**Optimization of EEG Montages for Detection of Neurodegenerative Disorders**

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**Abstract** Electroencephalography (EEG) offers a non-invasive, cost-effective approach to detect neurophysiological biomarkers in neurodegenerative disorders. This study introduces a data-driven methodology to identify disease-specific EEG signatures and design minimal montages to identify neurodegenerative disorders like Alzheimer’s disease, Parkinson’s Disease etc. Using Alzheimer’s Disease (AD) and Frontotemporal Dementia (FTD), we propose a method to identify scalp region and frequency-specific disruptions across neurodegenerative disorders. Resting-state EEG data from AD, FTD, and healthy aging participants or control group were analyzed across different frequency bands. The analysis revealed distinct patterns across the three groups. In AD, significant delta/theta activity around the F7 electrode reflects temporal lobe differences, while posterior alpha/beta hyperactivity suggests compensatory mechanisms, with minimal gamma activity indicating synaptic dysfunction. FTD showed no delta/theta changes with respect to controls but exhibited increased alpha activity. Frontal delta/theta differences distinguished their spatial profiles while comparing AD and FTD. This study develops a step-by-step method, including statistical validation, to find the best EEG montage for identifying neurodegenerative disorders. Our work shows that analyzing brainwave frequencies on the scalp is a useful step in this process, although we still need better understanding the FTD patterns and gamma wave interpretations. The methodology’s scalability supports deployment in resource-limited settings and integration with wearable technologies, advancing EEG as a practical tool for precision neurology.

**INTRODUCTION**

Neurodegenerative disorders, marked by progressive neuronal loss and pathological protein aggregation (e.g., amyloid-beta, tau, alpha-synuclein), pose a mounting public health threat, accounting for over 60% of dementia cases globally [1]. With aging populations and annual costs surpassing $2 trillion [2], diseases like Alzheimer’s (AD), Frontotemporal Dementia (FTD), Parkinson’s, and Lewy body dementia manifest as overlapping syndromes of cognitive, motor, and behavioral decline. Current diagnostics like Positron emission tomography (PET) and cerebrospinal fluid (CSF) biomarkers remain invasive, costly, and inaccessible in resource-limited settings [3], underscoring the need for scalable, non-invasive tools.

AD, the most prevalent dementia (55 million cases, projected $1.3 trillion cost by 2050) [4], is characterized by amyloid-beta plaques and tau tangles, often diagnosed late due to insidious neuronal loss [5]. Similarly, FTD, affecting younger cohorts (45–65 years), disrupts behavior and language via frontal/temporal degeneration [6].

EEG offers a portable, cost-effective alternative, capturing AD-associated spectral disruptions (e.g., elevated delta/theta, reduced beta/gamma coherence) [7]. Symmetrical EEG activation, though not dementia-specific, shows deviations in AD, including reduced alpha synchrony [8]. Computational advances (machine learning, graph theory) enhance EEG’s diagnostic potential, yet clinical adoption is hindered by methodological heterogeneity and artifact sensitivity. Channel optimization—reducing electrodes while preserving accuracy—could address these limitations by focusing on AD-vulnerable regions (temporoparietal hubs, default mode network) [9].

This study identifies optimal EEG biomarkers through parametric analysis of spectral power and functional connectivity in resting-state AD vs. healthy cohorts, using EEGLAB/MATLAB for standardized preprocessing. Unlike prior work, we integrate frequency-band and spatial analyses within a unified framework, prioritizing neurobiologically relevant electrodes to design a minimal montage. This approach balances interpretability and clinical feasibility, enabling scalable, low-cost AD detection—particularly in resource-limited settings—and fostering wearable EEG integration for remote monitoring. By standardizing EEG protocols, this work advances dementia care democratization and paves the way for similar optimizations in Parkinson’s and Lewy body dementia [10].

**LITERATURE REVIEW**

**EEG Biomarkers in Alzheimer’s Disease**

Electroencephalogram (EEG) biomarkers are emerging as a revolutionary frontier in the fight against AD, offering a dynamic, non-invasive lens into the brain’s deteriorating neural symphony. Unlike costly or invasive imaging techniques, EEG captures real-time electrical activity with millisecond precision, revealing subtle disruptions long before overt cognitive symptoms manifest. The implications are profound. For clinicians, EEG biomarkers could enable earlier diagnosis during the critical window of neuroplasticity, personalize therapies based on a patient’s unique spectral profile, and objectively gauge drug efficacy in trials. For researchers, they illuminate how amyloid plaques and tau tangles hijack neural networks, offering targets for neuromodulation therapies. And for patients, they promise a future where AD is intercepted not at the cliff’s edge of dementia, but in the faint tremors of a theta wave—a future where EEG doesn’t just diagnose disease but helps defy it. Table 1 shows the current EEG biomarkers in recent years.

**Table 1.** EEG Biomarkers in Alzheimer’s disease

|  |  |  |  |
| --- | --- | --- | --- |
| **Frequency Band** | **Change** | **Stage** | **Clinical Implication** |
| Theta (4–8 Hz) | Increased | Preclinical, Progressive | Early dysfunction, memory decline [11] |
| Alpha2 (10–13 Hz) | Decreased | Progressive | Linked to memory/executive loss [12] |
| Alpha (8–13 Hz) | Decreased | Progressive | Neural desynchronization [13] |
| Beta (13–30 Hz) | Decreased | Progressive | Cognitive/motor decline [14] |
| Delta (1–4 Hz) | Increased | Advanced | Cortical dysfunction in wakefulness [12] |

**Affected Scalp Regions and EEG Channel Montages in Alzheimer’s Studies**

AD selectively targets parietal, occipital, and frontal regions—critical hubs for cognition—disrupting spectral dynamics and functional connectivity. Parietal degradation manifests as delta/theta surges overwhelming alpha/beta rhythms, eroding attention and spatial processing [15]. Occipital alpha suppression mirrors visual-perceptual decline [16], while frontal theta invasions destabilize executive functions [17]. These asymmetrical disruptions, particularly in left temporal-parietal areas and the default mode network, create EEG biomarkers of synaptic collapse [18]. EEG montages—strategic electrode arrangements—balance resolution and practicality in capturing these regional vulnerabilities. High-density arrays resolve subtle anomalies, while sparse configurations enhance clinical feasibility [19]. Studies increasingly adopt adaptive montages, prioritizing occipital-frontal-parietal electrodes to align with AD’s spatial trajectory (Table 2). For instance, 10–20 system variants (16–32 channels) optimize detection of parietal-occipital slowing and frontal coherence loss [20]. Emerging approaches propose dynamic montages that shift focus as AD progresses, coupling machine learning with neuroanatomical specificity.

**Statistical and Spectral Analysis for EEG in AD Research**

Robust statistical methods are essential for group EEG analysis in Alzheimer’s Disease (AD) due to noise, non-normality, and high dimensionality. Parametric tests (e.g., t-tests, ANOVA) are effective under assumptions, while non-parametric alternatives (e.g., permutation tests, Wilcoxon) and bootstrapping offer flexibility and reliability [25]. Table 3 lists recent AD EEG studies employing statistical analysis. Parametric spectral methods (e.g., theta/alpha ratios [21]) also provide reliable biomarkers. Spectral analysis through power spectral density (PSD) decomposes EEG into frequency bands (delta–gamma) using FFT [26]. This technique enables detection of spectral slowing in AD—marked by elevated delta/theta and reduced alpha/beta power [27]. Table 4 summarizes the recent EEG studies using power spectrum features and statistical methods in AD.

**Table 2.** Recent AD Studies: Affected scalp regions and EEG montages

|  |  |  |  |
| --- | --- | --- | --- |
| **Paper Title** | **Year** | **Scalp Region** | **Montage** |
| EEG Biomarkers in Alzheimer’s and Prodromal Alzheimer’s: Spectral and Connectivity Features [21] | 2024 | Frontal, parietal, temporal | 10–20 (32 Chan) |
| Identification of Alzheimer’s Disease Brain Networks Using EEG Phase Synchronization [20] | 2025 | Parietal-occipital | 10–20 (19 Chan) |
| Optimizing EEG Electrode Configurations for MCI Detection [22] | 2025 | Occipital | 10–20 (32 Chan) |
| Diagnosis of Alzheimer’s disease via resting-state EEG [23] | 2022 | Frontal, occipital | 10–20 (16 Chan) |
| A Novel CNN-Based Framework for Alzheimer’s Disease Detection Using EEG [24] | 2025 | Temporal, occipital | 10–20 (19 Chan) |

**Table 3.** Recent studies on Statistical Method for EEG in AD research

|  |  |  |
| --- | --- | --- |
| **Author(s)** | **Statistical Method** | **Reference** |
| Xia et. al. (2023) | Non-parametric (DL-based) | [28] |
| Liu et al. (2025) | Non-parametric (DL-based) | [29] |
| Chetty et al. (2024) | Parametric | [21] |
| Cao et al. (2025) | Non-parametric (DL-based) | [20] |
| Pirrone et al. (2022) | Parametric | [30] |
| Kim et al. (2024) | Non-parametric (ML-based) | [31] |

Table 4. Recent studies on Spectral Analysis for EEG in AD research

|  |  |  |
| --- | --- | --- |
| **Author(s)** | **Key Features** | **Reference** |
| Chetty et al. (2024) | Theta/alpha, theta/beta ratios, connectivity | [21] |
| Li et al. (2024 | Beta-band multitaper PSD | [32] |
| Lee et al. (2023) | PSD + brain impedance (rEEG) | [33] |
| Benwell et al. (2020) | (α+β)/(δ+θ) ratio | [34] |
| Kim et al. (2024) | PSD during cognitive tasks | [31] |
| Pirrone et al. (2022) | FIR-filtered delta-to-beta power | [30] |

**METHODOLOGY**

**Dataset and Preprocessing Pipeline**

This study employs a publicly accessible EEG dataset [35], comprising resting-state recordings obtained with participants’ eyes closed. The cohort includes eighty-eight individuals: thirty-six diagnosed with Alzheimer’s disease (AD group), twenty-three with Frontotemporal Dementia (FTD group), and twenty-nine neurologically healthy controls (HC group). Signals were acquired at a sampling frequency of 500 Hz with a resolution of 10 µV/mm, utilizing both anterior-posterior bipolar and referential montages (Cz reference).

Preprocessing commenced with the application of a Butterworth band-pass filter (0.5–45 Hz) and re-referencing of the signals to the linked mastoids (A1-A2). Subsequent artifact mitigation employed the Artifact Subspace Reconstruction (ASR) algorithm, implemented within the EEGLAB MATLAB toolbox. This step eliminated data segments exceeding a conservative artifact threshold (standard deviation > 17 within 0.5-second windows). Following this, Independent Component Analysis (ICA) was executed using the RunICA algorithm, decomposing the original 19 EEG channels into 19 independent components. Components automatically classified as ocular or muscular artifacts (specifically "eye" or "jaw" artifacts) by EEGLAB’s ICLabel routine were discarded.

Comprehensive descriptions of the dataset and the full preprocessing methodology are documented in [36] [35].

**Method**

The proposed methodology employs a systematic, multi-stage pipeline to analyze resting-state EEG data for AD detection, integrating signal processing, artifact mitigation, and data-driven feature optimization. This methodology has been previously employed to investigate eye movement dynamics during reading and eye-tracking data [37]. We have utilized a similar method but for continuous resting state data. This approach starts with collecting raw EEG data, the process involves preprocessing to clean the data. Preprocessing includes filtering to remove unwanted noise (e.g. electrical interference), artifact rejection to discard non-brain signals (e.g., eye blinks), and dimensionality reduction to simplify complex datasets. Independent Component Analysis (ICA) is used to isolate distinct neural signals (e.g., separating visual processing from distractions). The cleaned data can be analyzed across three domains which are time domain (tracking rapid signal changes over milliseconds), frequency domain (identifying rhythmic brain-wave patterns), and time-frequency domain (combining both to study dynamic responses). For group studies, power spectrum is selected as the feature to extract meaningful patterns, followed by statistical model selection and parameter tuning to ensure accurate hypothesis testing. The final analysis interprets results to link brain activity with specific behaviors, providing insights into processes like reading comprehension. This approach balances technical rigor with accessibility, transforming raw data into clear, reproducible findings about brain function.

**RESULTS ANALYSIS AND DISCUSSION**

To investigate neurophysiological differences among AD, Frontotemporal Dementia (FTD), and healthy aging control group, three groups were compared: Control vs AD, Control vs FTD, and AD vs FTD. Power spectral density (PSD) features were precomputed across five bands: delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), beta (13–30 Hz), and gamma (30–45 Hz). Parametric tests (t-tests, ANOVA) assessed group differences (p < 0.05). with exact p-values and effect sizes reported.

In Table 5 and 6, the head outline shows standard EEG electrode positions.

**Table 5.** Statistical analysis of delta (0.5-4 Hz) and theta (4-8 Hz) band across three comparison groups: Control vs AD, Control vs FTD, and AD vs FTD

|  |  |  |  |
| --- | --- | --- | --- |
| **Frequency range (Hz)** | **Control vs AD (p<0.05)** | **Control vs FTD (p<0.05)** | **AD vs FTD**  **(p<0.05)** |
| 0.5-4 Hz |  |  | A diagram of a group  AI-generated content may be incorrect. |
| 4-8 Hz |  | A diagram of a group  AI-generated content may be incorrect. | A diagram of a param  AI-generated content may be incorrect.  A diagram of a group  AI-generated content may be incorrect. |

Red dots mark electrodes with statistically significant differences (p < 0.05), and black dots indicate non-significant ones. The topographical maps spatially represent p-values across the scalp. The color scale (yellow to red) denotes p-values from 1 to 0.001; yellow/green indicates higher p-values (less significance), red shows lower p-values (greater significance), with the color bar providing exact references. Contour lines emphasize regions of significant changes. Group-wise comparisons of EEG spectral power across different frequency bands and electrode sites were performed using two-tailed unpaired t-tests, comparing each disease group (AD and FTD) against healthy controls. The unpaired t-test was chosen due to its suitability for comparing the means of two independent groups when the data distribution is approximately normal. Significant delta and theta activity at F7 (left frontal-temporal) in AD patients aligns with early temporal lobe damage and may reflect neuronal loss. Strong alpha and beta activity in posterior electrodes (T5, P3, Pz, P4, T6, O1, O2) suggests compensatory or residual function in visual-spatial areas. Minimal gamma activity supports known high-frequency network disruptions in AD due to amyloid pathology. No significant delta/theta differences in Control vs FTD contrast with AD, reflecting FTD’s frontal-temporal behavioral focus. Unexpected widespread alpha in FTD (frontal: Fp2, F3, Fz, F4, F8; posterior: T5, P3, O1, O2) may reflect early-stage preservation or subtype variability. Posterior beta significance in FTD warrants further analysis to rule out artifacts or muscle noise. Frontal delta/theta dominance in AD vs FTD reflects differing spatial degeneration (AD: posterior; FTD: frontal). Lack of alpha/beta/gamma differences between AD and FTD suggests limited discriminative value of high-frequency bands alone.

Interestingly, the absence of significant delta/theta slowing in FTD compared to control aligns with known neuropathological distinctions. Unlike AD, which shows early involvement of the posterior cortex and widespread synaptic loss, FTD primarily affects the frontal and anterior temporal lobes. These regions may not exhibit the same electrophysiological slowing, particularly in early stages. This suggests that EEG-based detection of FTD may rely more on changes in alpha activity or frontal network connectivity rather than traditional delta/theta markers. These findings highlight the need for disease-specific biomarker strategies rather than a one-size-fits-all EEG approach.

Symmetrical activation in delta, beta, and gamma across groups challenges expected asymmetry in neurodegeneration. This may reflect bilateral compensatory mechanisms, diffuse pathology, or resting-state conditions. It contrasts with posterior alpha asymmetry in AD, associated with cognitive decline. Though not diagnostically specific, symmetry may inform disease staging, with asymmetry possibly emerging in later stages. Frequency-resolved analysis remains critical for identifying adaptive versus pathological EEG patterns.

An optimized 12-channel EEG montage enhances AD and FTD detection by focusing on key spectral-spatial patterns. Frontal-temporal electrodes (F7, F3, F4, Fz) capture AD slow-wave and FTD frontal anomalies. Posterior sites (T5, T6, P3, Pz, P4, O1, O2) identify AD posterior alpha/beta hyperactivity and FTD beta changes. Fp2 targets FTD frontal alpha activity. This selection emphasizes AD biomarkers and FTD network disruptions, with bilateral posterior coverage for robustness. It improves clinical feasibility, computational efficiency, and diagnostic power, offering a scalable EEG-based screening method. Validation in independent datasets and connectivity integration is recommended for further assessment.

**Table 6.** Statistical analysis of frequency ranges alpha (8-12 Hz), beta (13-30 Hz) and gamma (30-45 Hz) across three comparison

|  |  |  |  |
| --- | --- | --- | --- |
| **Frequency range (Hz)** | **Control vs AD (p<0.05)** | **Control vs FTD (p<0.05)** | **AD vs FTD**  **(p<0.05)** |
| 8-12 Hz | A diagram of a param  AI-generated content may be incorrect.  A diagram of a group  AI-generated content may be incorrect. | A diagram of a param  AI-generated content may be incorrect.  A diagram of a group  AI-generated content may be incorrect. | A drawing of a circular object  AI-generated content may be incorrect.  A diagram of a group  AI-generated content may be incorrect. |
| 13-30 Hz | A diagram of a param  AI-generated content may be incorrect.  A diagram of a param  AI-generated content may be incorrect. | A diagram of a param  AI-generated content may be incorrect.  A diagram of a group  AI-generated content may be incorrect. | A circular object with black lines  AI-generated content may be incorrect.  A diagram of a group  AI-generated content may be incorrect. |
| 30-45 Hz | A drawing of a circular object  AI-generated content may be incorrect.  A diagram of a group  AI-generated content may be incorrect. | A drawing of a circular object  AI-generated content may be incorrect.  A diagram of a group  AI-generated content may be incorrect. | A drawing of a circular object  AI-generated content may be incorrect.  A diagram of a group  AI-generated content may be incorrect. |

While the 12-channel montage offers practical advantages—faster setup, lower cost, and greater portability—it may also limit the spatial resolution and sensitivity to detect subtle or diffuse abnormalities, particularly in advanced disease stages or atypical variants. For instance, network-level disruptions or hemispheric asymmetries may go undetected with a sparse montage. Future iterations of this approach may consider adaptive montages based on patient phenotype or combine the 12-channel setup with targeted high-density.

**CONCLUSION**

This study identified EEG channels and scalp regions with significant frequency alterations in Alzheimer’s Disease (AD), guiding the design of a clinically feasible minimal-channel montage. Parametric analysis of resting-state EEG revealed spectral changes in AD, including increased delta in temporal and beta disruptions in occipital areas aligning with AD pathology like posterior degeneration and cholinergic dysfunction. The proposed 12-channel montage, focusing on left frontal-temporal, frontal midline, and posterior regions, balances diagnostic accuracy for AD and FTD with clinical practicality. This approach reduces electrode count, improving EEG accessibility. Spectral-spatial analysis showed AD’s posterior alpha/beta hyperactivity and temporal delta/theta, while FTD exhibited frontal alpha/theta anomalies, supporting the montage’s discriminative power. Future research should validate this montage in larger cohorts to confirm diagnostic performance and monitor progression. The observed symmetrical EEG activity across delta, beta, and gamma bands warrants exploration, potentially reflecting disease stage or compensatory mechanisms. Incorporating connectivity measures (e.g., coherence, phase-locking) may clarify functional network disruptions. Extending this approach to other neurodegenerative disorders could enhance EEG-based diagnostics and enable personalized dementia care.

Future research should aim to validate the proposed montage across larger, demographically diverse populations, accounting for variables such as age, sex, comorbidities, and disease stage. Stratified analysis could further elucidate how EEG patterns evolve across disease progression or differ in early-onset versus late-onset cases. Such efforts will be crucial to ensure the robustness and generalizability of EEG-based diagnostics across real-world clinical settings.

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