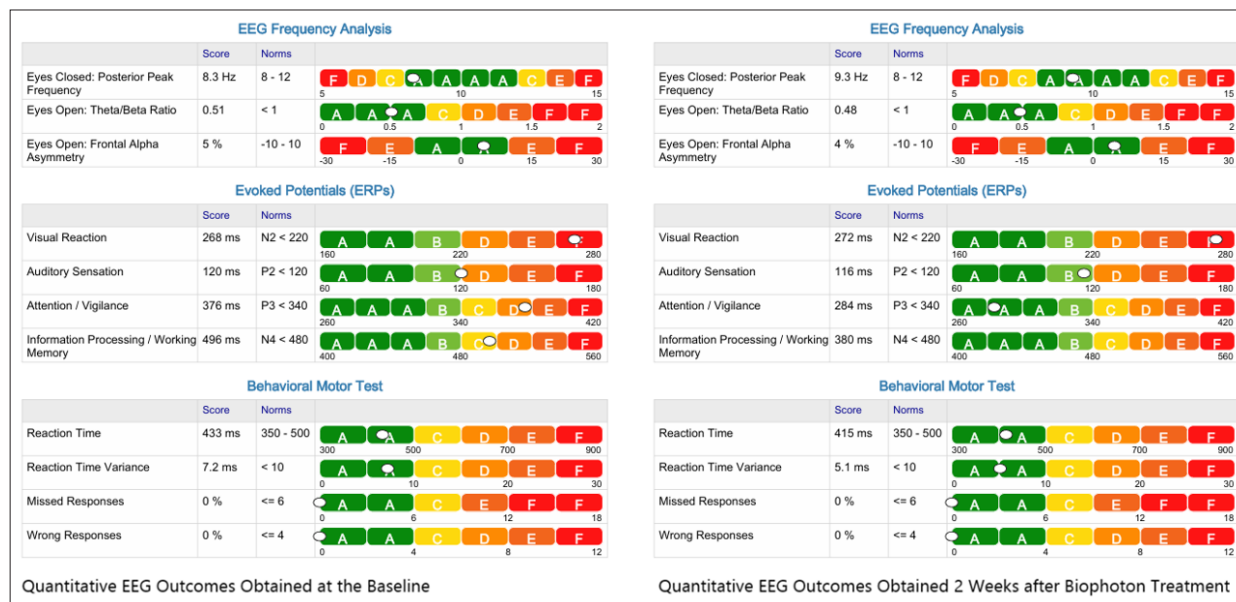
Adjusted group differences are reported as estimated marginal means with corresponding p-values. No imputation was performed for missing data, and analyses were conducted using complete cases only. Given the exploratory nature of this study and the limited sample size, no adjustments were made for multiple comparisons. All statistical tests were prespecified and applied uniformly across groups.

## Results

### Participant Characteristics

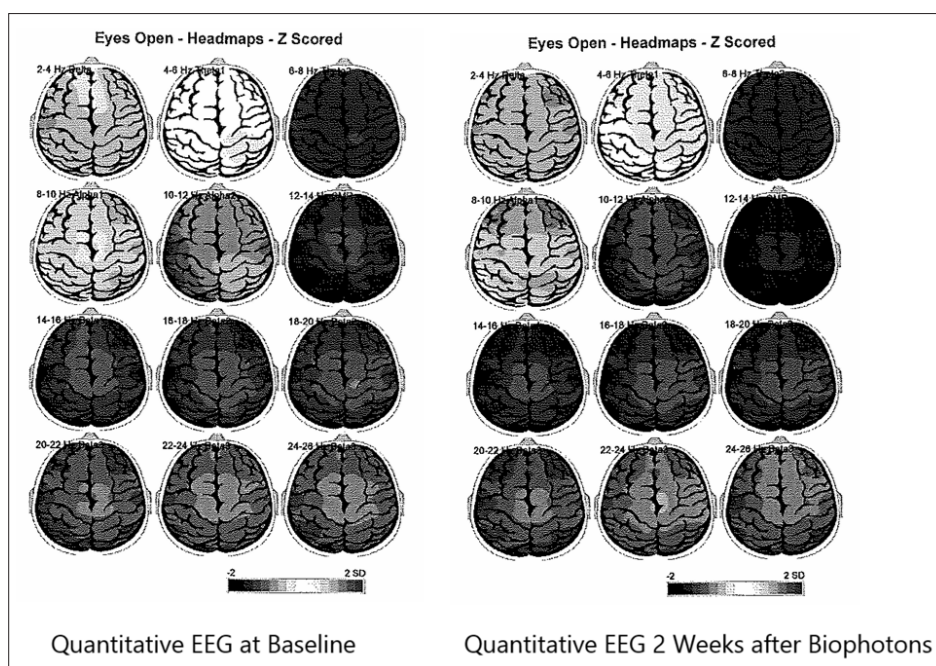
A total of 16 participants were included in the analysis, comprising 10 patients in the biophoton treatment group (4 strong biophoton generators were placed around the volunteer's bed) and 6 in the control group (same weight and looking non-active 4 devices). Baseline functional age values were elevated relative to chronological age in both groups, consistent with underlying neurological impairment. No data imputation was performed, and all analyses were conducted on complete cases.

To quantitatively assess changes in neurophysiological function, Figure 1 summarizes key quantitative electroencephalography (qEEG) outcomes obtained at baseline and two weeks following biophoton treatment. The figure integrates EEG frequency analysis, event-related potentials (ERPs), and behavioral motor performance measures, providing a multidimensional evaluation of cortical arousal, attentional control, information processing, and response efficiency. Presenting these metrics alongside age-adjusted normative ranges allows direct visualization of treatment-associated shifts toward more optimal neural function, complementing the statistical analyses and supporting changes observed in EEG-derived functional brain age.



**Figure 1:** Quantitative EEG Outcomes at Baseline and Two Weeks After Biophoton Treatment. Quantitative electroencephalography (qEEG) results obtained at baseline (left) and two weeks after biophoton treatment (right), including EEG frequency analysis, event-related potentials (ERPs), and behavioral motor measures. Frequency metrics assess cortical arousal and attentional balance, ERPs index sensory processing, attention, and working memory, and behavioral tests evaluate reaction time and response accuracy. Color-coded bands indicate age-adjusted normative ranges. Compared with baseline, post-treatment measures show a shift toward more optimal frequency organization, faster ERP latencies particularly in attention and working memory components and improved reaction time consistency. These changes are consistent with enhanced neural processing efficiency and a reduction in EEG-derived functional brain age following biophoton treatment.

To further illustrate spatial and frequency-specific changes in neural activity, Figure 2 presents eyes-open quantitative EEG (qEEG) topographic maps expressed as Z-scores relative to age-matched normative data at baseline and two weeks after biophoton treatment. These maps provide a visual representation of regional deviations in EEG power across canonical frequency bands, enabling assessment of large-scale cortical network organization and normalization patterns over time. Comparison of baseline and post-treatment maps highlights treatment-associated shifts in frequency distribution and spatial coherence that complement the numerical qEEG metrics and support changes observed in EEG-derived functional brain age.



**Figure 2:** Z-Scored Eyes-Open Quantitative EEG Topographic Maps at Baseline and Two Weeks After Biophoton Treatment. Topographic maps of eyes-open quantitative EEG power expressed as Z-scores relative to age-matched normative databases at baseline (left) and two weeks after biophoton treatment (right). Each map represents frequency-specific power distribution across the scalp from

delta (2–4 Hz) through beta (24–28 Hz) bands. Color intensity reflects deviation from normative values ( $\pm 2$  SD). Compared with baseline, post-treatment maps demonstrate reduced abnormal low-frequency activity and improved normalization of alpha and beta band distributions, consistent with enhanced cortical arousal, network efficiency, and reduced EEG-derived functional brain age following biophoton treatment.

### Change in Functional Age

In the treatment group, functional age decreased from baseline to last follow up in the majority of patients, indicating functional improvement. The mean change in functional age was  $-5.1 \pm 4.0$  years. In contrast, the control group showed no change in functional age over the same period (mean change  $0.0 \pm 0.0$  years).

Table 1 summarizes the chronological ages and EEG-derived functional brain ages of participants in the treatment and control groups at baseline and at last follow up. Presenting individual-level data allows comparison of functional age relative to chronological age within and between groups and illustrates longitudinal changes over time. The table highlights baseline elevation of functional brain age relative to chronological age in both groups, as well as differential trajectories between treated and untreated participants across the 4-week study period.

**Table 1: The Chronological and Functional Ages of the Two Groups**

	Patient ID	Chronological Age	Functional Age Baseline	Functional Age at last follow up
Treatment	PD-102	51	67	60
	PD-107	63	79	72
	PD-126	60	64	58
	PD-127	72	79	74
	PD-129	51	56	55
	PD-142	65	76	69
	PD-146	69	74	71
	PD-139	69	82	76
	PD-193	71	74	62
	PD-195	39	68	71
Control	PD-145	73	74	74
	PD-155	62	70	70
	PD-183	68	73	73
	PD-123	69	75	75
	PD-124	65	78	78
	PD-116	63	74	74

### ANCOVA-Adjusted Treatment Effect

To account for baseline functional age and potential regression-to-the-mean effects, an analysis of covariance (ANCOVA) was performed with last follow up functional age as the dependent variable and baseline functional age included as a covariate. After adjustment, a significant treatment effect remained evident. The adjusted mean functional age at follow up was 67.4 years in the treatment group compared with 72.9 years in the control group, corresponding to an adjusted between-group difference of  $-5.5$  functional years favoring treatment ( $p = 0.005$ ).

Baseline functional age was a significant predictor of follow up functional age ( $p < 0.001$ ), confirming its strong association with longitudinal outcomes. The magnitude of the treatment effect

was large, with a partial eta-squared ( $\eta^2_p$ ) of 0.47, indicating that approximately 47% of the variance in follow up functional age was attributable to treatment status after adjusting for baseline functional age.

Table 2 presents a statistical summary of changes in EEG-derived functional brain age for the treatment and control groups. The table reports group sample size, mean change in functional age, and standard deviation, enabling direct comparison of the magnitude and variability of longitudinal functional age changes between groups. Negative values indicate reduction (or improvement) in functional brain age over the study period.

**Table 2: Statistical Summary of the Changes of the Two Groups in Functional Ages**

Group	n	Mean $\Delta$ (years)	SD
Treatment	10	-5.1	4.0
Control	6	0.0	0.0

Between-group comparison of change scores demonstrated a statistically significant difference favoring treatment (Welch's  $t$ -test,  $p = 0.003$ ), indicating that treated patients experienced greater functional age improvement than controls.

### Summary of Findings

Together, both unadjusted and baseline-adjusted analyses consistently demonstrate a robust and clinically meaningful reduction in functional brain age among treated patients, while no improvement was observed in the control group.

### Discussion

The present findings demonstrate that biophoton treatment was associated with a statistically significant and clinically meaningful reduction in EEG-derived functional brain age in patients with Parkinson's disease. Both unadjusted and baseline-adjusted analyses consistently favored treatment, with an estimated improvement of approximately five functional years relative to controls. These results suggest that biophoton therapy may modulate neurophysiological aging processes detectable at the network level.

### Mechanistic Interpretation

The convergence of EEG-derived functional brain age improvement with previously reported hemorheological and microvascular restoration suggests a plausible multilevel biological mechanism linking systemic circulation, mitochondrial bioenergetics, and neural network function. Parkinson's disease is increasingly recognized as a multisystem disorder in which impaired microcirculation, chronic inflammation, and mitochondrial dysfunction jointly contribute to progressive neural impairment [1-3].

Live blood microscopy findings from the published 12-day biophoton therapy report demonstrated marked normalization of red blood cell morphology, dispersion, plasma clarity, and flow dynamics, indicating restoration of blood rheology and microvascular perfusion [18].

Improved red blood cell zeta potential and reduced rouleaux formation are expected to enhance capillary transit, tissue oxygen delivery, and nutrient exchange processes critical for sustaining neuronal metabolism and synaptic function. In Parkinson's disease, microvascular compromise and impaired oxygenation exacerbate

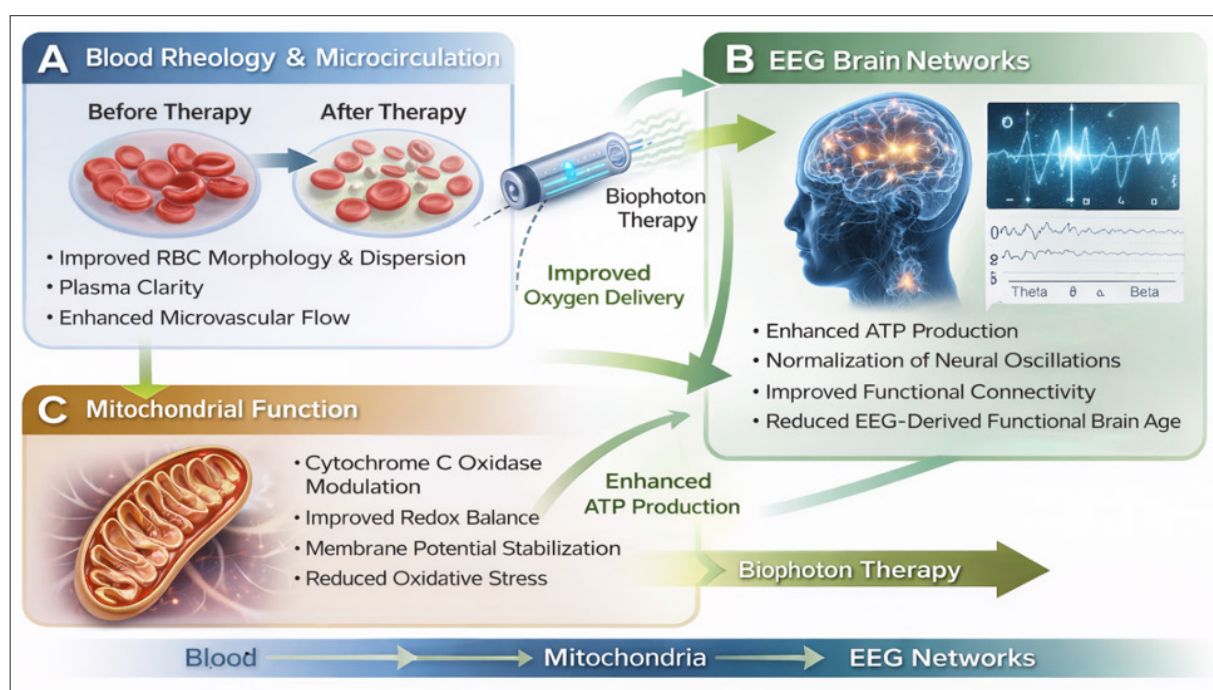
mitochondrial stress and accelerate neurodegenerative cascades [1-4].

At the cellular level, biophoton exposure has been proposed to influence mitochondrial function through modulation of cytochrome c oxidase activity, redox signaling, and membrane potential stabilization [12-15]. Improved mitochondrial efficiency would be expected to reduce oxidative stress, enhance ATP availability, and stabilize neuronal firing patterns. These effects are consistent with the observed reduction in systemic inflammatory markers and detoxification debris reported during the blood-based time course [18].

EEG-derived functional brain age likely reflects the downstream integration of these systemic and cellular improvements at the network level. Neural oscillatory activity is highly sensitive to metabolic state, oxygen availability, and synaptic efficiency. Restoration of microcirculatory flow and mitochondrial bioenergetics may therefore translate into improved synchronization, reduced pathological slowing, and enhanced

functional connectivity manifesting as a measurable reduction in EEG-derived functional age [5-9]. The persistence of the treatment effect after adjustment for baseline functional age supports a model in which systemic biological changes contribute meaningfully to neural functional recovery rather than reflecting statistical artifact alone.

To conceptualize the multilevel biological processes potentially underlying the observed EEG-derived functional brain age improvements, Figure 3 presents a schematic model linking systemic circulation, cellular bioenergetics, and large-scale neural network function. The figure illustrates a proposed pathway in which improvements in blood rheology and microcirculation support mitochondrial function and redox balance, which in turn facilitate normalization of cortical network dynamics measurable by quantitative EEG. This integrative framework provides a mechanistic context for interpreting convergent blood-based, mitochondrial, and electrophysiological findings observed following biophoton treatment.



**Figure 3:** Proposed Mechanistic Pathway Linking Blood Rheology, Mitochondrial Function, and EEG-Derived Functional Brain Age. Biophoton therapy is hypothesized to initiate systemic restoration beginning with improved blood rheology and microcirculation (A), which enhances mitochondrial function (B), ultimately leading to improved brain network dynamics measurable by EEG-derived functional brain age (C). The arrows indicate a proposed directional relationship from systemic circulation to cellular energy metabolism and ultimately to network-level brain function.

This is a schematic overview of the hypothesized multilevel mechanism underlying functional brain age improvement following biophoton therapy. Biophoton exposure is proposed to initiate systemic restoration at the level of blood rheology, including improved red blood cell (RBC) morphology, dispersion, plasma clarity, and microvascular flow, as demonstrated by longitudinal live blood analysis in Parkinson's disease patients [18]. Enhanced microcirculation and oxygen delivery are expected to support mitochondrial bioenergetics through improved cytochrome c oxidase activity, redox balance, and membrane potential stabilization. These cellular-level improvements may, in turn, facilitate normalization of large-scale neural network dynamics, reflected in improved EEG oscillatory organization and a reduction

in EEG-derived functional brain age [17]. The arrows indicate a proposed directional relationship from systemic circulation to cellular energy metabolism and ultimately to network-level brain function.

Taken together, these findings support a coherent mechanistic framework in which biophoton therapy may initiate systemic restoration beginning with blood rheology and microcirculation, propagating through mitochondrial and redox pathways, and culminating in improved large-scale neural network function measurable by EEG. While causal inference cannot be definitively established in the present study, the alignment of blood-based, mitochondrial, and electrophysiological evidence provides a

biologically plausible explanation for the observed reduction in functional brain age and warrants further investigation in larger, multimodal studies.

Importantly, the EEG-based findings are concordant with previously reported systemic and cellular changes observed during biophoton therapy. In the published 12-day live blood analysis case report, progressive restoration of red blood cell morphology, plasma composition, and microvascular flow was documented during continuous biophoton [18]. Improved hemorheology and microcirculation are likely to enhance cerebral oxygen delivery and mitochondrial efficiency processes essential for maintaining neuronal electrophysiological stability.

Additional clinical investigations have reported improvements in motor and non-motor symptoms of Parkinson's disease during biophoton therapy, with no significant adverse effects [16-17]. Together, these studies support a convergent biological framework in which biophoton exposure may reduce systemic inflammation, normalize redox signaling, and enhance mitochondrial function mechanisms that are strongly implicated in PD pathophysiology [1-4, 16-18].

EEG-derived functional brain age offers a complementary and objective measure that captures brain-level functional organization rather than peripheral or symptomatic changes alone [19, 20]. The persistence of a significant treatment effect after adjustment for baseline functional age strengthens the inference that observed improvements are not solely attributable to regression to the mean or baseline imbalance.

While the present study is limited by sample size and observational design, the consistency between EEG-based outcomes and prior biological and clinical findings provides convergent support for further investigation. Future studies integrating EEG, blood-based biomarkers, clinical scales, and longer-term follow up will be essential to clarify mechanisms, assess durability, and determine clinical relevance.

In summary, when interpreted alongside existing biological and clinical evidence [12-18], the present findings support the hypothesis that biophoton therapy may favorably influence systemic and neural aging processes in Parkinson's disease. EEG-derived functional brain age may serve as a valuable quantitative endpoint for future interventional studies in neurodegenerative disorders.

### Limitations

Several limitations should be considered when interpreting the findings of this study. First, the sample size was relatively small, which limits statistical power and generalizability. Although statistically significant effects and large effect sizes were observed, these results should be interpreted as exploratory and hypothesis-generating.

Second, the study employed a nonrandomized, observational design. Consequently, unmeasured confounding factors, such as disease duration, medication status, comorbidities, or lifestyle variables, may have influenced outcomes. While adjustment for baseline functional brain age using ANCOVA reduces the impact of baseline imbalance and regression-to-the-mean effects, residual confounding cannot be excluded.

Third, functional brain age was derived from EEG-based metrics using a predefined algorithm. Although EEG-derived brain age measures are supported by prior literature, the specific weighting scheme and composite metric used in this study have not yet been independently validated across external cohorts. As such, absolute functional age values should be interpreted cautiously, with greater confidence placed on within-subject and between-group changes.

Fourth, follow up duration was limited to a short-term interval, precluding assessment of the durability of observed effects. It remains unknown whether improvements in EEG-derived functional brain age persist over longer periods or translate into sustained clinical benefit.

Finally, clinical symptom scales and standardized functional assessments were not systematically integrated with EEG outcomes. Future studies combining biological, clinical, and electrophysiological endpoints will be necessary to better establish clinical relevance and mechanistic specificity.

### Conclusion

Biophoton-based treatment for two weeks was associated with a statistically significant and clinically meaningful reduction in EEG-derived functional brain age in patients with Parkinson's disease. These findings support further investigation of biophoton interventions as a potential modality for modulating neurofunctional aging in neurodegenerative disorders.

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### References

1. Sengupta T, Devine M, Petzinger GM (2020) Microvascular contributions to Parkinson's disease pathology. *Front Neurol* 11: 1000.
2. Gao HM, Hong JS (2008) Why neurodegenerative diseases are progressive: uncontrolled inflammation drives disease progression. *Trends Immunol* 29: 357-365.
3. Lin MT, Beal MF (2006) Mitochondrial dysfunction and oxidative stress in neurodegenerative diseases. *Nature* 443: 787-795.
4. Klassen BT, Hentz JG, Shill HA, Driver Dunckley E, Evidente VG, et al. (2011) Quantitative EEG as a predictive biomarker for Parkinson disease dementia. *Neurology* 77: 118-124.
5. Sun H, Paixao L, Oliva JT, Rosenthal ZP, Valerio J, et al. (2019) Brain age from the electroencephalogram of sleep: Conceptualization and machine learning models. *Nat Sci Sleep* 10: 85-95.
6. Zhang D, She Y (2024) Brain age estimation from overnight sleep electroencephalography with multi-flow sequence learning. *Nat Sci Sleep* 16: 115-129.
7. Vecchio F, Francesca Miraglia, Francesca Alù, Matteo Menna, Elda Judica, et al. (2020) Classification of Alzheimer's disease with respect to physiological aging using innovative EEG biomarkers. *J Alzheimers Dis* 75: 1253-1261.
8. Paixao L, Sun H, Hogan J, Robert Thomas, M Brandon Westover, et al. (2020) Excess brain age in the sleep electroencephalogram predicts life expectancy: Brain age index as a biomarker. *Brain Stimul* 13: 1300-1306.

9. Novak K, Chase BA, Narayanan J, Indic P, Markopoulou K (2022) Quantitative electroencephalography as a biomarker for cognitive dysfunction in Parkinson's disease. *Front Aging Neurosci* 13: 804991
10. Di Biase L, Ricci L, Caminiti ML, Pecoraro PM, Carbone SP, et al. (2023) Quantitative high-density EEG brain connectivity evaluation in Parkinson's disease: The phase locking value (PLV). *J Clin Med* 12: 1450.
11. Cozac VV, Popa L (2016) Quantitative EEG and cognitive decline in Parkinson's disease: A review of mechanisms and predictive value. *Clin Neurophysiol Pract* 1: 58-66.
12. Mould RR, Mackenzie AM, Kalampouka I, Nunn AVW, Thomas EL, et al. (2024) Ultra weak photon emission a brief review. *Front Physiol* 15: 1348915.
13. Hamblin MR (2016) Shining light on the head: Photobiomodulation for brain disorders. *BBA Clinical* 6: 113-124.
14. Hamblin MR (2022) Do Biophotons Play Any Role in Transcranial Photobiomodulation of the Brain? Photobiomodulation, Photomedicine, and Laser Surgery 40: 11-15.
15. Paolis LD, Francini R, Davoli I, De Matteis F, Scordo A, et al. (2024) Biophotons: A Hard Problem. *Appl Sci* 14: 5496.
16. Liu JZ, Smotrysts M, Robinson SD, Liu S, Gu HY (2025) Therapeutic Benefits of Biophoton Therapy in Parkinson's Disease: Clinical Evidence from a Pilot and Real-World Study. *J Neurol Res Rev Rep* 7: 1-6.
17. Liu JZ, Smotrysts MA, Robinson SD, Yu HX, Liu SX, et al. (2025) Quantitative EEG Evidence of Functional Brain Recovery in Parkinson's Disease Following Biophoton Therapy. *J Neurol Res Rev Rep* 7: 14-20.
18. Hu Y, Gu HY, Liu JZ (2025) Twelve-Day Live Blood Analysis Reveals Hemorheological and Microvascular Restoration in a Parkinson's disease patient Following Biophoton Therapy. *Journal of Neurology Research Reviews & Reports* 7: 1-6.
19. Latreille V, Montplaisir J (2016) Electroencephalographic prodromal markers of dementia in Parkinson's disease: REM sleep EEG slowing predicts development of dementia. *Brain* 139: 1189-1199.
20. Spoa M (2025) Resting-state quantitative EEG measures and cognitive decline in Parkinson disease: A systematic review. *J Neural Transm (Vienna)* 132: 501-517.

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