

The Effects of Edge Weights on Correlating Dynamical Networks *Comparing Unweighted and Weighted Brain Graphs of nervus opticus Patients*

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Abstract: We are interested in the regression analysis of dynamical networks. Our goal is to predict real-valued function values from a given observation which is manifested as series of graphs. Every observation is described by a set of dependent variables that we want to predict using the dynamical graphs. These graphs change their edges over time, while the set of nodes is assumed to be constant. Such settings can be found in many real-world applications, e.g., communication networks, brain connectivity, microblogging. We apply several measures to every graph in the series to globally describe its evolution. The resulting multivariate time series is used to learn vector autoregressive (VAR) models. The parameters of these models can be used to correlate them with the dependent variables. The graph measures typically depend on the type of edges, i.e., weighted or unweighted. So do the VAR models and thus the regression results. In this paper we argue that it is beneficial to keep edge weights in this setting. To support this claim, we analyze electroencephalographic (EEG) networks from patients suffering from visual field defects. The edge weights are in the unit interval and might be thresholded. We show that dynamical network models for weighted edges lead to similar regression performances compared to those of unweighted graphs.

1 INTRODUCTION

Complex networks be constructed from any set of objects that “interact” with each other. So, based on the large number different objects with this property, complex networks can be found in nearly every part of our world, e.g., in social science (Wassermann and Faust, 1994), computer science (Faloutsos et al., 1999), medicine (Pereira-Leal et al., 2004), biology (Fischhoff et al., 2007), neuroscience (Sporns, 2010), and the World Wide Web (Kleinberg et al., 1999). Many researchers in these and other fields think that the analysis of complex networks may be useful to extract their intrinsic regularities, causal relations, and other useful pieces of information.

Probably the most complex network in the universe is the human brain. Since (Varela et al., 2001), if not earlier, it became very common in neuroscience to analyze functional networks. One property of these complex networks is their nature to dynamically—sometimes even chaotically—change their edges over time. They are typically obtained from neuroimaging methods, e.g., electroencephalography (EEG), electrocorticography (ECoG), magnetoencephalography (MEG), or functional magnetic resonance imaging

(fMRI). These methods record the activity of different brain regions—on the skull, on the brain meninges, or even inside the brain.

Whenever two brain regions, i.e., the nodes of the brain graph, are *co-active*, they are functionally *connected* to each other. These connections lead to a complex brain network which represents a high-level abstraction of the biological nervous cells. The analysis of these brain networks has already led to a better understanding of the functionality of different brain centers and the brain as a whole (Sporns, 2010).

Most research on complex graphs—no matter if neuroscientific or not—has mainly focused on static graphs. Such a static network is simply obtained by averaging the connections over time. Of course, it is far easier to analyze static networks than a series of networks. Nevertheless averaging diminishes an important property, i.e., the dynamics of such complex networks.

We argue that the dynamical nature of networks can also be exploited to analyze complex networks and some of their functions. Some first findings support this claim, at least for “damaged” human brain webs: In (Moewes et al., 2013) patients’ EEG has been correlated to the size of visual field deficits that

resulted from optic nerve damages (Wüst et al., 2002). The authors identified a relation between the extend of the vision loss and the dynamics of the functional brain connectivity. More precisely, the authors of this paper propose the first model-based approach to capture the relevant features of a dynamical complex network (Moewes et al., 2013).

In the above-mentioned work of the authors, unweighted graphs have been constructed to facilitate the computation of the network models. Therefore some patient-independent threshold for the edge weights had to be identified. The question we address in this paper is whether the tedious and somewhat arbitrary choice can be omitted. We thus claim that it is beneficial to use weighted graphs instead of unweighted ones.

To support this hypothesis, we analyze the same kind of EEG networks used in (Moewes et al., 2013). We apply different thresholds to the weighted edges to obtain an unweighted graph. Eventually we show that dynamical network models for weighted edges lead to similar if not better regression results than those of unweighted graphs. Thus the tedious and somewhat arbitrary choice of an edge weight threshold is superfluous.

The rest of the paper is organized as follows. Section 2 introduces the concept of dynamical networks to describe functional brain connectivity. Section 3 describes the experimental setup we have used to compare the regression performance of weighted and unweighted graphs. The results of this experiment are listed in Section 4. Finally, Section 5 concludes the paper.

2 DYNAMICAL NETWORK ANALYSIS

Having defined the functional connectivity of two brain regions, the corresponding dynamical brain network can be obtained. We remark that functional connectivity is just a statistical relationship between brain regions without implying any causal coherence (Pearl, 2009). There exist many ways to compute functional connectivity. We refer the interested reader to (Wendling et al., 2009) for a review on EEG connectivity measures.

2.1 Synchronization Likelihood

Out of these methods, we have used the synchronization likelihood (SL) (Stam and van Dijk, 2002) since it has been used in the literature to study the relation-

ship between structural network damage and functional connectivity (Stam et al., 2007).

Consider a multivariate time series (e.g. a multi-channel EEG recording) of length N with n variables. Let measurement $x_{i,k}$ be observed at timestamp i in channel k . For the SL, a time-delay embedding is computed by

$$X_{i,k} = (x_{i,k}, x_{i+L,k}, x_{i+2L,k}, \dots, x_{i+(m-1)L,k})$$

where L is the lag and m the dimension of the embedding. The state vectors $X_{i,k}$ shall capture the relevant patterns of the signal.

Now consider two channels A, B . The probability that $X_{i,k}$ are closer to each other than ϵ is

$$P_{i,k}^{\epsilon} = \frac{1}{2(W_2 - W_1)} \sum_{\substack{j \\ W_1 < |i-j| < W_2}} \theta(\epsilon - d(X_{i,k}, X_{j,k}))$$

where d is the Euclidean distance (or any other distance measure). For each k and i , a critical distance $\epsilon_{i,k}$ is computed such that $P_{i,k}^{\epsilon_{i,k}} = p_{\text{ref}}$ whereas $p_{\text{ref}} \ll 1$ is some user-defined threshold. For each pair of points in time (i, j) within $W_1 < |i-j| < W_2$, the number of channels $H_{i,j}$ for which $d(X_{i,k}, X_{j,k}) < \epsilon_{i,k}$ is computed by

$$H_{i,j} = \theta(\epsilon_{i,A} - d(X_{i,A}, X_{j,A})) + \theta(\epsilon_{i,B} - d(X_{i,B}, X_{j,B}))$$

where $\theta(x) = 0$ if $x \leq 0$ and $\theta(x) = 1$ for $x > 0$. Then the synchronization likelihood is then given by

$$SL_i = \frac{1}{2p_{\text{ref}}(W_2 - W_1)} \sum_{\substack{j \\ W_1 < |i-j| < W_2}} (H_{i,j} - 1). \quad (1)$$

Now the final brain graph can be computed. Note that only two of these the parameters are needed to compute SL if prior information about the frequency range and temporal resolution of the signal are given (Montez et al., 2006).

2.2 Graph Measures

We remark that any functional connectivity measure based on a sliding window will change its value over time. So do the edge weights of the corresponding brain graph. In (Moewes et al., 2013) the authors described these changes by learning how graph measures evolve.

Therefore we consider a set of different graph measures, i.e., the *number of cliques*, the *density*, and the *squared distance* between the current graph and the previous one (Bunke, 1997).

2.3 Vector Autoregressive Models

Vector autoregressive (VAR) models (Lütkepohl, 2005) have been shown to capture a high proportion of variance in these data.

A VAR model with p lags is given by

$$\vec{x}_t = c + \sum_{i=1}^p A_i \vec{x}_{t-i} + \varepsilon_t.$$

where c is a constant, A_i is a matrix storing the relationships between every pair of variable at point $t - i$, and ε_t is white noise. Flattening the coefficients A_i to a feature vector is beneficial for statistical learning methods.

3 EXPERIMENTS

In our experiments we used EEG data from 33 visually impaired subjects suffering from optic nerve damages (Wüst et al., 2002). For every patient, several optometric tests had been performed to describe the location and the size of the optic nerve damage for both eyes (Sabel et al., 2011). Among them are high resolution perimetry (HRP) (Kasten et al., 1998), static perimetry, kinetic perimetry, and the assessment of visual acuity (Bailey and Lovie, 1976). Based on these tests an expert defined the following clinical variables relevant to quantify the vision loss of both eyes:

- detection accuracy in HRP visual field (%),
- foveal threshold in static perimetry (dB),
- mean threshold in static perimetry (whole 30° visual field, dB),
- mean eccentricity in kinetic perimetry (°),
- visual acuity of near vision (LogMAR scale),
- visual acuity of far vision (LogMAR scale).

Nearly every optometric test is very tiring, time-consuming, and error-prone. Each one of them served as variable assumed to be depended from the dynamics in the EEG. It is our motivation to find good correlates of the EEG signal and these tests. If successful, a good prediction model may determine the size of the optic nerve damage by solely looking at EEG data that can be recorded much faster.

To preprocess the EEG data we did the following steps in EEGLAB (Delorme and Makeig, 2004):

- manually removal of noisy time frames at beginning/end of each recording,
- removal of uncommon EEG channels across all subjects (28 were used),

- high-pass filtering with cutoff frequency at 1 Hz to remove slow movements,
- notch filtering 50Hz to remove the European power line frequency,
- low-pass filtering with cutoff frequency at 95 Hz,
- re-referencing by the average electrode,
- down-sampling to 250Hz to reduce the costs of SL computation,
- manual removal of biological artifacts using independent component analysis (Makeig et al., 1996).

Biological artifacts that stem from electromyographic (EMG) or electrocardiograph (EKG) signal appear as noise in the recorded EEG signal in all variations. To remove EMG and ECG signals ICA was applied to very carefully remove noisy components.

We applied FIR filters to obtain the conventional frequency bands. They are associated with different brain states (Edwards, 2007). These bands are δ : $f \in (1, 4]$ Hz, θ : $f \in (4, 7]$ Hz, α : $f \in [8, 12]$ Hz, β : $f \in [13, 30]$ Hz, and γ : $f \in [30, 50]$ Hz. We expect the optometric variables to explainable by the dynamics of functional connectivity in these frequency bands. Functional connectivity was computed by the synchronization likelihood (Stam and van Dijk, 2002): We used an outer window length of $W_2 = 3$ s and a reference probability of $p_{\text{ref}} = 0.02$. To capture most of the dynamics, the sliding window shifted every 0.5 s, i.e. an overlay of $1/6$. Note that the analysis of the averaged graphs did not result in any useful model (Held et al., 2012).

We applied the measures mentioned in Section 2 to every brain graph. We used the Python package `igraph` (Csárdi and Nepusz, 2006) to accomplish this task. This resulted in a multivariate time series for each subject and each frequency band. Every time series was then fitted by a VAR model with $p = 1, 2$ for simplicity. Eventually we obtained $p \cdot 3 \cdot 3 = 9$ and 18 parameters, respectively, describing the dynamics of the corresponding multivariate time series.

4 RESULTS

We used ordinary least-squares regression and computed its leave-one-out (LOO) estimation of the true error. All models have been fit using the Python packages `sklearn` (Pedregosa et al., 2011) and `statsmodels` (Seabold and Perktold, 2010). Both the VAR parameters and the optometric variables have been z-score normalized, i.e., we subtracted the mean and divided by the standard deviation. The regression per-

Table 1: Mean-squared errors (MSE) of the VAR models to describe the vision loss of the right eye. The results from the weighted networks are shown on the left-hand side, the ones from the unweighted network are shown on the right-hand side.

weighted	δ	θ	α	β	γ	$SL \geq 0.5$	δ	θ	α	β	γ
HRP_DA	0.963	1.297	1.055	1.403	2.015	HRP_DA	1.337	1.291	1.448	1.204	2.440
SP_FT	1.335	1.447	1.330	1.142	1.869	SP_FT	1.202	0.941	1.571	2.159	1.627
SP_MT	1.134	1.542	0.897	1.436	2.087	SP_MT	1.197	1.140	1.396	2.261	1.949
KP_ME	1.259	1.559	1.369	1.354	1.844	KP_ME	1.318	1.601	1.435	2.329	1.245
VA_N_log	1.235	1.229	1.385	1.313	1.532	VA_N_log	1.215	1.365	1.483	2.115	1.779
VA_F_log	1.209	1.283	1.348	1.254	1.431	VA_F_log	1.181	1.411	1.580	2.656	1.411

Table 2: Mean-squared errors (MSE) of the VAR models to describe the vision loss of the right eye. The results from two unweighted networks that used different thresholds are shown on both sides of the table.

$SL \geq 0.7$	δ	θ	α	β	γ	$SL \geq 0.2$	δ	θ	α	β	γ
HRP_DA	1.371	1.580	1.144	1.148	0.979	HRP_DA	0.963	1.485	1.290	1.422	1.187
SP_FT	1.314	1.275	1.385	1.070	0.807	SP_FT	1.335	1.375	1.751	0.992	1.175
SP_MT	1.336	1.546	1.282	0.857	1.055	SP_MT	1.134	1.280	1.130	1.358	1.244
KP_ME	1.493	1.518	1.626	0.971	1.081	KP_ME	1.259	1.459	1.476	1.575	1.428
VA_N_log	1.270	1.321	1.292	1.183	0.752	VA_N_log	1.235	1.143	1.959	0.733	1.136
VA_F_log	1.256	1.242	1.325	1.295	0.838	VA_F_log	1.209	1.169	2.199	0.758	1.014

Table 3: Mean-squared errors (MSE) of the VAR models to describe the vision loss of the left eye. The results from the weighted networks are shown on the left-hand side, the ones from the unweighted network are shown on the right-hand side.

weighted	δ	θ	α	β	γ	$SL \geq 0.5$	δ	θ	α	β	γ
HRP_DA	1.580	1.572	1.450	1.629	1.397	HRP_DA	1.164	0.760	1.398	1.217	1.054
SP_FT	1.511	1.239	1.089	1.873	1.647	SP_FT	1.490	1.178	1.253	1.368	1.434
SP_MT	1.259	1.559	1.369	1.354	1.844	SP_MT	1.080	1.021	1.065	1.109	1.189
KP_ME	1.534	1.103	1.305	1.456	1.471	KP_ME	1.286	1.011	1.221	1.047	1.222
VA_N_log	1.541	1.140	1.298	2.193	1.578	VA_N_log	1.568	1.064	1.299	1.328	1.347
VA_F_log	1.425	1.117	1.433	1.913	1.682	VA_F_log	1.551	1.465	1.412	1.527	1.359

Table 4: Mean-squared errors (MSE) of the VAR models to describe the vision loss of the left eye. The results from two unweighted networks that used different thresholds are shown on both sides of the table.

$SL \geq 0.7$	δ	θ	α	β	γ	$SL \geq 0.2$	δ	θ	α	β	γ
HRP_DA	1.357	1.259	1.401	0.665	3.353	HRP_DA	1.580	0.967	1.174	1.755	1.783
SP_FT	1.423	1.524	1.283	1.698	1.412	SP_FT	1.511	1.128	1.451	1.166	1.234
SP_MT	1.230	1.267	1.122	1.502	1.056	SP_MT	1.291	0.931	0.745	1.344	1.367
KP_ME	1.283	0.987	1.324	1.037	0.992	KP_ME	1.534	1.106	1.471	1.651	1.299
VA_N_log	1.484	1.173	1.413	1.520	1.391	VA_N_log	1.541	1.091	1.726	1.367	1.124
VA_F_log	1.454	1.325	1.387	1.414	1.378	VA_F_log	1.425	1.257	1.717	1.340	1.240

formance of the normalized data was measured by the mean-squared error (MSE).

Tables 1 and 2 on the next page summarize our analyzes for the right eye. Weighted networks lead to MSE that are competitive to the ones of the thresholded networks. For low-frequency bands (i.e., δ , θ , α), the weighted networks produce even smaller errors. This is in line with the previous results in (Moewes et al., 2013) where 8 subjects less have been used. Equivalently, the results for the left eye are shown in Tables 3 and 4. They do confirm the findings.

5 CONCLUSIONS

We have advocated not to threshold edge weights when analyzing dynamical networks. The choice of a threshold to obtain an unweighted graphs can be omitted if the graphs are used for regression models. To show this we have analyzed EEG networks from patients suffering from visual field defects. We have thresholded the edge weights using several different values. Still, the models describing the weighted dynamical network lead to similar errors compared to those of unweighted graphs.

We plan to enlarge the data set to further increase the quality of our models. We also want to establish a way to combine the optometric tests of both eyes. A comparison of brain networks to classical EEG representations is in preparation. Furthermore we are working on the creation of different data sets, e.g., communication networks and microblogs, to generalize our findings.

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