Multi-Dimensional Fractal Vulnerability Study Of Alzheimer's Brain Networks

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***Abstract*—Alzheimer's disease (AD) is associated with widespread disruption of functional brain networks. While graph-theoretic studies have characterized altered connectivity patterns in Alzheimer's disease, most of the analyses rely on single-scale representations and fixed threshold choices, therefore obscuring critical structural transitions. In this study, the multi-scale fractal vulnerability of EEG-derived functional networks in Alzheimer's disease is investigated using a density-controlled graph framework. Resting-state EEG recordings from AD subjects are converted into functional connectivity networks, systematically sparsified across a range of network densities, and analyzed using the box-counting fractal dimension (FD), restricted to the largest connected component (LCC). Fractal structure in AD networks degrades non-monotonically with decreasing density, and a transitional density regime ($\sim 15-25\%$) was identified within the analyzed Alzheimer's disease cohort, corresponding to a regime of heightened structural vulnerability. These findings accentuate the importance of multi-scale analysis for understanding brain network organization.**

***Keywords*—Fractal dimension; graph theory; Alzheimer's disease; EEG; transitional density regime; functional connectivity; human health**

I. Introduction

Complex systems across different fields, like physics, biology, and neuroscience, often exhibit self-similar organization, where structural patterns recur across multiple spatial scales. Such systems cannot be fully characterized by single-scale descriptors, as their essential properties emerge from interactions involving multiple levels of organization. The human brain is a canonical example of a complex system, portraying hierarchical architecture from neurons to large-scale functional networks. Understanding how this hierarchical organization is preserved or disrupted under pathological conditions requires methods capable of capturing multi-scale structure. Fractal analysis provides a powerful mathematical approach for quantifying self-similarity and scale invariance in complex systems. The concept of fractal dimension extends beyond classical Euclidean measures by describing the efficiency with which a structure occupies its space across different scales [1,2]. In the context of networks, fractal dimensions describe that the number of nodes required to cover the network grows with increasing topological radius, thereby reflecting hierarchical organization. Unlike traditional graph metrics that quantify local or global properties at a fixed scale, fractal measures inherently integrate information across multiple scales.

There have been many recent advances in network science that have demonstrated that real-world networks, including biological and technological systems, exhibit fractal or quasi-fractal properties. Brain networks have been shown to display scale-free and fractal characteristics, reflecting the balance between local specialization and global integration [3,4,5]. These properties are thought to support efficient information processing, robustness to perturbation, and adaptive reconfiguration. Importantly, fractal organization is not a static feature but is highly sensitive to network density and connectivity patterns [6,7,8].

Despite this, most network neuroscience studies rely on single-scale representations, typically obtained by thresholding connectivity matrices at a fixed value or density. Such approaches implicitly assume that the extracted network correctly represents the underlying structure, while different thresholds emphasize various aspects of connectivity [9,10]. Excessive sparsification may fragment the network and conceal meaningful structure, whereas overly dense networks may mask hierarchical organization.

Consequently, single-threshold analyses risk overlooking critical structural transitions that occur across scales [11]. Multi-scale network analysis addresses this limitation by systematically inspecting network properties across a range of thresholds or densities. Within this framework, fractal dimension emerges as a particularly informative measure, as it directly analyzes the extent to which network organization evolves as connectivity is progressively weakened [12,13]. However, the application of fractal analysis to brain networks presents methodological challenges, especially when networks fragment at low densities.

Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized clinically by cognitive decline and neuropathologically by misfolded beta-amyloid proteins, neurofibrillary tangles, and widespread synaptic loss [14,15]. Beyond localized damage, AD fundamentally modifies large-scale patterns of brain connectivity, leading to compromised communication between brain regions. Increasing evidence suggests that AD should be conceptualized not merely as an isolated regional pathology, but as a network disorder, affecting the integrity and organization of functional and structural brain networks [16,17,18].

Network neuroscience approaches have provided valuable insights into the altered topology of AD brain networks. Studies using electroencephalography (EEG), functional magnetic resonance imaging (fMRI), and diffusion imaging have reported reductions in clustering, efficiency, and modular segregation, alongside increased path lengths and disrupted hub structure. These changes reveal a shift away from the optimal balance between segregation and integration that characterizes healthy brain networks. Importantly, such alterations are observed even in early stages of the disease, emphasizing the sensitivity of network-level measures to neurodegeneration.

EEG-based functional networks are particularly well-suited for studying AD, as EEG provides direct measures of neural synchronization with high temporal resolution and is widely accessible in clinical settings. EEG connectivity analyses have revealed reduced long-range synchronization, altered frequency-specific coupling, and breakdown of coordination in AD patients [19]. When comprehended through a graph-theoretic lens, these findings point to progressive loss of network coherence and resilience.

While there have been numerous graph-theoretic metrics that have been applied to AD networks, including degree distribution, clustering coefficient, small-worldness, and efficiency, most of these studies focus on single-scale network representations [20]. Fractal analysis has been proposed as a complementary approach for capturing aspects of network organization that are not accessible through traditional graph metrics. Prior studies have reported altered fractal properties in AD-related brain networks and signals, suggesting reduced hierarchical complexity and self-similarity [21,22]. However, these investigations typically employ fixed thresholds and do not explicitly account for network fragmentation. Consequently, it remains unclear whether observed changes in fractal dimension reflect genuine structural degradation or are artifacts of threshold choice and network disconnection.

When taken together, existing research highlights two critical gaps. First, there is a lack of systematic multi-scale analysis of fractal organization in Alzheimer's disease brain networks. Second, the interplay between fractal structure and network connectivity across sparsification levels remains poorly understood. It is unknown whether fractal organization degrades gradually with decreasing connectivity or whether it collapses abruptly at a characteristic scale.

In this work, a multi-scale fractal vulnerability framework is proposed for EEG-derived functional brain networks in Alzheimer's disease. Resting-state EEG recordings are converted into functional connectivity networks and systematically thresholded across a range of network densities. To ensure mathematical validity, fractal dimension is computed exclusively on the largest connected component, thereby isolating intrinsic structural organization from trivial effects of network fragmentation. By tracking both fractal dimension and connectivity as functions of network density, a critical density is identified at which fractal structure collapses while global connectivity remains intact.

A. Related Work

The use of fractal and complexity-based methods for the study of structural and functional changes occurring in neurodegenerative diseases has grown. Changes in fractal dimension, multifractal properties, entropy measures, and cortical complexity have been previously reported in Alzheimer's disease and indicate a progressive decrease of hierarchical organization and self-similarity [10-15].

Network analyses of EEG have also shown considerable changes in functional connectivity, synchronization patterns, and graph-theoretic parameters in Alzheimer's disease. The following effects of reduced efficiency of the network, disruption of hub organization, change of clustering, and loss of long-range communication have been reported in various EEG studies [21,22].

Despite these developments, most existing studies assess network organization at a single threshold or network density. Such approaches may overlook density-dependent structural transitions and fail to capture network complexity evolution during progressive sparsification.

In this present study, these issues are confronted by proposing a multi-scale fractal vulnerability framework with density control. The proposed method allows for fractal dimension and connectivity to be studied at various densities while systematically exploring the fractal dimension and connectivity from the largest connected component, which is not possible with traditional single-threshold analysis.

In this study, Section II contains methods that have been employed, Section III discusses the source and description of data considered, and Section IV gives a brief interpretation of the results obtained.

II. METHODOLOGY

This section outlines the methodological framework employed to construct and analyze EEG-derived functional brain networks. The proposed approach integrates signal-level processing, graph theoretic modeling, and multi-scale fractal analysis to investigate the evolution of network structure under progressive sparsification

A. EEG Preprocessing and Signal Extraction

Raw EEG signals were loaded channel-wise for each subject. No spatial interpolation or channel removal was performed, making sure that all 19 electrodes contributed equally as network nodes. Representative EEG time-series segments were extracted to confirm signal quality and temporal variability. To reduce non-physiological noise, all the EEG channels were inspected and analyzed in their raw form. By maintaining minimal intervention into the technique of extraction, the extracted signals serve faithfully as the representations of the neural dynamics, thus providing a solid foundation for graph-theoretic analysis. No dedicated artifact-removal procedures were applied, and minimally processed EEG recordings were used to preserve the original network structure.

B. Functional Connectivity Estimation

Functional connectivity between EEG channels was quantified using the Pearson correlation coefficient. For a pair of EEG signals $x_i(t)$ and $x_j(t)$, the Pearson correlation was computed as:

$$r_{ij} = \frac{cov(x_i, x_j)}{\sigma_{x_i}\sigma_{x_j}}$$

This resulted in a 19×19 symmetric connectivity matrix for each subject. Diagonal elements were set to zero to remove self-connections.

C. Density-Based Network Thresholding

To avoid arbitrary threshold selection, functional networks were constructed using density-controlled thresholding. For a given density $d \in \{0.05, 0.10, 0.15, 0.20, 0.25\}$, the top $d \times 100\%$ of absolute correlation values were retained, which yields a binary adjacency matrix with fixed edge density across subjects.

Importantly, varying the density parameter enables unbiased exploration of network organization across multiple levels. Lower density values emphasize a robust network, revealing the backbone, whereas higher density values incorporate fragmented network interactions.

D. Graph Construction and Largest Connected Component

Each threshold adjacency matrix was converted into an undirected graph G = (V,E), where nodes represent EEG electrodes and edges represent retained functional connections.

Because fractal dimension is poorly defined on disconnected graphs, all fractal analyses were performed on the largest connected component (LCC) of each network. The size of the LCC was recorded at each density as a measure of global connectivity.

E. Fractal Dimension Estimation

Fractal dimension (FD) was estimated using a box-counting algorithm adapted for networks. For a given box of radius l, the minimum number of boxes N(l) required to cover the LCC was computed based on shortest-path distances.

Fractal dimension was obtained from the scaling relationship:

$$N(l) \sim l^{-D} \quad (1)$$

where, D is the fractal dimension, estimated as the slope of the linear region in the log-log plot of log N(l) versus log l.

Fractal Dimension is characterized as follows:

$$D_F = \lim_{l \to 0} \frac{log\, N(l)}{log\left(\frac{1}{l}\right)} \quad (2)$$

Only scaling regions exhibiting adequate linearity, quantified by the coefficient of determination (R^2), were considered reliable. When the LCC was too small to support meaningful scaling, FD was recorded as undefined (NaN).

F. Cohort-Level Aggregation

Subject-level results were aggregated across the AD subjects. For each density, the mean and standard deviation of fractal dimension and LCC size were calculated, yielding cohort-level multi-scale vulnerability profiles.

G. Algorithmic Structure

The following pipeline was used in the study:

Step 1: EEG Data Acquisition

Step 2: Signal Loading and Channel Verification

Step 3: Functional Connectivity Computation

Step 4: Density-Controlled Network Thresholding

Step 5: Graph Construction

Step 6: Largest Connected Component Extraction

Step 7: Fractal Dimension Estimation

Step 8: Multi-Scale Vulnerability Assessment

Step 9: Cohort-Level Aggregation and Statistical Summary

H. Implementation Details

All analyses were performed using MATLAB R2024a. Functional connectivity matrices were generated using Pearson correlation, and graph construction and network analysis were conducted using custom MATLAB scripts, and fractal dimension estimation was implemented using a box-counting approach applied to the LCC of each network.

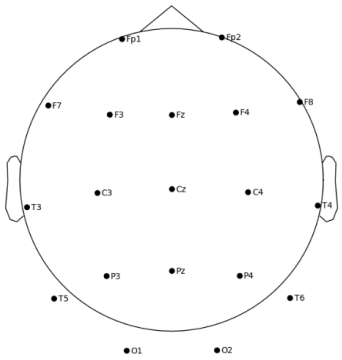

Fig. 1. Scalp electrode configuration used for EEG recordings, following the international 10 – 20 system. Each electrode represents a node in the constructed functional brain network.

Fig. 1 shows the standard international 10-20 system for the placement of electrodes in EEG recordings with 19 electrodes distributed over the surface of the scalp. The electrodes are labeled, and each electrode position is represented as a node for analysis of the functional brain network. The diagram of the circular head shows the location of the electrodes on the head, ranging from frontal (Fp1, Fp2) to occipital (O1, O2) areas. This setup allows an anatomic consistency across all 25 AD subjects, which allows valid cross-subject comparisons. The electrode layout is the base node set that is used for subsequent graph-theoretic and fractal analysis.

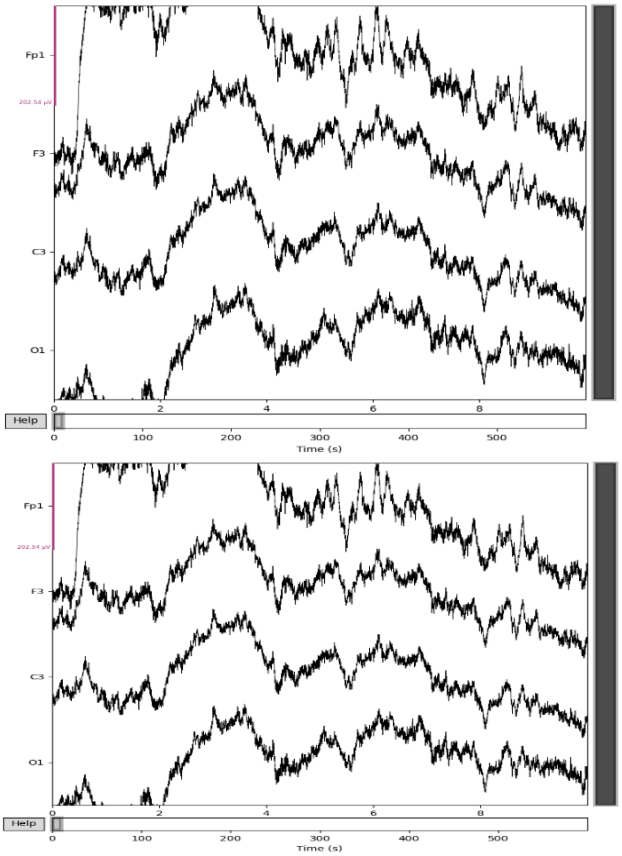

Fig. 2. A representative of EEG time series (10 seconds) from selected scalp electrodes of an Alzheimer's disease subject, illustrating multichannel electrical activity used for functional connectivity estimation.

Fig. 2 shows a 10-second resting state EEG recording from selected electrodes of the scalp in an Alzheimer's disease patient, showing simultaneously recorded multi-channel electrical activity. The traces correspond to different channels of the electrodes, showing the temporal fluctuation of brain electrical signals detected when the eyes are closed while resting. The signals show typical oscillatory, irregular patterns characteristic of neural activity in different regions of the brain. This is a multi-channel representation of raw input data from which functional connectivity matrices were estimated. The complexity and distinctiveness of the different channels indicate the heterogeneity of neural dynamics in AD.

III. Experimental Data Description

The study utilized resting-state EEG recordings from 25 Alzheimer's disease subjects obtained from the OpenNeuro repository (dataset ds004504) [23]. EEG signals were recorded at 500 Hz sampling frequency under eyes-closed resting-state conditions using 19 scalp electrodes positioned according to the international $10-20$ system. All recordings were stored in EEGLAB.set format and processed using an identical computational pipeline.

International $10-20$ electrode placement system (Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T3, T4, T5, T6, Fz, Cz, Pz) is as follows. The same electrode configuration was used for all the subjects, thus ensuring consistency across analyses.

IV. Results and Discussion

This section provides the results of the proposed multi-scale fractal vulnerability analysis applied to EEG-derived Alzheimer's disease brain networks. The evolution of fractal structure and network connectivity as a function of network density is systematically explained, highlighting the identification of critical regimes where structural organization degrades. All results are reported for AD subjects only, with healthy control data used exclusively for qualitative reference in supplementary analyses. Consequently, the identified critical density should be interpreted as a characteristic property of the analyzed Alzheimer's disease cohort rather than a disease-specific biomarker.

Functional brain networks are constructed from EEG recordings by progressively thresholding the connectivity matrices across a predefined range of network densities. This density-based thresholding approach enables controlled sparsification of the network, allowing for a systematic analysis of the response of the network structure to a gradual decline in connections. As network density decreases, the total number of edges is reduced while preserving the strongest functional connections at the global level. At higher densities, networks remain fully connected and exhibit strong topological organization. However, as density decreases, networks begin to fragment, leading to the emergence of disconnected components. To avoid misleading effects arising from trivial disconnection, all subsequent analyses of fractal properties were performed exclusively on the largest connected component (LCC) of the network at each density level.

The size of the LCC decreases progressively with decreasing density. Notably, the reduction in LCC size is gradual rather than abrupt, indicating that topological disintegration occurs progressively rather than through a sudden percolation-like transition.

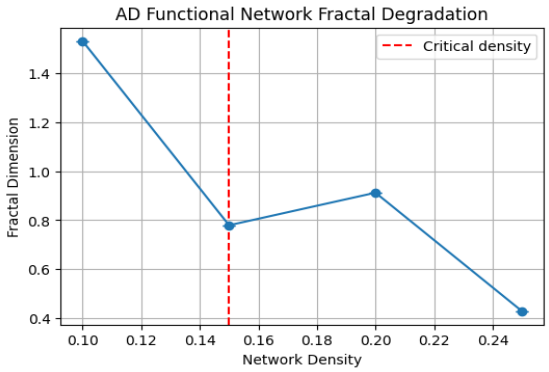

Fig. 3. Cohort-level mean fractal dimension as a function of network density.

Fig. 3 depicts the average fractal dimension of the cohorts as a function of network density $(0.10-0.25)$. The higher the density, the more stable the FD values are, which are in the range of $1.4-1.5$, indicating that the hierarchical organization is preserved. Around the critical density of 0.15, a strong drop is observed, suggestive of an abrupt change in the structure and not a gradual change. At lower densities, FD drops significantly to 0.4, indicating the almost full absence of fractal organization at

low density. The multi-scale analytical framework will be motivated by this non-monotonic, threshold-like behavior.

At higher densities, FD values remain relatively stable, representing preserved self-similar organization across multiple scales. As density decreases, FD begins to decline, reflecting a progressive loss of hierarchical structure and scale invariance.

Importantly, this decline is nonlinear. Rather than decreasing smoothly across all densities, FD exhibits a pronounced drop within a narrow density range. This behavior suggests the existence of a critical density regime where fractal organization becomes unstable.

The core finding of this study is the identification of a transitional density regime (approximately $15-25\%$) at which fractal structure collapses even though the network remains largely connected. This critical density is consistently observed across subjects and is marked by a sharp reduction in FD values. Importantly, this collapse occurs before severe topological fragmentation, as evidenced by the persistence of a substantial LCC at the same density levels. This decoupling between fractal degradation and network disconnection demonstrates that loss of fractal organization is not just merely a consequence of reduced network size but indicates a deeper structural vulnerability.

The critical density identified in this study represents a regime where the network transitions from a complex, multi-scale structure to a simpler, less organized topology. This phenomenon is not captured by conventional single-threshold analyses, underscoring the necessity of a multi-scale framework.

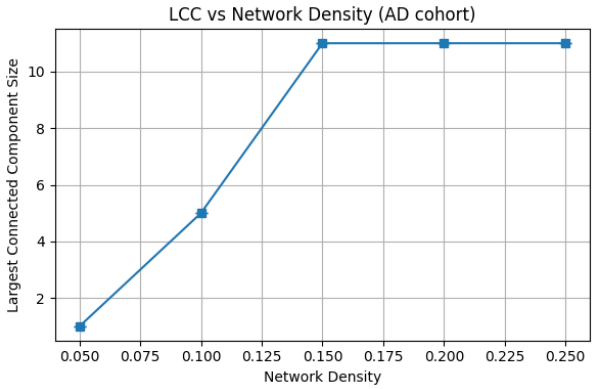

Fig. 4. Size of the largest connected component as a function of density.

Fig. 4 represents the size of the largest connected component plotted as a function of network density for the AD cohort, as shown in Fig. 4, and is found to increase in a monotonically increasing and smooth fashion from density $0.05-0.25$. Unlike the fractal dimension, the size of the LCC increases progressively with no sharp collapse or critical transition, ending at an LCC size of about $11-12$ nodes at a density of $0.15-0.175$. This smooth trajectory is in contrast to the steep FD drop at the same density range, which clearly indicates that connectivity and fractal organization deteriorate at a completely different rate. The finding that there is a difference between the FD and LCC trajectories lends credence to the hypothesis that fractal collapse is an earlier and more sensitive indicator of structural instability.

Unlike fractal dimensions, LCC size increased smoothly with density and did not exhibit abrupt collapse. Prominently, at the density where FD reached its minimum, the LCC remained large and stable across subjects. Progressive fragmentation is observed at lower densities, thus indicating increased network vulnerability.

To further explicate the relationship between structural complexity and connectivity, FD and LCC size were jointly analyzed across a range of densities. Even when both metrics decrease with decreasing density, the rates of decline differ substantially. LCC size decreases gradually across densities, whereas FD exhibits a steeper decline within a narrow range. This divergence indicates that fractal collapse precedes major connectivity loss, highlighting FD as a sensitive early marker of structural instability.

This result supports the hypothesis that fractal measures capture aspects of network organization that are not reflected by purely topological metrics. In the context of AD, this suggests that pathological changes may first manifest as disruptions in hierarchical organization rather than outright disconnection. To assess the robustness of the observed patterns, results were aggregated across all AD subjects by computing the mean and standard deviation of FD and LCC at each density level. The cohort-level trends are summarized in Table I and visualized in Fig. 5. The mean FD curve exhibits a clear inflection point corresponding to the critical density identified at the individual level. The associated standard deviation increases near this region, indicating heightened inter-subject variability during the transition phase. This increased variability suggests that the critical regime represents a structurally unstable state where individual networks diverge in their response to sparsification.

TABLE I. COHORT LEVEL MEAN ± STANDARD DEVIATION OF FRACTAL DIMENSION (FD) AND LARGEST CONNECTED COMPONENT (LCC) ACROSS NETWORK DENSITIES

	Threshold	FD (mean ± SD)	LCC (mean ± SD)
0	0.05	Nan	1.0
1	0.1	1.53	5.0
2	0.15	0.779	11.0
3	0.2	0.912	11.0
4	0.25	0.43	11.0

Table I shows the mean FD and LCC values across five density levels $(0.05-0.25)$ for the AD cohort, where the value of FD is not defined at the lowest density because the LCC size is too small. We observe a critical transition at density 0.15, where FD rapidly changes to 0.779, while LCC remains mostly constant at 11.0 nodes, thus signifying that fractal organization and connectivity break down in different ways.

Substantial FD reduction was observed within the intermediate density regime (approximately $15-25\%$), indicating structural instability across this sparsification range.

Traditional network analyses often rely on metrics computed at a single threshold or density, implicitly assuming stability across scales. To contextualize our findings, it is noted that FD values computed at a fixed density would fail to capture the observed non-monotonic behavior and critical transition. By

contrast, the proposed multi-scale approach reveals continuous evolution of structural complexity and identifies regimes of instability that are invisible to single-scale analyses. This highlights a fundamental limitation of fixed-threshold methods in the study of neurogenerative disorders.

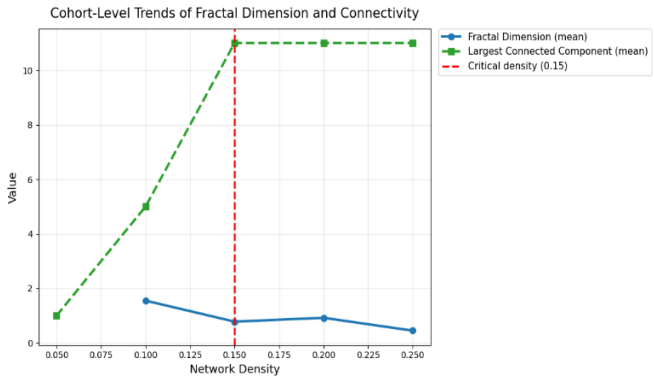

Fig. 5. Cohort-level trends.

Fig. 5 shows a dual-metric plot where the mean fractal dimension and the mean LCC size are plotted over the range of network densities, with the critical density of 0.15 indicated by a red dashed vertical line. The LCC curve increases sharply and levels off; the FD curve is relatively flat at high densities and drops quickly when approaching and reaching the critical density. The distance between the two curves is the largest exactly at the critical density, visually reinforcing that the two measures measure different but complementary aspects of network organization. The key evidence of the main claim of the study, that multi-scale fractal analysis reveals instability regimes that are not captured by single threshold connectivity measures, is the joint visualization.

The observed early collapse of fractal organization in AD networks has important implications. Fractal structure reflects the brain's ability to integrate information across multiple scales. Its degradation suggests a loss of hierarchical coordination that may underlie cognitive dysfunction in AD. By identifying a density regime where this collapse occurs independently of gross disconnection, the proposed framework provides a principled way to detect subtle structural vulnerabilities that precede large-scale network failure.

A. Identification of a Critical Density

The simultaneous observation of 1) a sharp reduction in fractal dimension and 2) preserved global connectivity identifies a critical density ($\sim 15-25\%$) at which structural self-similarity degrades before global network disconnection. This indicates that fractal organization is more sensitive to connectivity loss than conventional topological measures.

This study demonstrates that Alzheimer's disease EEG networks exhibit multi-scale structural vulnerability that cannot be captured by single-threshold analyses. The identification of a critical density at which fractal structure collapses while global connectivity remains intact suggests a distinct regime of pathological instability.

Importantly, the emergence of undefined fractal dimensions at very low densities is not a methodological artifact but a reflection of genuine network fragmentation. By explicitly restricting fractal analysis to the largest connected component, we ensure that observed changes reflect intrinsic network organization rather than trivial disconnection effects.

The proposed framework provides a systematic approach to threshold selection and highlights fractal dimension as a sensitive marker of early structural disintegration. This methodology is extendable to larger datasets and other neurodegenerative conditions.

The present study focuses exclusively on Alzheimer's disease networks and therefore does not establish whether the observed critical density is unique to Alzheimer's disease or reflects a broader property of EEG-derived functional networks. Future work incorporating healthy control cohorts will be necessary to determine disease specificity and evaluate the potential diagnostic relevance of the identified density regime.

V. LIMITATIONS

Several limitations should be acknowledged. First, the study utilized a relatively small Alzheimer's disease cohort obtained from a public EEG repository. Second, healthy control networks were not incorporated into the primary quantitative analysis, limiting conclusions regarding disease specificity. Third, functional connectivity was estimated using Pearson correlation, which captures linear relationships only. Future studies involving larger cohorts, multimodal neuroimaging data, and alternative connectivity measures may provide a more comprehensive characterization of multi-scale network vulnerability. Fourth, the present study employed minimally processed EEG recordings and did not incorporate dedicated artifact-removal procedures; therefore, the influence of residual artifacts cannot be completely excluded.

VI. CONCLUSION

In this study, we introduced a multi-scale fractal vulnerability framework to investigate the structural organization of EEG-derived functional brain networks in Alzheimer's disease from a graph-theoretic perspective. By systematically varying the density of the network and by integrating fractal dimension analysis with connectivity measures, we moved beyond conventional single-threshold approaches and demonstrated that Alzheimer's disease networks exhibit scale-dependent structural instability, which cannot be captured by traditional graph metrics. A core finding in this study is the transitional density regime ($\sim 15-25\%$), at which fractal organization collapses abruptly, even though global connectivity is preserved as measured by the largest connected component. There is a separate distinction between fractal degradation and topological disconnection. It reveals that loss of hierarchical, self-similar organization precedes network disintegration. Subtle and minute organizational breakdowns cannot be detected by pure topological metrics. In conclusion, this work demonstrates that multi-level fractal analysis reveals a distinct and early form of network vulnerability in Alzheimer's disease. By shifting from single-scale, static metrics to multi-scale, dynamic analysis, we have provided a new insight into the concept of structural vulnerability.

Reproducibility Statement

The entire analysis procedure, from EEG loading, functional connectivity estimation, density-controlled thresholding, graph construction, to the extraction of the largest connected component and calculation of fractal dimension, was applied equally among all the subjects. The parameters used and the density levels in the study are all explicitly explained to ensure that the proposed framework can be reproduced.

Data Availability Statement

All the EEG data taken in this study are from the publicly available OpenNeuro repository (dataset ds004504). All the data were fully anonymized before public release, and no personal information was accessible to authors. All the analyses were conducted in accordance with ethical guidelines and regulations involving human data.

Conflict of Interest

The authors declare that there are no commercial or monetary relationships that could be construed as a potential conflict of interest in the conduct of this research.

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