Information Flow in Ising Models on Brain Networks

Sebastiano Stramaglia[1,2,3], Jesus M. Cortes[2,3], Leonardo Angelini[1], Mario Pellicoro[1], and Daniele Marinazzo[4]

[1] Dipartimento di Fisica, Universitá degli Studi di Bari and INFN, 70126 Bari, Italy
[2] Biocruces Health Research Institute. Hospital Universitario de Cruces. E-48903, Barakaldo, Spain
[3] Ikerbasque, The Basque Foundation for Science, E-48011, Bilbao, Spain
[4] Faculty of Psychology and Educational Sciences, Department of Data Analysis, Ghent University, B-9000 Ghent, Belgium

Abstract. We analyze the information flow in the Ising model on two real networks, describing the brain at the mesoscale, with Glauber dynamics. We find that the critical state is characterized by the maximal amount of information flow in the system, and that this does not happen when the Ising model is implemented on the two-dimensional regular grid. At criticality the system shows signatures of the law of diminishing marginal returns, some nodes showing disparity between incoming and outgoing information. We also implement the Ising model with conserved dynamics and show that there are regions of the systems exhibiting anticorrelation, in spite of the fact that all couplings are positive; this phenomenon may be connected with some evidences in real brains (the default mode network is characterized by anticorrelated components).

Keywords: Ising model, criticality, brain, Transfer entropy, Granger causality.

1 Introduction

The prototypical example of system exhibiting *criticality* is the Ising model, and criticality has been proposed to characterize brain signals [1]. Recently, the occurrence of a nonequilibrium critical dynamics in brain activity during sleep has been has been reported in [2]. In this contribution we implement the Ising model, with Glauber dynamics, on two real brain networks and analyze its critical behavior in terms of information flow, and find signatures of the law of diminishing marginal returns. We also compare with the results of the Ising model on a regular two-dimensional lattice, and show that, differently from this network, the critical state for the brain networks is characterized by the maximal amount of circulating information. We measure the information flow by transfer entropy [3], a technique that has been introduced in the context of the inference of the underlying network structure of complex systems from these time series. Recently [4] it has been shown that transfer entropy is strongly related

V.M. Mladenov and P.C. Ivanov (Eds.): NDES 2014, CCIS 438, pp. 301–308, 2014.

to Granger causality, a major tool to reveal drive-response relationships among variables, and it is connected to the inverse ising problem [5]. Initially developed for econometric applications, Granger causality has gained popularity also among physicists (see, e.g., [6,7,8,9]) and eventually became one of the methods of choice to study brain connectivity in neuroscience [10,11]. In a recent paper it has been shown that the pattern of information flow among variables of a complex system is the result of the interplay between the topology of the underlying network and the capacity of nodes to handle the incoming information, and that, under suitable conditions, it may exhibit the law of diminishing marginal returns, a fundamental principle of economics which states that when the amount of a variable resource is increased, while other resources are kept fixed, the resulting change in the output will eventually diminish (see [12] and references therein). The origin of such behavior resides in the structural constraint related to the fact that each node of the network may handle a limited amount of information. In [12] the information flow pattern of several dynamical models on hierarchical networks has been considered and found to be characterized by exponential distribution of the incoming information and a fat-tailed distribution of the outgoing information, a clear signature of the law of diminishing marginal returns.

In this work we also implement the Ising model with conserved dynamics (Kawasaki [13]) on a brain network and show that, although all couplings are positive, different regions of the brain exhibit anticorrelation, a feature that is observed in real brain (the default mode network is characterized by anticorrelated components [14]).

2 Bivariate Transfer Entropy of Ising Systems

In this section we describe how the transfer entropy is evaluated. Let us consider the configurations $\{\sigma_i(t)\}_{i=1,\ldots,n}$ of an Ising system of n spins living on network, obtained by Montecarlo with Glauber or Kawasaki dynamics. The lagged spin vectors are denoted $\Sigma_i(t) = \sigma_i(t-1)$.

For each pair of spins (i, j) connected by a link in the underlying network, the bivariate transfer entropy c, measuring the information flow $i \to j$ is evaluated as follows:

$$c_{ij} = \sum_{\sigma_j=\pm 1} \sum_{\Sigma_j=\pm} \sum_{\Sigma_i=\pm 1} p\left(\sigma_j, \Sigma_j, \Sigma_i\right) \log \frac{p\left(\sigma_j, \Sigma_j\right)\ p\left(\Sigma_j, \Sigma_i\right)}{p\left(\sigma_j, \Sigma_j, \Sigma_i\right)\ p\left(\Sigma_j\right)}, \tag{1}$$

where $p\left(\Sigma_j, \Sigma_i\right)$ is the fraction of times that the configuration (Σ_j, Σ_i) is observed in the data set, and similar definitions hold for the other probabilities. c_{ij} is zero if spins σ_i and σ_j are not connected by a link in the network. The total information flow is then given by $C = \sum_{i=1}^{n} \sum_{j=1}^{n} c_{ij}$.

3 Two Dimensional Ising Model and Brain Networks

In this section we report the results obtained on the two-dimensional Ising model (on a regular grid), and implemented on real structural networks. Considering

the 2D Ising model, we find that the total transfer entropy C has a peak in the paramagnetic phase, see figure (1): the critical point is not the one with the maximal circulation of information. Moreover we stress that the amount of information flow depends on the updating scheme, but the maximum is attained in correspondence of the same temperature, see e.g. figure (2) where Metropolis and Glauber dynamics are compared for the 2D Ising model.

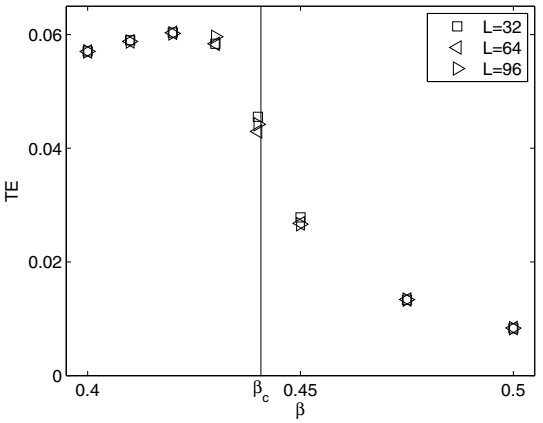

Fig. 1. The sum of bivariate transfer entropies for all pairs of spins, connected by a non-vanishing interaction, is depicted versus the coupling β for the 2D Ising model on a square lattice of size L^2, with $L = 32, 64$, and 96, with periodic boundary conditions. Transfer entropies have been evaluated averaging over 20 runs of 10000 iterations. The vertical line corresponds to the critical point. Since the maximum of the heat capacity matches quite well the critical point, this suggests that the system of linear size 96 is a reasonably good approximation to the thermodynamic limit, and that the transfer entropy has its maximum in the paramagnetic phase.

Turning to consider the Ising model with Glauber dynamics on a 74 nodes brain network obtained from diffusion tractography and contained in The Virtual Brain platform, a simulator of primate brain network dynamics [15], with couplings $J = \beta A$ (A is the connectivity matrix), in figure (3) we depict four quantities as a function of the inverse temperature β: R, the ratio between the standard deviation of c_{out} and those of c_{in}, where c is the information flow estimated by transfer entropy; the total causality C, i.e. the sum of all information flows; the ratio S between the intra-communities information flow and the inter-communities information flow, measuring the *segregation* of the network; the susceptibility χ. C, S and χ attain their maximum value in correspondance of the same temperature, whilst R is maximal at a lower temperature.

As another example, we consider a network which describes at low resolution the anatomical connectivity in brain (66 nodes), obtained via diffusion spectrum imaging (DSI) and white matter tractography [16]. In figure (4) we depict R, C, S and χ for the Ising model with Glauber dynamics where couplings are $J = \beta A$,

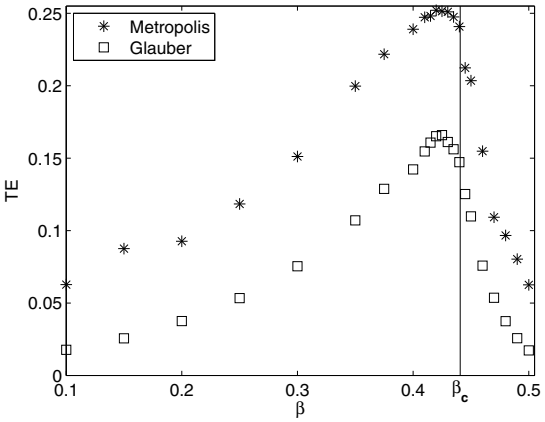

Fig. 2. The sum of bivariate transfer entropies for all pairs of spins, connected by a non-vanishing interaction, is depicted versus the coupling β for the 2D Ising model on a square lattice of size L^2, with $L = 16$, with periodic boundary conditions. Transfer entropies have been evaluated averaging over 20 runs of 10000 iterations. The vertical line corresponds to the critical point. Stars refer to Metropolis updating scheme, whilst empty squares refer to Glauber dynamics.

where A is the brain connectivity. In this case R, C and χ are unimodal and they peak nearly at the same temperature, so the critical state in this case is also the one with maximal circulation of information and the most affected by the law of diminishing marginal returns. The segregation of the system increases as the temperature is lowered. In both the brain networks considered here, the critical state is also the one maximizing the information flow.

It is interesting to report the analysis of equal-time correlations for the Ising model on the brain network. For each temperature we evaluate the correlational pattern obtained by Glauber dynamics and we find the corresponding modular decomposition by modularity maximization. Before the transition, the system has a stable decomposition in four moduli, after the transition by a single module for all nodes. In figure (5) the modularity of the best decomposition is plotted versus the temperature.

In order to compare the spin correlations by Glauber dynamics with those obtained by implementing the Ising model with conserved dynamics on the same brain network, using the Kawasaki update rule [13]. At the critical point the spin correlations for the Kawasaki dynamics can be both positive and negative: negative correlations emerge due to the conservation constraint. This is clear if we plot the Kawasaki correlations versus the Glauber correlations for the same pair of spins, at the critical point , see figure (6). We note that the pattern of correlations drastically change due to the conservation of the magnetization

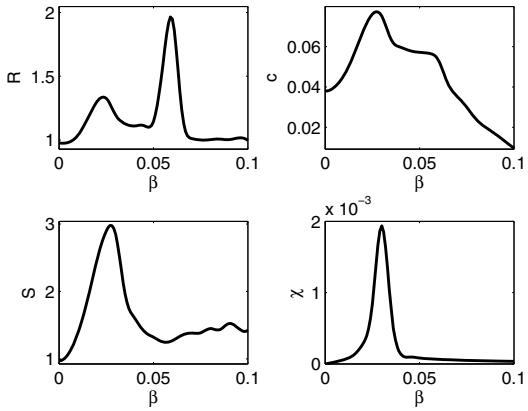

Fig. 3. Concerning the Ising model on the 74 nodes brain network, the following quantities are depicted versus the inverse temperature β: R, the ratio between the standard deviations of outgoing and incoming information flows (Top Left); the total causality C, i.e. the sum of all information flows in the network (Top Right); the ratio S between the intra-hemispheres and the inter-hemispheres information flows, measuring the segregation of the network (Bottom Left); the susceptibility χ (Bottom Right)

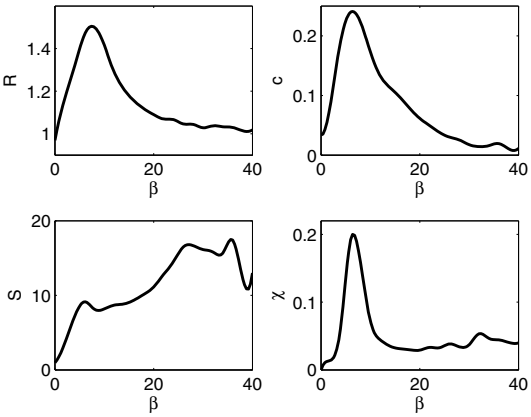

Fig. 4. Concerning the Ising model on the 66 nodes brain network, the following quantities are depicted versus the inverse temperature β: R, the ratio between the standard deviations of outgoing and incoming information flows (Top Left); the total causality C, i.e. the sum of all information flows in the network (Top Right); the ratio S between the intra-hemispheres and the inter-hemispheres information flows, measuring the segregation of the network (Bottom Left); the susceptibility χ (Bottom Right)

(in the spirit of the neural interpretation of the Ising model, we may speak of conservation of the average neural activation in the system). The modular decomposition of the Kawasaki correlational pattern corresponds to four modules:

in figure (7) we depict the average intra and inter correlations among moduli, and note that the correlation between different modules is negative: negative correlations between brain areas arise here as a consequence of a conservation law, even in a model where all the couplings have the same sign.

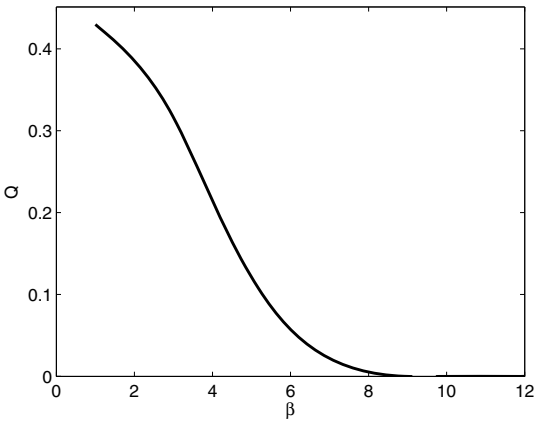

Fig. 5. The modularity of the best partition of the correlational network, for the 66 nodes network, is plotted versus the temperature

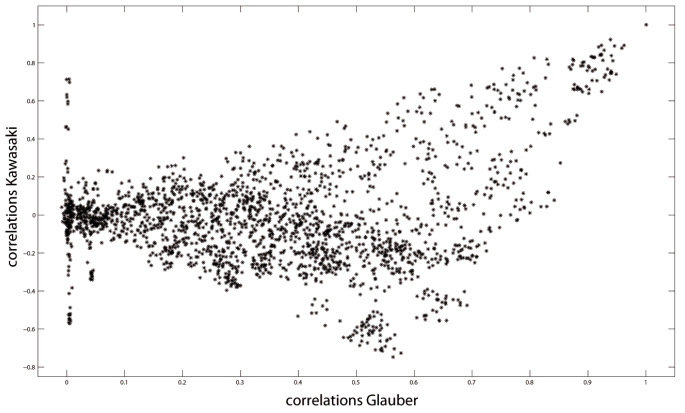

Fig. 6. For the brain network, the spin correlations by Kawasaki dynamics is plotted versus the spin correlations by Glauber dynamics, at criticality for the 66 nodes brain network

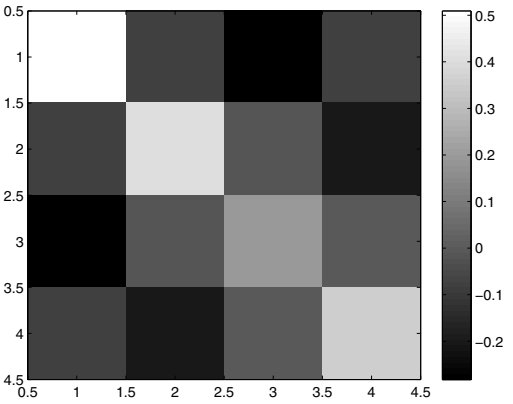

Fig. 7. The pattern of correlations, at criticality, by Kawasaki dynamics leads to four moduli in the 66 nodes brain network, obtained by maximization of the modularity. In the figure, along the diagonal the average intra correlation in the moduli is depicted, the off diagonal squares correspond to the average inter-correlations among the four moduli.

4 Conclusions

We have shown that the critical state of the Ising model on a brain network is characterized by the maximal amount of information transfer among units and brain effective connectivity networks may also be considered in the light of the law of diminishing marginal returns. This property does not hold for a generic network: we have shown that on the regular 2D grid the maximal circulation of information is not attained at criticality. It is a matter for further research the search for the largest class of networks in which the peaks of C and χ coincide.

We also reported that, in Ising models of brain, there is no need to introduce negative couplings to get anticorrelation between regions, indeed the implementation of a conserved dynamics leads to negative correlations. Negative correlations are observed, at the mesoscopic brain scale, for example w.r.t. the default mode network: whilst at the microscopic level the presence of both inhibitory and excitatory synapses justifies positive and negative couplings, at the mesoscopic scale the fibers connecting regions are similar hence all the couplings should all have the same sign. If the conservation law of the Kawasaki dynamics admits or not a physiological basis (e.g. a sort of homeostasis of the overall activity of the brain) is matter for future research. However, our findings may be relevant for the construction of brain models to reproduce the critical behavior of the brain.

References

1. Chialvo, D.R.: Critical brain networks. Physica A 340(4), 756–765 (2004)
2. Lo, C.C., Bartsch, R.P., Ivanov, P.C.: Asymmetry and basic pathways in sleep-stage transitions. EPL 102, 10008 (2013)
3. Schreiber, T.: Measuring Information Transfer. Phys. Rev. Lett. 85, 461–464 (2000)
4. Barnett, L., Barrett, A.B., Seth, A.K.: Granger Causality and Transfer Entropy are Equivalent for Gaussian Variables. Phys Rev. Lett. 103, 238701 (2009)
5. Roudi, Y., Tyrcha, J., Hertz, J.: Ising model for neural data: Model quality and approximate methods for extracting functional connectivity. Phys. Rev. E 79(5), 051915 (2009)
6. Smirnov, D.A., Bezruchko, B.P.: Phys Rev. E 79, 046204 (2009)
7. Faes, L., Nollo, G., Chon, K.H.: Assessment of Granger causality by nonlinear model identification: Application to short-term cardiovascular variability. Annals of Biomedical Engineering 36, 381–295 (2008)
8. Marinazzo, D., Pellicoro, M., Stramaglia, S.: Kernel Method for Nonlinear Granger Causality. Phys. Rev. Lett. 100, 144103 (2008)
9. Marinazzo, D., Pellicoro, M., Stramaglia, S.: Kernel-Granger causality and the analysis of dynamical networks. Phys. Rev. E 77, 056215 (2008)
10. Marinazzo, D., Liao, W., Chen, H., Stramaglia, S.: Nonlinear Connectivity by Granger causality. NeuroImage 58, 330–338 (2011)
11. Friston, K.J.: Functional and effective connectivity: A review. Brain Connectivity 1, 13 (2011)
12. Marinazzo, D., Wu, G., Pellicoro, M., Angelini, L., Stramaglia, S.: Information Flow in Networks and the Law of Diminishing Marginal Returns: Evidence from Modeling and Human Electroencephalographic Recordings. PLoS ONE 7(9), e45026 (2012)
13. Kawasaki, K.: Diffusion Constants near the Critical Point for Time-Dependent Ising Models. I. Phys. Rev. 145, 224–230 (1966)
14. Broyd, S.J., Demanuele, C., Debener, S., Helps, S.K., James, C.J., Sonuga-Barke, E.J.S.: Default-mode brain dysfunction in mental disorders: A systematic review. Neuroscience and Biobehavioral Reviews 33(3), 279–296 (2009)
15. Sanz Leon, P., Knock, S.A., Woodman, M.M., Domide, L., Mersmann, J., McIntosh, A.R., Jirsa, V.: The Virtual Brain: A simulator of primate brain network dynamics. Front. Neuroinform. (2013), doi:10.3389/fninf.2013.00010
16. Hagmann, P., Cammoun, L., Gigandet, X., Meuli, R., Honey, C.J., Weeden, V.J., Sporns, O.: Mapping the Structural Core of Human Cerebral Cortex. PLoS Biology 6(7), e159 (2008)