Causal ECN Estimation for Brain Disorders from EEGs

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***Abstract*—Alzheimer's disease (AD) and Frontotemporal Dementia (FTD) are severe neurodegenerative disorders.**

The main goal of this study is to investigate how the connections between different areas of the brain are affected in subjects suffering from AD and FTD when compared to healthy ones. This is performed by estimating the Effective Connectivity Networks (ECNs) from the causal analysis on the EEG signals.

We apply two different causal methods: Granger causality and LiNGAM.

The results show that ECNs derived from Granger causality highlight a progressive reduction of connectivity from Cognitively Normal (CN) to FTD and, more markedly, to AD subjects, with disruptions involving frontal, pre-frontal, temporal and parietal regions in agreement with known clinical patterns. LiNGAM reveals more subtle differences between groups, mainly reflected in the reduced robustness of causal links rather than in their number. Compared with previous PCMCI-based analyses, the findings confirm the deterioration of global connectivity in AD and frontal involvement in FTD, while also suggesting a reduced activity in parietal regions in AD. Overall, the study highlights the potential and the limitations of different causal discovery approaches for characterizing neurodegenerative alterations in brain connectivity.

***Index Terms*—Alzheimer's disease, frontotemporal dementia, causality, Granger, LiNGAM, EEG**

I. Introduction

Since Alzheimer's Disease (AD) and Frontotemporal Dementia (FTD) are widespread neurodegenerative disorders; researchers have suggested that Effective Connectivity Networks (ECN) could be a very powerful tool both for their analysis and detection. Actually, ECN could show how activity in one brain region causally influences another, allowing to capture the neural changes caused by AD and FTD when comparing their ECNs with the ones coming from Cognitively Normal (CN) subjects [1]. For this purpose, it is also essential to discover the direction of the causation.

AD is caused by the accumulation of abnormal proteins disrupting brain functions [1]. The authors in [2] report that the atrophy caused by AD is mainly located in the medial temporal and parietal cortices. They also provide a deep analysis of the distinctive traits of AD in terms of Functional Connectivity (FC) between brain areas.

The study revealed lower FC in AD subjects compared to healthy controls, in particular within the precuneus and the posterior cingulate cortex (PCC), respectively in the parietal lobe and in the posterior "limbic lobe", and both belonging to the so called Default Mode Network (DMN). Further studies have highlighted a decrease in the connectivity between the precuneus or the PCC and several brain regions, including temporal cortex, hippocampus, prefrontal cortex and thalamus. Hippocampus is located in the temporal lobe and its functions mainly refer to memory, whose loss is a very typical symptom in people suffering from AD.

In addition, two other networks displayed FC changes in AD, namely the right occipital regions of the secondary visual network and the network consisting of basal ganglia and cerebellum. The latter is deeply correlated to the thalamus, which is part of the DMN and, together with basal ganglia and cerebellum, has many cognitive functions that are highly compromised by the progress of the disease, such as learning and planning.

On the other hand subjects affected by FTD show similar traits to AD ones, but this disorder belongs to a group of non-Alzheimer dementias and only in the last decades its complexity and impact has been fully recognized.

FTD is caused by a progressive atrophy involving the frontal and temporal lobes [3]. However, more specifically, FTD can be subdivided into three main clinical syndromes: behavioural variant FTD (bvFTD), semantic dementia (SD) and progressive non-fluent aphasia (PNFA).

The bvFTD is the most common and typically causes abnormal behaviours, it involves the frontal and anterior temporal lobe. It also affects the paralimbic network, which has important cognitive roles and includes anterior cingulate cortex (ACC), the orbitofrontal cortex (OFC), in the pre-frontal cortex, and the frontoinsular cortex (FIC).

SD leads to the decline of semantic memory, i.e. associated with the knowledge of concepts and objects accumulated by the individual's experience of the world. Semantic dementia affects the anterior temporal and inferior frontal network.

Finally, PNFA is characterized by a progressive breakdown in language output with non-fluent speech. Actually it influences the language network and often atrophies the left sided temporo-parietal junction.

It's important to underline that the severity of all these three FTD syndromes (i.e. bvFTD, SD and PNFA) varies widely among individuals.

In [1], the authors estimate ECNs from electroencephalograms (EEGs), i.e. time-series data, employing the PCMCI

(Peter-Clark Momentary Conditional Independence) as the causal discovery method. For their study, the authors selected the dataset in [4], which collects 88 EEGs from three groups of participants: 36 of them were diagnosed with Alzheimer's disease (AD group), 23 were diagnosed with Frontotemporal Dementia (FTD group) and 29 were healthy subjects (CN group). The EEGs were measured with 19 scalp electrodes while participants were in a resting state with closed eyes. The 19 electrodes are coherent with the 10-20 international system (Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, and O2). Specifically, the preprocessed EEGs contained in the dataset were employed.

In this work, we are interested in computing the ECNs as in [1], adopting the same dataset, but applying two different baseline methods for causal analysis: Granger causality [5] and LiNGAM [6].

In Sec. II a brief overview of causal analysis is carried out, especially explaining the two adopted methods, i.e. Granger and LiNGAM; in Sec. III the implementation of the methods is described along with the experimental setup; in Sec. IV the obtained ECNs are shown and analysed, also comparing them with the figures from [1]; finally, in Sec. V the last observations and conclusions are discussed.

II. METHODS

In general, causal inference states that, given two variables, if the value of a variable is changed and that of some other variable also changes, the former is the cause and the latter is the effect. The causal structures between the variables can be represented through the so called causal graph [6].

When dealing with time series, as for EEGs, the considered variables are time-dependent and their influence should be analysed over time. For this reason, also lags should be taken into account, namely the delay between an event and its effect on the data being analysed.

A. *Granger causality*

Granger causality is a statistical approach which tests if past values of one variable improve predictions of another variable; especially X_t Granger-causes Y_t if the behaviour of Y_t is better predicted including past information of X_t rather than Y_t's past information alone.

More specifically, consider the prediction error ϵ as the difference between the real value of the variable and its predicted value; in addition let's denote σ^2 as the variance of ϵ. We say that X_t is causing Y_t if if the following equation holds:

$$\sigma^2(Y|\overline{U}) < \sigma^2(Y|\overline{U-X}) \tag{1}$$

where U is all the information in the universe, $(U-X)$ is the same information excluding X and, given a variable A, $\overline{A}$ represents the set of all past values of the variable itself (i.e. A_{t-j} with $j = 1, 2, ..., \infty$).

An important assumption of the test, is the stationarity of the signals. If any of the series is not stationary, it must first be made stationary, typically using differencing [7].

Granger causality test on time series is performed for each time lag τ_i with $i = 1, ..., \tau_{max}$. The null hypothesis states that the past values of X_t, at a certain lag, do not improve the prediction of Y_t. If the p-value returned by the test is lower than the significance level α, then the null hypothesis could be rejected, indicating the presence of predictive information from X to Y, namely X Granger-causes Y.

B. *LiNGAM*

Causal inference was typically performed with Structural Equation Models (SEM) which most of the times assume, implicitly or explicitly, the Gaussianity of data; however these classical models are often not able to estimate the causal directions between variables.

For this reason, more recently some new methods assuming a non-Gaussian structure of the data were proposed, including LiNGAM (Linear non-Gaussian Acyclic Model).

LiNGAM is an extension of the linear acyclic SEMs, where the goal is still the estimation of the causal strength matrix B between the variables, but the difference is that exogenous variables are assumed to be non-Gaussian and independent of each other (i.e. there are no latent confounding variables). [1] A LiNGAM model in matrix form is described by:

$$x = Bx + e \tag{2}$$

where x represents the n variables x_i with $i \in [0, n-1]$, $B \in \mathbb{R}^{n \times n}$ is the causal strength matrix and e refers to the n exogenous variables e_i with $i \in [0, n-1]$. In particular, we say that x_j (X) causes x_i (Y) if the causal strength $b_{i,j} \in B$ is different from zero. Note that due to the acyclicity, the diagonal elements of B are always zeros.

It is proved that the connection strength matrix B can be uniquely identified based on the data x only.

The basic form of LiNGAM captures instantaneous causality, so when dealing with time series the concept of lagged variables should be introduced. For this purpose an extension of LiNGAM was proposed, combining the classic LiNGAM model with a VAR (Vector Auto-Regressive) one, which accounts for time into its structure. This method is known as VAR-LiNGAM [8]. In this case, $(\tau_{max}+1)$ causal strength matrices are computed, B_0 addresses instantaneous causality and B_τ with $\tau = 1, 2, ..., \tau_{max}$ contains the lagged strengths, up to a maximum lag τ_{max}.

Since a VAR model is applied, the stationarity of the signal is still assumed [9]. If stationarity isn't respected, differencing could be employed.

Furthermore, VAR-LiNGAM doesn't provide directly a way to assess reliability to the results. A common procedure to solve this limitation consists in applying bootstrap [8], which is a resampling technique allowing us to get a probability matrix for each strength matrix B.

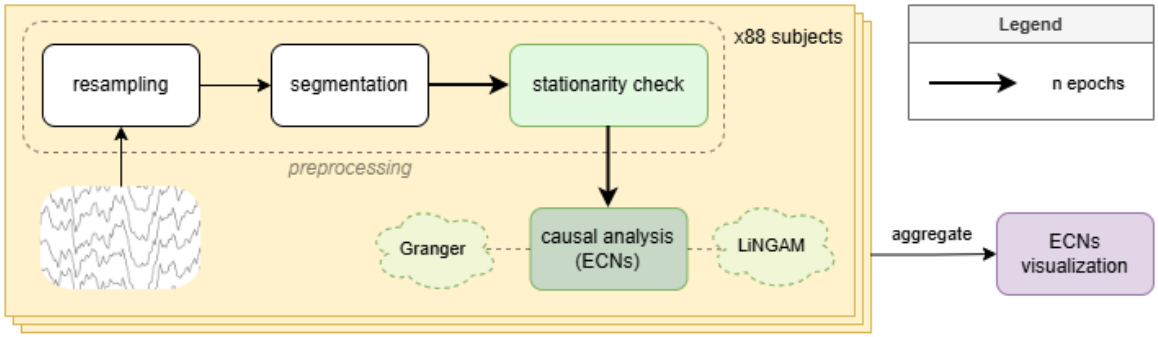


Fig. 1: Block diagram.

III. Implementation

The analysis, performed in python, is depicted in its main blocks in Fig. 1. In this section we briefly describe how this phases have been implemented.

First of all, some preprocessing steps are executed, especially the same adopted by the authors in [1] were chosen in order to obtain comparable results with the paper.

In particular, for each subject we first load the preprocessed EEG.

Given that the sampling frequency of the dataset is 500 Hz, then we *resample* the EEGs at 50 Hz, which allows us to analyse causality in a more distributed way between samples.

Subsequently, the signal is *segmented* into n epochs, so that the causality could be investigated within more specific time windows, capturing local relationships. According to the paper, it was decided to set n equal to 10, subdividing the EEG into segments with same lengths. Note that this leads the subjects to have different lengths for their epochs; anyway our choice holds since the recordings of the participants in the dataset have approximately the same duration.

Before proceeding with causal discovery, one last preprocessing step was necessary, that is to guarantee *stationarity* as it is assumed both by Granger and VAR-LiNGAM. For this purpose, two tests are applied on each channel, namely the Augmented Dickey-Fuller (ADF) and the Kwiatkowski-Phillips-Schmidt-Shin (KPSS). If at least one test reveals that the signal isn't stationary, then the it is differenced.

Preprocessing is followed by *causal analysis*, which is the core of this study.

A. *Causal Analysis*

Causality is investigated between the 19 signals corresponding to the electrodes; given a subject, this is repeated for each epoch and considering all the time lags. For this reason, since the goal consists in obtaining a global ECN for each group (such as Fig.4 in [1]), an *aggregation* step is needed at the end of the pipeline.

The maximum number of lags τ_{max} is an important hyperparameter when dealing with causal discovery methods. A widespread way to select an appropriate value is by visualizing some curves of information criteria for a VAR model with varying order p (i.e. the number of lags); a reasonable value is the one corresponding to the elbow occurring when minimizing the curve, that can be considered close to its real minimum. In Fig. 2 a demonstrative example is illustrated, where the Final Prediction Error (FPE) and three information criteria are considered: Akaike (AIC), Bayesian (BIC), Hannan-Quinn (HQIC). The curves show that the elbow is approximately between 2 and 4 lags. The value $\tau_{max} = 4$ was finally selected, consistently with the one adopted by the authors in our reference paper [1].

1) ***Granger****:* the Granger causality test is performed employing the *statsmodels* library. [2]

The test is pairwise, thus involving two channels at once. Differently from the examples in [7] we didn't just extract the minimum from the p-values returned by the function at the different time lags. Instead, we keep track of all the p-values and exploit this information for the aggregation phase (and, therefore, also for the final visualization of the ECNs) in the following sense: the more occurrences of p-values under a certain significance level of α, the more the causal link between the channels is robust across lags. This concept could be considered a kind of *empirical strength* and its utility is better described in Sec. IV.

2) ***LiNGAM****:* the adopted algorithm is VAR-LiNGAM for causality in time series, contained in the official *lingam* package. [3]

The fitting of the VARLiNGAM object computes the adjacency matrices containing the causal strengths between channels. The set of matrices is composed of both an instantaneous matrix B_0 (i.e. pure LiNGAM) and the lagged ones B_τ with $\tau = 1, ..., \tau_{max}$ (coming from the VAR extension).

However, LiNGAM doesn't provide a method to directly assess a measure of *reliability* to the results. Thus, the suggested method in literature it to apply *bootstrap*, which has revealed to be a huge problem in terms of computational effort. For this reason, we looked for an alternative or, at least, a compromise.

[1]Remind that exogenous variables are external influences whose values are generated outside the model, while latent confounding variables are the ones that cause the exogenous variables to be dependent.

[2]Available at: https://www.statsmodels.org/dev/generated/statsmodels.tsa.stattools.grangercausalitytests.html.

[3]See: https://github.com/cdt15/lingam.

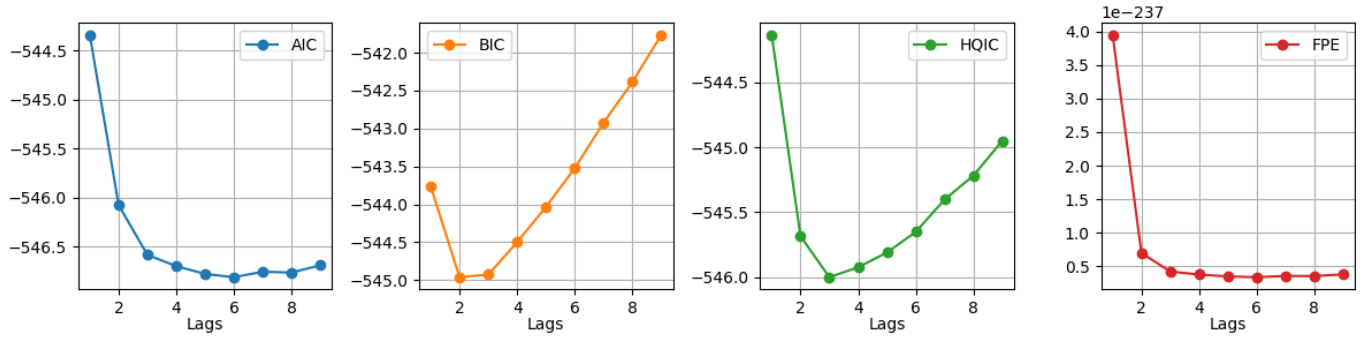


Fig. 2: Representative example for the selection of τ_{max}.

First of all, we tried to adopt a resampling technique known as *jackknife* [10]. It consists of a leave-one-out resampling and it was tested at the epoch-level for one subject, namely leaving one epoch out at once: the less the variance estimated by jackknife for a causal strength of a link, the more the link itself could be considered robust and reliable. Even though jackknife is less time-consuming than bootstrap, it requires quite much time anyway. In addition, by applying it at epoch-level we obtain an estimation at a higher abstraction, which does not convey a measure of stability within the epoch itself where the causal strengths are computed. Furthermore, jackknife is known to be very limited in literature when dealing with time series [10]. For this reason we excluded this option in the end.

An alternative was to compute the p-values coming from a Wald test on the VAR coefficients. However this turned into an inconclusive test since all the values were quite high (e.g. much more than $\alpha = 0.05$), thus not allowing us to detect reliable links. Moreover, this test does not account for the instantaneous strength matrix B_0, which is outside of the VAR model.

Finally, we opted to apply an *optimized bootstrap* version. Given that nowadays bootstrap's speed is an open issue, in the LiNGAM community it was suggested to speed it up by exploiting the *numba* package. [4] This is an optimized python compiler, very useful when dealing with heavy computation such as resampling. Even if the computation with optimized bootstrap is much faster, it is still costly to apply it on the entire dataset: in order to run it in a reasonable time we set the number of resamples equal to 10.

IV. Results

Causal analysis is performed for each epoch of every subject and across the lags up to τ_{max}. In order to show the results we aggregate the obtained values within each group.

First of all the results for each subject are averaged across the epochs. Then two slightly different procedures are adopted for Granger and VAR-LiNGAM, since the former provides p-values while the latter releases matrices of causal strength along with the corresponding bootstrap probabilities.

Regarding Granger, given a subject, the p-values lower than the threshold of $\alpha = 0.05$ are kept for each lag (we use one-hot-encoding). It follows an averaging across the lags for these accepted values, so that each subject is characterized by a kind of "empirical strength" matrix; i.e. the strength of a link is proportional to the number of lags where causality was discovered for the link itself.

This is done for all the subjects within a group, and the group-level ECN is obtained by averaging across the subjects' results.

A similar process is performed for LiNGAM. Given a subject, we keep track of the strengths corresponding to a probability higher than 95%. Then the final strength matrix for each subject is given by averaging the accepted strengths across lags. At the same time, an "empirical strength" is computed according to the accepted and rejected links across the time lags (again with one-hot-encoding), similarly to Granger; by averaging these matrices we get a value for each connection which is proportional to the number of lags where causality was discovered and accepted for the same connection.

As for Granger, this is done for all the subjects within a group, and the group-level ECN is again the average across the subjects' results.

The choice of $\alpha = 0.05$ for Granger and 95% as minimum bootstrap probability for LiNGAM was made in order to have comparable results with the ECNs in [1], in which the authors adopted $\alpha = 0.05$.

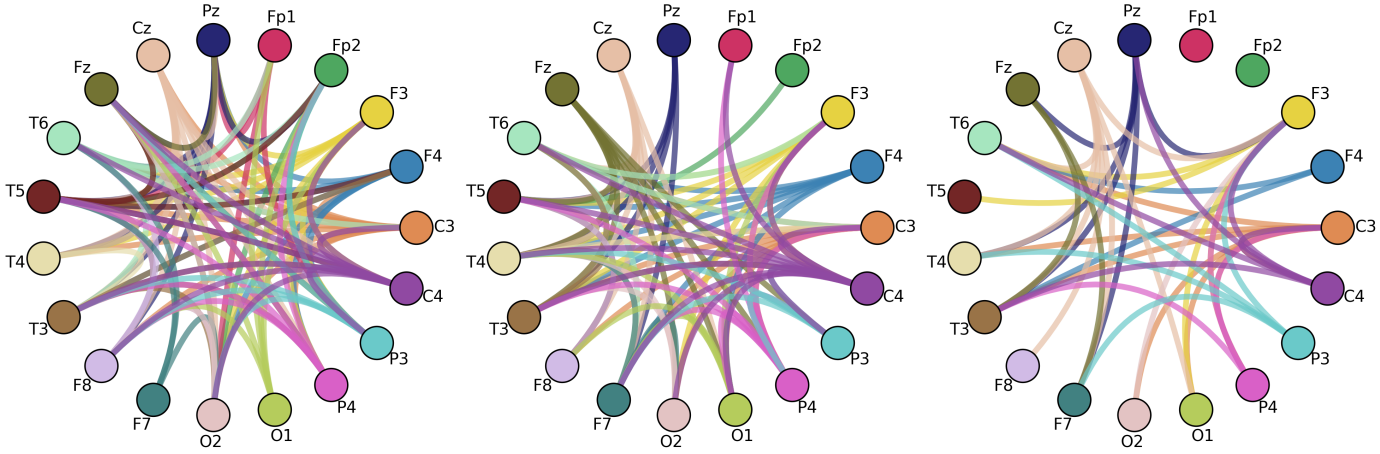
The ECNs for Granger and LiNGAM are illustrated respectively in Fig. 3 and Fig. 4. For sake of clarity only links with strengths above the 80° percentile are shown for Granger and LiNGAM. [5] Note that the width of a link is proportional to the mean rate of detected causality along time lags (i.e. what we call "empirical strength"); hence the thicker a link, the more reliable and robust it is, on average for the group, across lags.

A. Discussion

*1) **Granger**:* In Fig. 3 we can see at a glance how the ECN for the CN group (Fig. 3a) is well connected, indicating

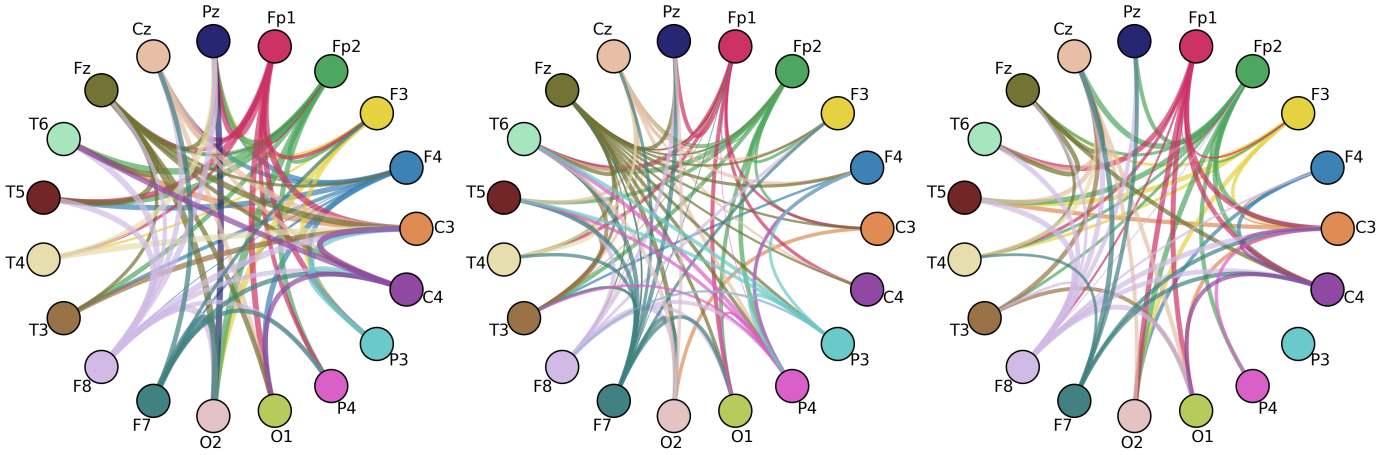
[4] See: https://github.com/cdt15/lingam/issues/130. Note the use of the *@jit* decorator, i.e. the main feature of numba.

[5] Since Granger doesn't provide a causal strength measure, the threshold refers to its "empirical strength". Instead, for LiNGAM the threshold is directly applied to the causal strengths.



(a) CN (b) FTD (c) AD

Fig. 3: Granger ECNs.



(a) CN (b) FTD (c) AD

Fig. 4: LiNGAM ECNs.

high integration between all the different brain areas, when compared to the ECNs for FTD and AD.

The FTD group (Fig. 3b) shows a decreased activity especially in the pre-frontal areas (Fp1 and Fp2) both in terms of received and emitted links. Moreover, some frontal regions are affected, such as F4, F8 and Fz. This aspect is coherent with the clinical knowledge of FTD since the frontal cortex is one of the main areas attacked by the disease. Also the pre-frontal cortex is often affected in FTD patients, specifically the ones with bvFTD which is the most common related syndrome.

On the other hand, in Fig. 3b a decrease in the temporal activity doesn't clearly emerge, besides some links between a temporal region and a frontal or pre-frontal one. For example, the links from T5 to Fp2 and to F4 are present for CN but are missing in the FTD group.

For what concerns the AD group (Fig. 3c), the links are very limitated. Central areas of the brain appear to aggregate less information compared to the CN group.

P4 and P3 have few connections in output and zero in input; this could be associated to fact that the parietal lobe is one of the most affected area by the disease.

Furthermore, as known in the clinical knowledge of AD, the atrophy involving the Default Mode Network (DMN) disrupts connections with the temporal and pre-frontal cortex. Actually within electrodes T3, T4, T5 and T6 there are poor links, while Fp1 and Fp2 do not even show one connection.

Finally, also the occipital regions, namely O1 and O2, are widely affected.

Notice that in Fig. 3a, 3b and 3c, the thickness of the links is approximately the same, suggesting that all the plotted connections are robust across time lags. Actually, a *limitation* for Granger causality is the fact that there are many links with p-values close to zero, which are filtered out by the threshold on the "empirical strength" even though they aren't much distant from the ones in the figure. P-values are often a limitation in the frequentist statistics, for this reason a

Bayesian approach maybe might solve this problem.

*2) **LiNGAM**:* On the other hand in Fig. 4 we still can distinguish some traits between the different groups, but with more subtle differences.

First of all, the CN group (Fig. 4a) is composed of well distributed links, thus reporting a high integration of information between many different brain areas. Unfortunately, the FTD group (Fig. 4b) isn't much different, sometimes it even shows more connections. It can be observed how frontal and temporal regions don't appear to be affected as for Granger, besides some frontal areas such as F3 and F4.

However, by focusing more on the details, it's interesting to note that the *thickness* of the links for the CN group is greater on average than the one for FTD group, revealing a higher robustness of the links across the time lags. Mainly, this phenomenon happens in pre-frontal regions (Fp1 and Fp2), which are known to be influenced in particular for people suffering from bvFTD, and in other frontal areas (for instance F4, F8, and Fz). In addition, there are also some temporal electrodes corresponding to a slight higher robustness (e.g. T3). The central regions, namely C3 and C4, look more robust, too.

About the AD group (Fig. 4c) we can observe that there are less links than for CN. As for Granger and in agreement with the clinical knowledge of the disease, the parietal lobe is damaged since P4 has less links and P3 has none.

Then, several temporal and frontal areas suffer from poor connections, moreover communication with the central regions as C3 and C4 is decreased.

Differently from Granger, the pre-frontal cortex isn't so disrupted and even the thickness of the lines doesn't change relevantly for Fp1 and Fp2.

In conclusion, the results for LiNGAM could indicate that the assumption of *independence* on exogenous variables (i.e. noise) might be not so helpful when performing causal analysis on EEGs. However, in addition it's important to point out that our analysis computes the reliability basing only on 10 *bootstrap resamples*. If there hadn't been severe computational problems, a higher number of resamples, e.g. 100, would have provided more reliable results.

*3) **PCMCI comparison**:* By comparing the ECNs retrieved in this study to the ones discovered with PCMCI in [1] some interesting observations could be discussed.

First of all, the crucial role of the central cortex emerges in the CN group in Granger results in Fig. 3, and progressively decreases when moving to FTD and, mostly, to AD. Moreover in the FTD group (Fig. 3b) the activity of the frontal and pre-frontal cortices diminishes, and in AD (Fig. 3c) the connections get even worse, also for the temporal regions. These aspects are quite similar to the ones encountered in our reference paper.

In our study, we also observed a low causality for parietal areas in AD group, which doesn't emerge in [1].

Regarding LiNGAM, the results in 4 do not show such a prominent role of the central cortex. Then, in order to capture differences between the CN and FTD group we must look at the robustness of the links, namely their stability across time lags; in these terms, the results show weaker connections for FTD (Fig. 4b) especially in pre-frontal and frontal regions, as in [1]. About the AD group (Fig. 4c), we can still observe lower activity for many cortices, as in the reference paper, but a distinct decrease for the pre-frontal lobe is missing.

On the other hand, as for Granger, the AD group highlights a really scarce activity for parietal areas, differently from [1].

V. Conclusions

In this work we estimated Effective Connectivity Networks from resting-state EEG signals of AD, FTD and cognitively normal subjects by applying two causal discovery approaches, namely Granger causality and VAR-LiNGAM, and compared the results with PCMCI-based findings reported in literature.

Granger causality revealed clear group differences, showing a progressive loss of connectivity from CN to FTD and, more prominently, to AD. The results highlighted reduced activity in frontal and pre-frontal regions in FTD and widespread connectivity impairment in AD, including temporal, parietal and occipital areas. LiNGAM provided more moderate group differences, mainly expressed as reduced robustness of causal links across time lags rather than a strong decrease in connectivity. Overall, AD subjects consistently exhibited the most disrupted connectivity patterns.

Several limitations must be considered. First, the reliance on frequentist p-values in Granger analysis may restrict interpretability, suggesting that Bayesian approaches could be explored in future work. Second, the reliability assessment for LiNGAM was constrained by the limited number of bootstrap resamples due to computational costs. Third, the dataset presents clinical imbalance across groups: AD subjects show lower MMSE (Mini-Mental State Examination) scores on average than FTD subjects, which may influence the observed differences. [6] Indeed, the average MMSE for the AD group is 17.75 (min=4, max=23) and for the FTD group is 22.17 (min=18, max=27): the presence of outliers could mask disease-specific patterns, especially when averaging across subjects.

Future developments may include increasing bootstrap resampling for VAR-LiNGAM (e.g. 100) and performing analyses on clinically similar groups based on MMSE. It could also be meaningful to investigate connectivity patterns across distinct FTD variants, which are not distinguished in the adopted dataset. Also different approaches of causal analysis on time-series data could be explored. In conclusion, ECNs derived from Granger and LiNGAM could be further evaluated as features for classification between AD and FTD, potentially supporting medical diagnoses.

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