# Causal Analysis of Graph Signals for Brain Effectome Inference

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***Abstract*—Understanding the directed interactions between brain regions is critical for analyzing neuro-degenerative diseases like Alzheimer's (AD). Traditional functional connectivity (FC) methods capture statistical associations but fail to infer causal relationships, limiting their ability to reveal structural disruptions in disease progression. In this exploratory work, we propose a causal graph inference and spectral analysis framework for EEG signals. Specifically, we leverage Granger causality and spectral graph methods to construct and analyze the effective connectome (effectome) of the brain. Our work reveals that AD networks exhibit lower algebraic connectivity ($\lambda_2$), reduced modularity (eigenvalue gaps), and increased structural sensitivity to perturbations, compared to healthy individuals. Additionally, a study of temporal dynamics over the inferred networks shows reduced and local node volatility patterns over time for AD brain networks.**

## I. Introduction

Study of effective connectivity through cause-effect relationships can aid in the study of second-order effects of, say, localized degeneration where a disease affects one region of the brain but its effects are seen elsewhere. Using (causal) interventions, this can aid the study of changes in overall behavior when connectivity between two nodes is disrupted. This motivates the key objective behind this project.

We aim to address the following broad research questions, primarily intended as a framing device for our work:

- **RQ1**: Can we reliably generate a connectivity model of the brain by applying causal methods on EEG time-series data?
- **RQ2**: Can we use spectral graph analysis to identify the differences in properties of the brain network within healthy individuals and patients with a neuro-degenerative disease (specifically, Alzheimer's)?
- **RQ3**: Can we expand this method to other datasets out-of-the-box?

Below is the approach taken to achieve the goals stated above:

- Collect EEG data signals for healthy individuals and patients with Alzheimer's Disease (AD), and preprocess this data to match resolutions and filter higher frequencies
- Perform preliminary analyses on EEG data to study their characteristics
- Use causal modeling to generate an adjacency matrix for brain network - explore the use of Granger Causality and other information-theoretic (IT) measures to capture complex dynamics
- Perform spectral analysis on the constructed graph using its Laplacian & obtain a Jordan decomposition. Analyze the Fiedler eigenvalue to study the algebraic connectivity of the graph and understand the emergence of communities in the network
- Run windowed analysis of EEG signals, and construct a multi-layer network to study temporal dynamics like node and edge volatility
- Use a diffusion kernel to simulate stimulus responses in the network

While the methods are designed to be generalizable, the current work only discusses them in the light of one dataset, thereby constraining the graph size to the number of regions within that data. The architecture, along with the effectomes generated, is shown in Figure 1.

Some of the key findings are given below:

- While each brain structure is unique, distinguishable features exist in the effectome between healthy individuals and AD patients
- Eigengaps and eigenvalues (specifically the Fielder value, $\lambda_2$) obtained through the spectral analysis show the healthy network being more robust to perturbations (changes in structure), and the AD network sensitive
- Temporal dynamics show node and edge volatility patterns which suggest AD brain networks exhibit reduced variability compared to healthy brain networks

## II. Data, Experiments & Results

For this work, we consider two EEG datasets - Vichietti (MV) [1] and Miltiadous (AM) [2]. As part of an ongoing effort, we are also experimenting with custom temporal summation of pain dataset (TSP). The presented results are from tests using the MV dataset for a randomly chosen subject in both the healthy and AD groups. Specifically, the data has 19 nodes, with 1024 samples acquired at resting state with a sampling rate of 128Hz, and total duration 8 seconds.

### A. Preliminaries

Consider $x_n[t]$, a discrete time series on node $v_n$ in graph $G = (V, E, W)$, where $n$ indexes the nodes of the graph and $t$ the time samples. Let $N$ be the total number of nodes and $K$ be the total number of time samples, and $x[t] \in \mathbb{C}^N$ represents

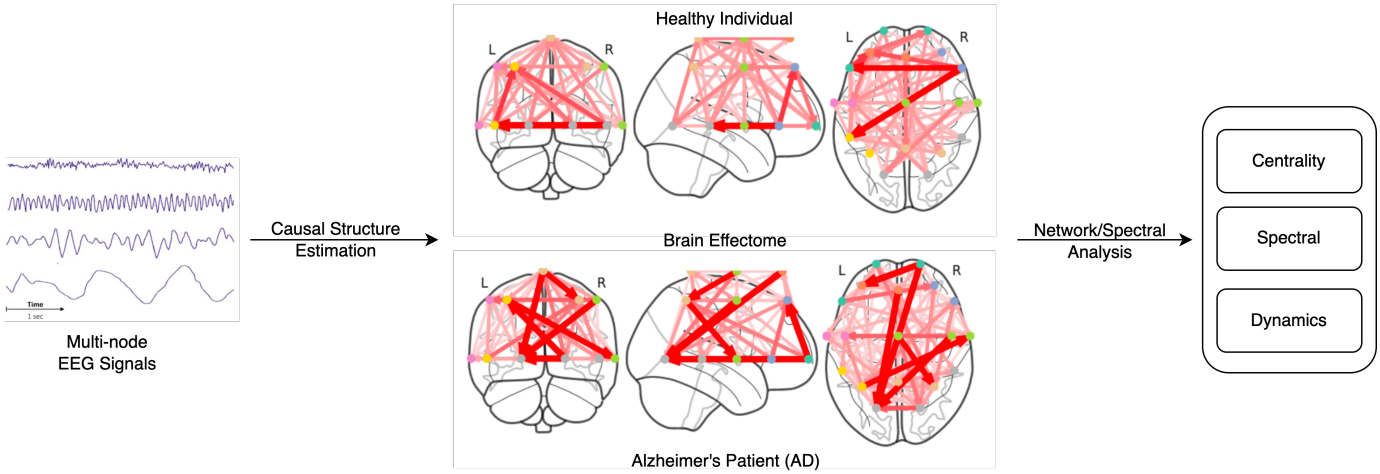


Fig. 1. System Architecture. We estimate the causal structure of the brain from EEG signals, and run analyses to study its properties.

the graph signal at time $t$. We generate the effectome graph $G$, with edges $E$ and weights $W$ determined through statistical tests. Within $W$, $w_{ij}$ represents the weight of the edge between nodes $i$ and $j$.

### B. Network Construction

We construct the effectome weight matrix W through causal inference with Granger causality. The heatmaps are presented in Figure 2. We observe a lack of connectivity from the T6 and T5 regions (posterior temporal and parietal occipital junctions) in AD, which signifies impaired memory retrieval and language comprehension. F8 and Fz have higher connectivity, suggesting possible compensatory mechanisms at play. Generally, while the healthy network is more uniform, we observe stronger causation in AD brain networks, showing increased and disorganized connectivity likely due to pathological processes resulting from the disease (figure 4). These results are also supported by prior works [3]–[5]. As a part of ongoing work, we are looking into other popular information-theoretic measures like Transfer Entropy (TE) [6], [7] and Permutation Entropy [8]. Preliminary results with TE (Figure 3) show sparser AD brain network compared to healthy brain networks.

### C. Network Analysis

We analyze the properties of the constructed graph G by looking at centrality, spectral decomposition, and study its dynamics.

*1) Centrality measures :* Figure 5 shows the centrality measures grouped by major regions. Typical brain activity happens in the posterior regions, but a higher eigenvector centrality in case of AD patients in the frontal region suggests a compensatory mechanism, likely due to loss of function in the posterior regions [3]–[5].

*2) Spectral Analysis:* An eigendecomposition of the Hermitian Laplacian matrix of G allows us to study its response under variations, especially when perturbed. Specifically, we are interested in the Fiedler value (FV) $\lambda_2$.

For the healthy brain network, the eigenvalues are larger, particularly in lower indices, indicating a more well connected and modular network structure. This is supported by $\lambda_2 \approx 0.5$ ($\mu = 0.44, \sigma = 0.23$ across all subjects). On the other hand, for the AD brain network, the eigenvalues are more closely spaced, suggesting reduced connectivity or weakened modularity in the network. This may reflect the loss of functional integration across brain regions. This is supported by $\lambda_2 \approx 0.3$ ($\mu = 0.38, \sigma = 0.13$ across all subjects), indicating a network comparatively more sensitive to perturbations. These findings are also supported through the Jordan decomposition, where the diagonals have a smoother gradient in healthy brain networks compared to the AD brain network. With Transfer Entropy, the $\lambda_2$ values are 0.83 and 0.57 for healthy and AD brains respectively, which remain largely consistent with our prior observations.

*3) Temporal Dynamics:* Multi-layer networks constructed using a rolling-window analysis (window size = 256, step size = 128) enable us to examine edge and node volatility over time. We observe that nodes within the AD brain network exhibit lower volatility compared to those in a healthy brain network, suggesting reduced flexibility in dynamic brain activity in AD patients. This is also consistent with the finding that AD disrupts functional connectivity and reduces variability [9]–[12]. In addition, our analysis of stationarity revealed that healthy EEG recordings were partially non-stationary, with several nodes displaying significant temporal fluctuations, whereas AD signals were predominantly stationary across nodes. The presence of non-stationarity in healthy brains points to richer temporal dynamics and greater functional adaptability, whereas the stationary nature of AD recordings suggests a pathological stabilization and diminished capacity for network reconfiguration [13].

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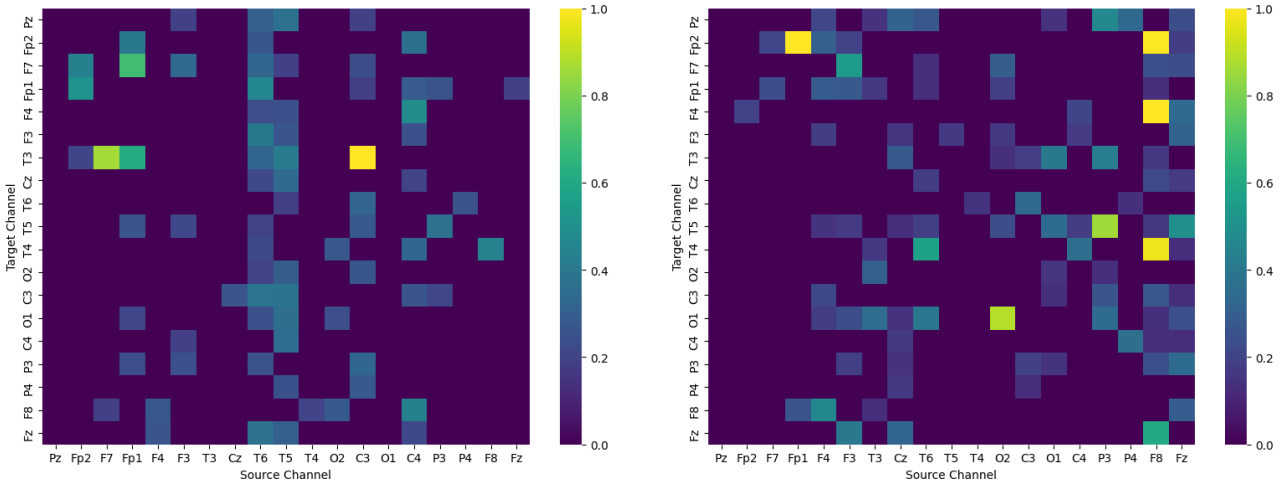
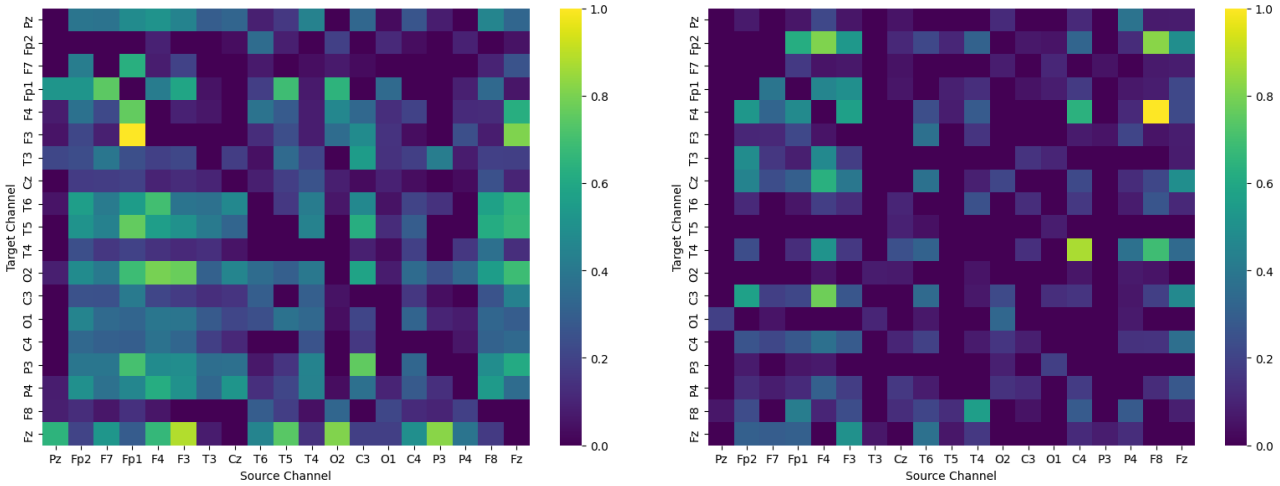


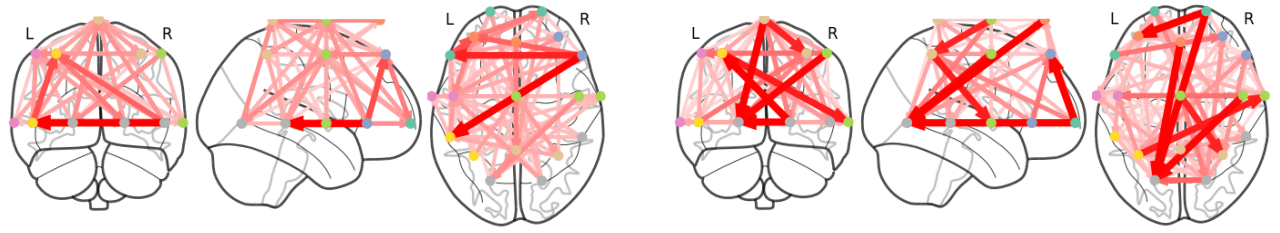

(a) Healthy Individual (b) AD Patient

Fig. 2. Heat map of effective connectome strength, obtained using Granger Causality. We can observe a lack of connectivity from the T6 and T5 regions (posterior temporal and parietal occipital junctions) in AD, which signifies impaired memory retrieval and language comprehension. Interestingly, F8 and Fz have higher connectivity, which shows possible compensatory mechanism at play



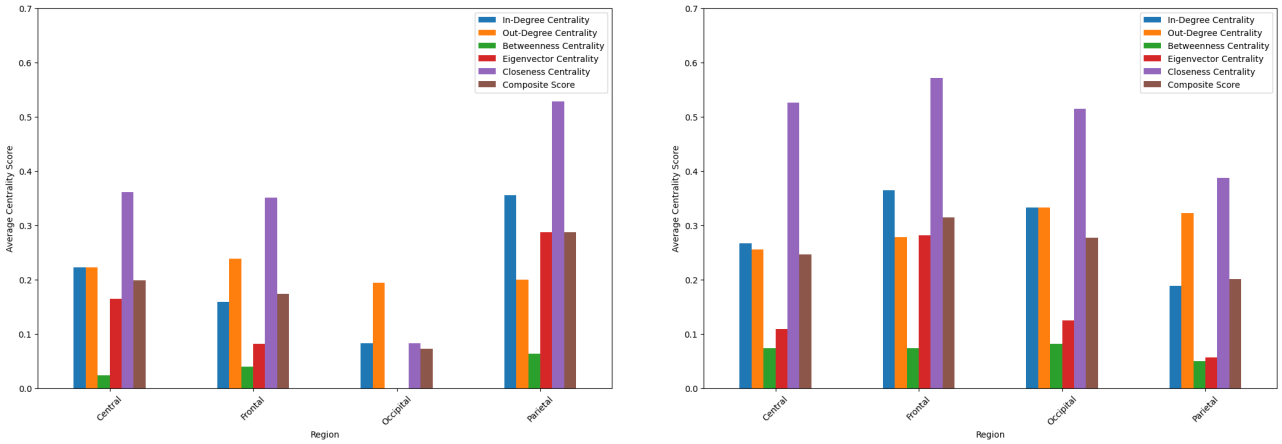

(a) Healthy Individual (b) AD Patient

Fig. 3. Heat map of effective connectome strength, obtained using Transfer Entropy. Each heatmap shows directed information transfer between EEG channels, with sources along the x-axis and targets along the y-axis. Warmer colors indicate stronger information flow. AD brains exhibit sparser and weaker transfer entropy connections compared to healthy brains, suggesting disrupted directed functional connectivity.



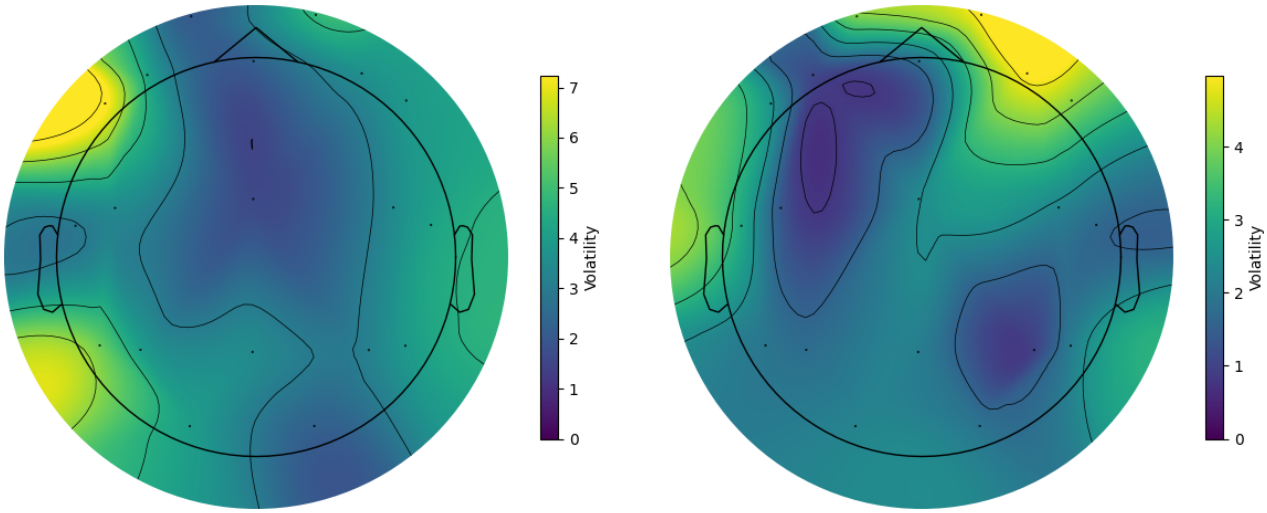

(a) Healthy Individual (b) AD Patient

Fig. 4. Plot of the directed effective connectome, obtained using Granger Causality. Opacity signifies strength of connection, with the most opaque one showing the strongest connection. We observe stronger causation in AD brain networks, showing increased and disorganized connectivity, likely due to the pathological processes resulting from the disease. In contrast, the healthy brain network is more uniform




(a) Healthy Individual (b) AD Patient

Fig. 5. Centrality measures grouped by regions. Typical brain activity happens in the posterior regions, but a higher eigenvector centrality in case of AD patients in the frontal region shows a compensatory mechanism at play, likely due to loss of function in the posterior regions




(a) Healthy Individual (b) AD Patient

Fig. 6. Topographical maps of node volatility across the scalp for (a) healthy individual and (b) AD patient. Node arrangement follows the standard International 10-20 format for EEG acquisition. Volatility is measured as the variance of node activity over time. Healthy individuals exhibit broader and higher volatility levels, while AD patients have it reduced and more spatially localized (note the changes in scale), indicating a disruption in dynamic network stability.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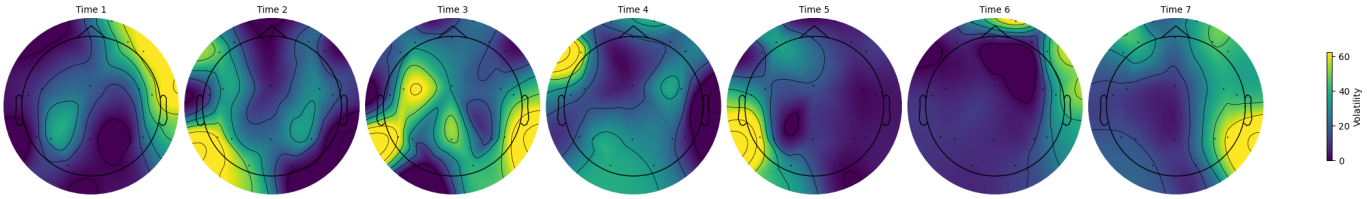
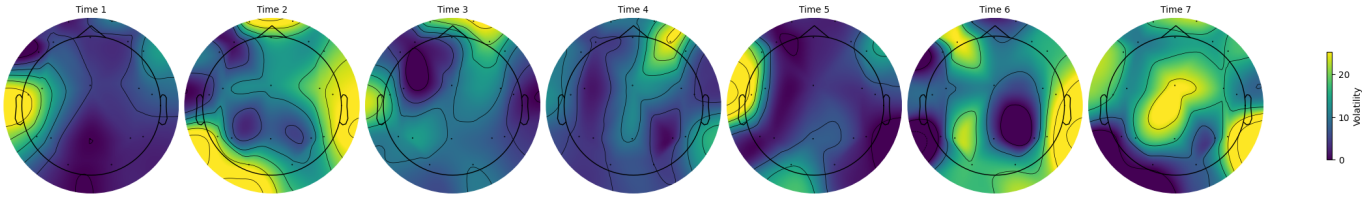



(a) Healthy Individual




(b) AD Patient

Fig. 7. Temporal evolution of node volatility across sliding windows for (a) Healthy individual and (b) AD patient. Each scalp map represents node-wise volatility at a specific time window. Healthy individuals display more spatially diverse and dynamic changes in volatility over time, while AD patients exhibit more local and reduced volatility patterns. As with Figure 6, note the changes in scale between healthy and AD subjects.

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