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Improvement in EEG-Derived Functional Brain Age During a 4-Week Biophoton Therapy Intervention in 5 Patients with Alzheimer's Disease – A Case Report

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ABSTRACT

Background: Alzheimer's disease (AD) is associated with progressive cognitive impairment and characteristic electrophysiological alterations on Electroencephalography (EEG). EEG-derived functional brain age, integrating quantitative EEG (qEEG) and Event-Related Potential (ERP) features, has emerged as a surrogate marker of neurophysiological aging that may diverge from chronological age in neurodegenerative conditions.

Methods: This report describes a longitudinal case series of five patients with mild to moderate Alzheimer's disease who underwent serial qEEG assessments during a 4-week biophoton therapy intervention. EEG recordings were obtained at baseline and at follow-up time points ranging from 2 to 6 weeks. Functional brain age was calculated using a fixed-weight composite model incorporating posterior alpha rhythm integrity, theta/beta balance, and ERP latency measures related to attention and memory, applied consistently across all time points.

Results: At baseline, all patients demonstrated electrophysiological profiles consistent with advanced functional brain aging relative to chronological age. Across cases, longitudinal analysis revealed directionally favorable changes in multiple EEG features, including normalization of posterior alpha rhythms and shortening of ERP latencies. These changes corresponded to reductions in EEG-derived functional brain age ranging from approximately 3 to 8 years over short observation intervals, with variability in magnitude and timing among individuals.

Conclusion: This case series demonstrates that qEEG-derived functional brain age is a sensitive and responsive surrogate biomarker capable of capturing short-interval neurophysiological changes in patients with Alzheimer's disease during biophoton therapy exposure. Although causality and clinical efficacy cannot be inferred from these observations, the findings support further controlled investigation of functional brain age as an objective outcome measure for evaluating non-pharmacologic interventions in neurodegenerative disease.

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Introduction

Alzheimer's Disease (AD) is characterized by progressive cognitive decline accompanied by reproducible electrophysiological abnormalities on Electroencephalography (EEG). Common EEG features include a reduction in posterior dominant alpha frequency, impaired vigilance regulation, increased slow-wave activity, and delayed Event-Related Potential (ERP) components such as the P300 and P3b, reflecting deficits in attention, information processing speed, and working-memory function [1-3]. These electrophysiological alterations correlate with disease severity and cognitive impairment and are increasingly recognized as objective biomarkers of functional neural aging in Alzheimer's disease [4,5].

Quantitative EEG (qEEG) based functional brain age integrates spectral and temporal electrophysiological features, including alpha rhythm integrity, theta/beta balance, and ERP latency measures, into a composite index reflecting the functional maturity and efficiency of neural networks relative to age-matched normative data [4-9]. Unlike chronological age, functional brain age captures neurophysiological organization and processing efficiency, which may diverge substantially in neurodegenerative conditions and can change dynamically over short time intervals [10,11].

Biophoton therapy is a non-pharmacologic intervention that delivers non-thermal photon emissions within biologically relevant wavelength ranges. Previous clinical studies have reported that cognitive performance, cerebral blood circulation, and EEG measurements were significantly improved following a 4-week biophoton therapy period. Although the underlying mechanisms remain under investigation, observational and experimental studies suggest that biophoton-based interventions may influence

cellular energy regulation, mitochondrial function, and large-scale neural network coherence, processes that are closely linked to electrophysiological aging and cognitive function.

In this context, EEG-derived functional brain age provides a quantitative and sensitive framework for assessing short-interval neurophysiological changes in Alzheimer's disease. The present case series evaluates longitudinal changes in functional brain age during a 4-week biophoton therapy intervention in patients with Alzheimer's disease, with the aim of characterizing electrophysiological trajectories and exploring the utility of functional brain age as a surrogate biomarker for monitoring non-pharmacologic interventions.

Materials and Methods

Study Design

The clinical investigation was conducted as a randomized, triple-blinded, placebo-controlled trial and was registered at ClinicalTrials.gov (Identifier: NCT06147999). Among enrolled participants, five individuals with mild to moderate Alzheimer's Disease (AD) who were able to complete high-resolution quantitative Electroencephalography (qEEG) assessments were included in this report. These cases were selected to evaluate changes in functional brain age and neurophysiological biomarkers associated with exposure to strong biophoton generators over a 4-week intervention period. Participants who were unable to undergo qEEG testing were excluded from the present analysis.

Ethics and Regulatory Compliance

The study protocol was reviewed and approved by an independent Institutional Review Board (IRB). Written informed consent was obtained from all participants or their legally authorized representatives in accordance with the principles outlined in the Declaration of Helsinki.

Investigational Product and Mode of Administration

Tesla BioHealing® biophoton generators are classified as wellness products intended for use by individuals in stable health conditions with minimal risk. During the study, four biophoton generators were positioned beneath the participant's hotel bed, and Participants slept on the bed nightly throughout the intervention period. Caregivers documented observed changes in cognition and brain-related function and completed standardized study questionnaires at baseline and at weekly intervals during the 4-week study period.

Outcome Measures

The primary outcomes were changes in established qEEG and Event-Related Potential (ERP) biomarkers, including:

- Posterior Peak Alpha Frequency (cognitive processing speed),
- Theta/Beta Ratio (attention and inattention),
- Frontal Alpha Asymmetry (mood-related activity), and
- P300 latency and amplitude (attention and memory-related ERP markers) [12].

Neurophysiological Assessment Platform

Quantitative EEG data were acquired using the Brain View system (Medeia), an FDA-cleared hardware and software platform employed in this study [13-16]. Brain View integrates EEG recordings, ERP processing, and neuropsychological testing to support objective and comprehensive brain function assessments. The system captures brainwave activity, auditory and visual evoked potentials, and physiological stress responses, enabling evaluation of neurocognitive function within approximately 30 minutes. Data are compared against a large normative database to identify deviations associated with neurological and neuropsychiatric conditions, including Alzheimer's disease, Mild Cognitive Impairment (MCI), Traumatic Brain Injury (TBI), depression, anxiety, PTSD, and ADHD, as described by the manufacturer (https://www.brainview.com). Brain View is widely utilized in both academic research and clinical settings to bridge subjective symptom reporting with quantifiable neurophysiological measures.

Duration of Intervention

Participants underwent continuous exposure to biophoton therapy for up to 4 weeks, with one participant followed for an extended period of 6 weeks. The intervention was non-invasive, involved no pharmacologic agents, and did not require alterations to concurrent medical treatments. The study design emphasized longitudinal electrophysiological monitoring rather than direct modification of clinical outcomes.

Definition of Functional Brain Age

Functional brain age was defined as an electrophysiological surrogate marker derived from quantitative EEG (qEEG) and Event-Related Potential (ERP) features known to change with normal aging and Alzheimer's disease. Key contributing features included posterior alpha rhythm integrity, theta/beta balance, and ERP latency measures related to attention and working memory. This metric reflects the functional maturity and efficiency of neural networks relative to age-matched normative data and does not constitute a diagnostic or clinical age determination.

Age-matched normative comparisons were used to contextualize EEG and ERP measures; however, functional brain age estimates were interpreted primarily as within-subject longitudinal changes, rather than as absolute population-level predictions.

EEG Acquisition

EEG recordings were obtained at multiple timepoints, including baseline (Week 0) and follow-up assessments at Week 2, Week 4, and, when available, Week 6. All EEG assessments were conducted using standardized recording and analysis protocols.
Each EEG evaluation included:

- Frequency-domain analysis, including posterior dominant frequency and spectral balance across alpha, beta, and theta bands
- Event-related potential analysis, assessing visual and auditory processing latencies, attention and vigilance (P3), and working-memory processing (P3b)

Functional Brain Age Model

Functional brain age was calculated using a fixed-weight composite model applied consistently across all patients and timepoints. The model incorporated the following components:

- Alpha rhythm maturity and vigilance regulation: 50%
- ERP latency measures (P2, P3, P3b): 30%
- Theta/beta balance and global slowing indices: 20%

Fixed component weights were selected to minimize model instability and ensure numerical comparability in longitudinal analysis, prioritizing internal consistency and reproducibility over optimization to a specific dataset.

Justification of a Physiology-Driven Approach

A physiology-driven composite model was selected in preference to a machine-learning–based brain age approach to preserve interpretability and enable direct linkage between individual electrophysiological features and established mechanisms of neural aging in Alzheimer's disease. This approach facilitates

transparent, biologically grounded interpretation and supports longitudinal within-subject comparison using stable and well-characterized qEEG and ERP markers.

Results (Case Presentation)

Due to the limited number of participants who were able to complete qEEG assessments, this report presents findings only from the Treatment group. Participants in the Control group exhibited minimal changes, and given the insufficient statistical power for meaningful comparison, their data were not included in the present case report.

Case 1: AD-128

Longitudinal EEG Findings. Figure 1 summarizes qEEG, ERP, and behavioral motor outcomes at baseline, Week 2, and Week 4. At baseline, electrophysiological patterns were consistent with accelerated functional brain aging, including reduced posterior dominant frequency, delayed attentional and working-memory ERP components, and slowed reaction time.

After 2 weeks of biophoton therapy, improvements were observed across multiple domains, including increased posterior alpha frequency, shortened ERP latencies related to information processing and working memory, and improved behavioral performance. These trends persisted and further stabilized by Week 4, indicating progressive normalization of electrophysiological and neurocognitive markers.

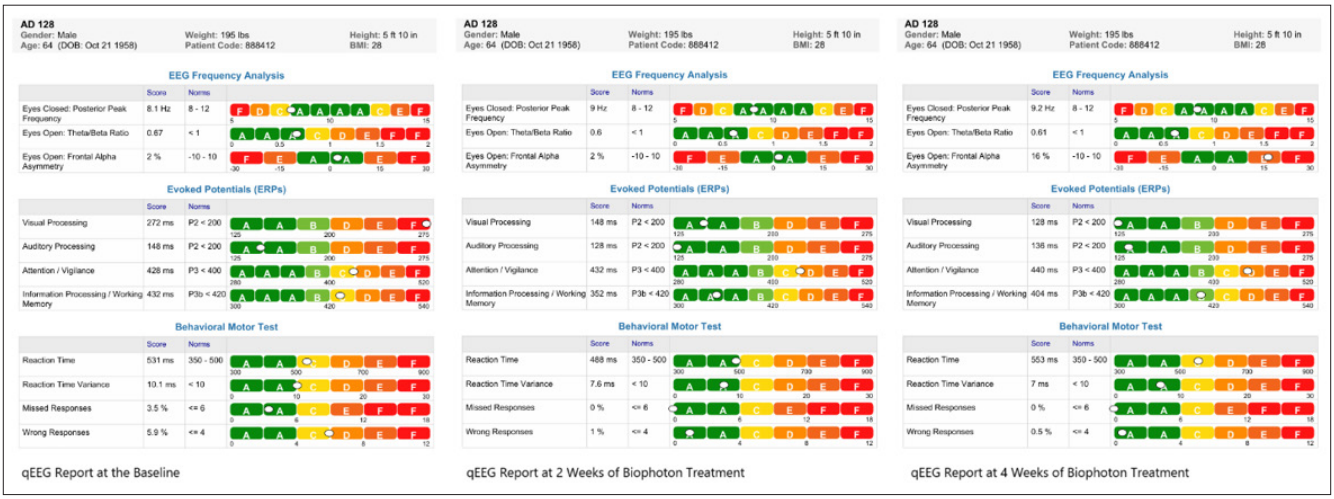

Figure 1: Longitudinal qEEG, ERP, and behavioral motor outcomes in patient AD-128 during biophoton therapy. Quantitative EEG (qEEG) frequency analysis, Event-Related Potentials (ERPs), and behavioral motor test results are shown for patient AD-128 at baseline, 2 weeks, and 4 weeks of biophoton therapy. EEG frequency measures include eyes-closed posterior peak frequency, eyes-open theta/beta ratio, and frontal alpha asymmetry, displayed relative to age-matched normative ranges. ERP measures include visual and auditory processing latencies, attention/vigilance (P3), and information processing/working memory (P3b). Behavioral motor outcomes include reaction time, reaction time variance, missed responses, and wrong responses. Across the 4-week intervention period, progressive shifts toward normative ranges are observed in multiple electrophysiological and behavioral indices, indicating improved neural oscillatory organization, information-processing efficiency, and task performance. These longitudinal changes are concordant with the reduction in EEG-derived functional brain age observed in this patient.

Figure 2 illustrates the longitudinal changes in eyes-open quantitative EEG (qEEG) headmaps in patient AD-128 at baseline, 2 weeks, and 4 weeks of biophoton therapy. Z-scored topographical maps across standard frequency bands (delta through high beta) demonstrate progressive normalization of cortical oscillatory patterns over the 4-week intervention period. At baseline, the qEEG shows widespread deviations from age-matched norms, characterized by altered low-frequency activity and reduced organization of alpha and beta rhythms. After 2 weeks of biophoton therapy, partial normalization is observed, particularly in mid-frequency bands associated with attention and cognitive processing. By 4 weeks, further stabilization and harmonization of spectral power distribution are evident, with reduced extreme z-score deviations and improved balance across alpha, SMR, and beta frequencies. These electrophysiological changes are consistent with the observed reduction in qEEG-derived functional brain age over the same period.

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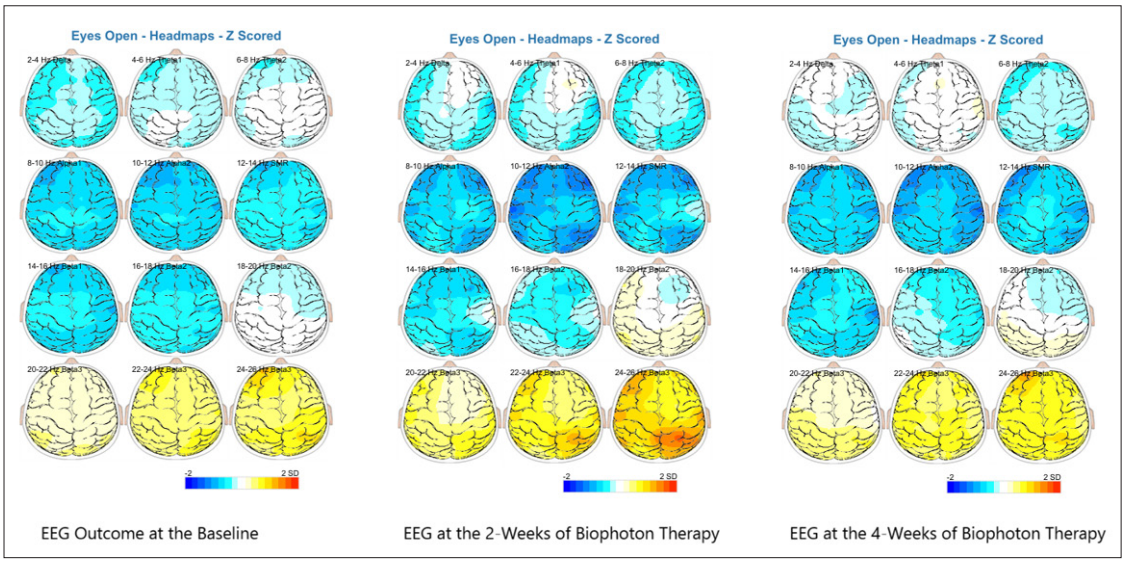

Figure 2: Longitudinal eyes-open quantitative EEG (qEEG) headmaps in patient AD-128 during biophoton therapy. Topographical Z-scored qEEG headmaps across standard frequency bands (2–26 Hz) are shown at baseline, 2 weeks, and 4 weeks of biophoton therapy. Colors represent deviations from age-matched normative values, expressed in Standard Deviation (SD) units (blue = below norm; yellow/red = above norm). At baseline, the EEG demonstrates widespread spectral deviations and altered organization across low- and mid-frequency bands. After 2 weeks of therapy, partial normalization of spectral power distribution is observed, particularly within alpha, SMR, and beta frequency ranges. By 4 weeks, further stabilization and reduction of extreme Z-score deviations are evident, indicating improved balance and organization of cortical oscillatory activity. These longitudinal qEEG changes parallel to the reduction in EEG-derived functional brain age observed over the same period.

Table 1: Functional Brain Age by Timepoint

Timepoint	Chronological Age	Functional Brain Age	Functional Brain Age Reversed from Baseline
Baseline	64	74	0
Week 2	64	70	4
Week 4	64	68	6

Baseline: Low-Normal Posterior Alpha Frequency with Impaired Alpha Arrest; Delayed P300 and P3b Latencies; Reduced Frontal and Temporal qEEG Power.
Week 2: Improved Posterior Alpha Organization; Shortened P3b Latency; Improved Vigilance Regulation.

Week 4: Further Stabilization of Alpha Rhythms; Continued ERP Efficiency Improvements; Improved Network Coherence.

Case 2: AD-110

Patient Characteristics

Patient AD-110 was a 70-year-old individual with Alzheimer's disease who underwent serial qEEG assessments at baseline, Week 2, and Week 4.

Baseline qEEG Profile

Baseline qEEG revealed slowed posterior alpha activity and delayed ERP latencies, particularly in attention and working-memory components. These findings were consistent with advanced functional brain aging.

Longitudinal qEEG Changes

At Week 2, EEG measures demonstrated partial normalization of ERP latencies, with modest changes in alpha frequency. By Week 4, electrophysiological patterns remained relatively stable, with continued improvement in ERP processing speed and preserved spectral balance.

Functional Brain Age Trajectory

Functional brain age demonstrated early improvement followed by stabilization across the three timepoints, with changes driven primarily by ERP latency normalization rather than alpha frequency shifts. Functional brain age was reduced by 3 years by week 4.

Case 3: AD-121

Patient Characteristics

Patient AD-121 was a 60-year-old individual diagnosed with Alzheimer's disease who underwent baseline and Week 2 qEEG assessments.

Baseline qEEG Profile

At baseline, qEEG demonstrated preserved alpha rhythm frequency with delayed ERP components and borderline theta/beta balance, suggesting inefficient cognitive processing despite relatively intact oscillatory frequency.

Longitudinal qEEG Changes

At Week 2, ERP latencies shortened, and theta/beta balance improved, indicating enhanced attentional regulation and processing efficiency.

Functional Brain Age Trajectory

Functional brain age decreased from 62 years at baseline to 57 years at Week 2, corresponding to a 5-year improvement over the short observation interval.

Case 4: AD-123

Patient Characteristics

Patient AD-123 was a 79-year-old individual with Alzheimer's disease who underwent qEEG assessments at Week 2 and Week 3.

qEEG Profile

Both qEEG assessments demonstrated pronounced ERP slowing, reduced vigilance regulation, and impaired behavioral task performance. While posterior alpha frequency was relatively preserved, higher-order processing measures remained delayed.

Functional Brain Age Trajectory

Functional brain age estimates remained within an advanced age range across the two timepoints, with modest short-interval fluctuations. This case illustrates inter-individual variability and demonstrates a reduction in functional brain age of approximately 5 years.

Case 5: AD-126 (Extended Follow-up Case)

Patient Characteristics.

Patient AD-126 underwent multiple qEEG assessments across baseline, Week 2, Week 4, and Week 6.

Baseline EEG Profile

Baseline qEEG demonstrated marked posterior alpha slowing, elevated theta activity, and delayed ERP components consistent with advanced functional brain aging.

Longitudinal EEG Changes

Serial qEEG assessments demonstrated progressive normalization of alpha rhythms and shortening of ERP latencies across successive timepoints, with the most pronounced improvements observed by Week 6.

Functional Brain Age Trajectory

Functional brain age decreased from approximately 80 years at baseline to 72 by Week 6, corresponding to a sustained reduction of 8 functional years relative to baseline.

Summary of Case Presentations. Across all Alzheimer's disease cases, baseline qEEG profiles demonstrated electrophysiological patterns consistent with accelerated functional brain aging. Longitudinal qEEG assessments revealed heterogeneous but consistent directional improvements in functional brain age, driven primarily by improvements in posterior alpha organization and ERP processing speed. The variability in response magnitude and timing highlights inter-individual differences in electrophysiological plasticity and underscores the importance of longitudinal within-subject analysis.

Table 2: Summary of Functional (Func) Brain Age Across Alzheimer's Disease Cases

Patient ID	Chron. Age	Baseline Func-Brain Age	Week 2 Func-Brain Age	Week 4 Func-Brain Age	Week 6 Func-Brain Age	Reduction of Func-Brain Age
AD-128	64	74	70	68	—	6
AD-110	70	79	76	77	—	3
AD-121	60	62	57	—	—	5
AD-123	79	88	87	83	—	5
AD-126	75	80	76	74	72	8

Taken together, this case series demonstrates that qEEG-derived functional brain age can exhibit measurable, short-interval changes in patients with Alzheimer's disease during biophoton therapy exposure. Improvements were observed across multiple electrophysiological domains and were detectable within 2 to 6 weeks, supporting the sensitivity of functional brain age as a longitudinal neurophysiological biomarker.

Discussion

In this multi-case series of patients with Alzheimer's disease, longitudinal quantitative EEG analysis revealed measurable short-interval changes in electrophysiological markers associated with functional brain aging during biophoton therapy exposure. Across cases, improvements were most consistently observed in posterior alpha rhythm organization and Event-Related Potential (ERP) latencies related to attention and working memory, corresponding to reductions in EEG-derived functional brain age over periods of weeks.

EEG abnormalities such as posterior alpha slowing, impaired vigilance regulation, and delayed P300/P3b components are well-established features of Alzheimer's disease and have been shown to correlate with disease severity and cognitive decline. Prior studies have demonstrated that reductions in alpha power and frequency, together with prolongation of ERP latencies, reflect decreased neural processing efficiency and disrupted large-scale network integration in AD. The baseline electrophysiological profiles observed across cases in this series are therefore consistent with accelerated functional brain aging relative to chronological age.

Functional brain age integrates multiple EEG-derived features into a composite index reflecting neural network efficiency rather than structural degeneration alone. Unlike chronological age, functional brain age can change dynamically and capture short-interval neurophysiological fluctuations. In the present series, reductions in functional brain age were primarily driven by normalization of posterior alpha rhythms and shortening of ERP latencies, particularly those associated with attentional processing (P3) and working memory (P3b). These findings align with prior work demonstrating that ERP latency measures are sensitive markers of cognitive aging and synaptic efficiency in both normal aging and Alzheimer's disease.

Notably, the majority of functional brain age reduction occurred within the first two weeks of observation in several cases, with continued stabilization or modest improvement through later timepoints. This temporal pattern is consistent with previous reports indicating that electrophysiological measures, particularly EEG spectral and ERP parameters, may respond more rapidly to neuromodulator influences than clinical cognitive scales. The magnitude of change observed in several patients exceeded typical short-term test–retest variability reported for qEEG and ERP measures, suggesting that the longitudinal trends are unlikely to be attributable solely to measurement noise.

Biophoton therapy is a non-pharmacologic intervention that delivers non-thermal photon emissions within biologically relevant wavelength ranges. Previous clinical and observational studies

have reported improvements in cognitive performance, cerebral blood circulation, and EEG measures following 4-week biophoton therapy interventions. Although the precise biological mechanisms remain under investigation, experimental and theoretical work suggests that biophoton-based interventions may influence cellular energy metabolism, mitochondrial function, and neural network coherence, processes that are closely linked to electrophysiological aging and cognitive efficiency. The electrophysiological changes observed in this case series are directionally consistent with these prior reports.

Prior studies have emphasized the utility of EEG-based biomarkers for monitoring disease progression and treatment-related changes in Alzheimer's disease, particularly in early or short-duration interventions. EEG was selected as the primary assessment modality due to its sensitivity to functional neural dynamics, high temporal resolution, and suitability for repeated short-interval measurements, which are less feasible with structural or molecular neuroimaging techniques.

Importantly, this five-case study does not establish causality between biophoton therapy and the observed electrophysiological changes. As an observational case series without randomization or sham controls in a larger Alzheimer's disease cohort, the findings should be interpreted as associative and hypothesis-generating. Nevertheless, reductions in EEG-derived functional brain age were consistently observed and are concordant with prior reports of clinical cognitive improvement, EEG-based measures of cognitive enhancement, and improvements in blood rheology following biophoton therapy [17–20]. Functional brain age thus represents a quantitative index of neural processing efficiency that may complement conventional clinical and imaging-based assessments.

In summary, this case series demonstrates that EEG-derived functional brain age is a sensitive and responsive surrogate biomarker capable of capturing short-interval electrophysiological changes in patients with Alzheimer's disease. While the heterogeneity in response magnitude and timing across individuals highlights the complexity of neurodegenerative processes, the consistent directionality of electrophysiological improvement supports further controlled investigation. Future randomized and sham-controlled studies will be required to determine reproducibility, durability, and clinical relevance, as well as to clarify the relationship between functional brain age changes and long-term cognitive outcomes [21,22].

Limitations

This study is limited by its observational case-series design, the absence of randomized or sham-controlled comparisons, and reliance on EEG-derived functional brain age as a surrogate electrophysiological biomarker rather than direct clinical outcome measures. The small number of cases and heterogeneity in follow-up duration further limit generalizability. Accordingly, the findings should be interpreted as hypothesis-generating, and confirmation in larger, well-controlled cohorts will be required to assess reproducibility, durability of effects, and their relationship to clinical outcomes.

Conclusion

In this case series of patients with Alzheimer's disease, longitudinal quantitative EEG analysis demonstrated measurable, short-interval changes in electrophysiological markers associated with functional brain aging during biophoton therapy exposure. Across cases, improvements were most consistently observed in posterior alpha rhythm organization and event-related potential latencies related to attention and working memory, corresponding to reductions in EEG-derived functional brain age from 3 to 8 years over periods of 2 to 6 weeks.

Although the present observations do not establish causality or clinical efficacy, the findings indicate that EEG-derived functional brain age is a sensitive and responsive surrogate biomarker capable of capturing neurophysiological changes in Alzheimer's disease over short timeframes. The heterogeneity in response magnitude and timing observed across individuals underscores the importance of longitudinal within-subject assessment and highlights the need for larger, controlled studies to determine reproducibility, durability, and clinical relevance.

Taken together, these results support further investigation of EEG-based functional brain age as a quantitative outcome measure for evaluating non-pharmacologic interventions in neurodegenerative conditions.

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