Editorial: On the relation of dynamics and structure in brain networks

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Despite more than a century-long effort, the functioning of the few-pound lump of white and grey matter that forms the brain remains at least partially a mystery. Physicists have made some significant contributions to the understanding of brain physiology, none perhaps more notable than Hodgkin and Huxley's, who discovered the ionic basis of nerve cell conduction. But could they also help shedding light on how large numbers of neurons interact to give rise to sophisticated behaviour?

Although complex, a neural system is in fact essentially a physical device meant to perform specific functions. As such, brain design must obey general engineering principles, which shape it at all scales from neuronal sub-components to the whole system scales.[1] Observable anatomy and physiology of the brain can be thought of as resulting from selective evolutionary pressures that managed trade-offs between energy consumption and adaptiveness, favouring energy-efficient wiring and coding patterns[2,3] and ultimately resulting in a non-random spatial and temporal structure of brain anatomy and dynamics. Making sense of this structure is therefore key to our understanding of the emergence of brain function.

SPATIAL ORGANIZATION: BRAIN NETWORKS

On the cold table of the neuroanatomist, the human brain appears as a gelatinous object approximately 1200 cc in volume and 1.5 kg in weight. An incision into its body reveals grey (actually rather more pinkish) matter interspersed with whiter fibres. Slicing a thin section of tissue with a simple microtome, and zooming in with a microscope, one can see that the former contains numerous cell bodies, while the latter is mainly composed of long-range myelinated axon tracts. In fact, the brain has an estimated 10^{11} neurons ($10^5/mm^3$), each with an average 10^4 connections with other neurons ($10^9/mm^3$). Wires connecting neurons have an estimated total length of 2×10^5 km (5 km/mm^3). Zooming in and out of this dense jumble of cells and cables reveals non-random structural patterns at different levels.

To the neurophysiologist's eye, possibly the most important aspect is how neurons process and transfer information. This is carried out by coupled neural units, each with its own dynamics. The waxing and waning of couplings between units induce observable transient network structures. Indeed, synchronization has been identified as a fundamental dynamical feature modulating cortical interactions by increasing the effectiveness of interactions between brain regions,[4] and there is now large consensus on its role in many aspects of the brain's cognitive function.

It is natural to think of these patterns as a network. A network can be endowed with topological properties at all spatial scales by resorting to the complex network theory, a statistical physics understanding of the graph theory, an old branch of mathematics.[5,6] Complex network theory appears as a tool of choice in tackling the challenges of brain activity, as it offers a qualitatively different view of brain mapping (offering a complex system vision of the brain), where networks are endowed with properties which stem in a non-trivial way from those of their constituent nodes [see Refs. 7 and 8 for comprehensive reviews]. Complex network theory is potentially applicable to any modality of neuroscientific data and allows characterizing brain organization at all spatial scales, from the cell to the system level.[9–12] Perhaps even more importantly given the brain's inherently multiscale nature, it also helps unveiling relationships *between* scales.[13] Zooming in and out of brain functional activity reveals a hierarchical fractal structure, with modules themselves containing other modules.[14] Moreover, it allows addressing classical but complex issues such as structure-dynamics relationships in a straightforward and elegant fashion. It also allows quantifying mechanistic properties such as its *efficiency* in performing the functions it is supposed to carry out and corresponding *costs*,[15] its *vulnerability* to lesions,[16,17] and its proneness to synchronize or to be controlled within or targeted towards given desirable regimes.[18,19] Finally, topological properties have important consequences for the system's information processing capacities. For example, the ability to process and propagate signals between nodes is affected by whether networks possess branching or loop-like features.[20]

TEMPORAL COMPLEXITY

Brain complexity is not only spatial but also temporal. This form of complexity becomes particularly observable at the long time scales of spontaneous brain activity.[21]

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Spontaneous brain activity has long been thought of as a null, amorphous state to which the brain reverts in the absence of stimulation. However, when observed long enough, brain spontaneous fluctuations appear to be characterized by structured patterns at all scales[22] re-edited across the cortical space in a non-random manner.[23]

The building blocks of this structure are fluctuations which the brain, as all dissipative out-of-equilibrium systems, generates even for fixed control parameter values and without external stimuli. Fluctuations are therefore signatures of brain activity which leave statistical and dynamical traces. At short time scales, the brain can be well approximated as an excitable system, and activity is temporally disordered, as fluctuations vanish exponentially fast and long-range temporal correlations can to some extent be ignored. At long time scales, however, various aspects of spontaneous activity display non-trivial glassy properties.

From a statistical view-point, as in a fractal object, brain activity shows similar properties in a wide range of temporal scales.[24–28] Self-similarity may not be exact so that brain activity displays *multifractality*.[29,30] Some regions of the phase space may take extremely long times to be reached, indicating that brain activity is generically (weakly) *non-ergodic*.[31] Memory of past activity decays sufficiently slowly that the time it takes for two time-points to totally decorrelate may diverge: scale separation is lost, microscopic fluctuations renormalize given rise to macroscopic effects, and a characteristic time ceases to exist.[32] Moreover, correlations are time-dependent, a phenomenon known as *ageing*.[31,33] The dynamics is *intermittent*, alternating relatively laminar and turbulent phases or avalanches,[34] various aspects of which are characterized by universal properties[35] and the respective onset of which can be used to define landmarks within resting activity.[36]

The presence of these complex fluctuations has led to the suggestion that resting brain activity operates near the critical point of a second-order phase transition.[35] Importantly, many studies have shown that criticality is associated with optimal information transfer[37] and storage,[38] communication,[39,40] computational power,[39] and dynamic range.[41,42] However, the neurophysiological aetiology and dynamics of critical activity are not yet entirely clear.

STRUCTURE-DYNAMICS RELATIONSHIPS

Overall, brain dynamics can be thought of as the collective dynamics of a network of excitatory-inhibitory units, coupled nonlinearly and with tunable strength.[43] Considering the spatial and temporal complexity and functional heterogeneity, the brain as a whole can be thought of as a spatially disordered system and its dynamics as a field $\phi(\vec{s}, t) \in \Phi$, which complex fluctuations endow with the spatio-temporal structure $\{\mathcal{S} * \Re\}$. $\Re$ can for instance be a relationship between time scales $\{\tau_i\}$, which may also have some spatial structure $\mathcal{S}$ with arbitrarily complex topological properties, and its behavioural repertoire as scale-dependent collective phenomena. The structure $\{\mathcal{S} * \Re\}$ is then a dynamical system in the space of fields $\Phi = \{\phi\}$, relating representations at different scales.[21]

Importantly, spatial and temporal structures can interact in a scale-dependent way.[44] Once again, a statistical mechanics approach to graphs can lend a hand in the representation of such a problem. Once nodes are endowed with their own dynamics, it is possible to distinguish between dynamics *in* the network, i.e., node dynamics, and topological dynamics *on* the network, i.e., the temporal evolution of the network's topological properties. The interdependence of these two dynamics is a defining feature of *adaptive networks* such as the brain.[45,46] By gauging the interactions between these two dynamics, it becomes possible to study how this relationship can be related to the emergence of function in healthy brains, normal aging, and in various pathologies. One of the major questions is how synchronizability depends on the network structure, particularly its topology. The significance of this sort of analysis is better appreciated in the light of evidence showing that non-Markov bursty dynamics can arise naturally in systems with quenched heterogeneity.[47] If brain activity could be modelled as resulting from the coupling of identical oscillators, a straightforward and elegant answer would be provided by the *master stability function*.[48] Unfortunately, oscillator homogeneity is not a realistic assumption in general, and alternative ways of gauging topology-dynamics interactions need to be devised.

It is important to appreciate that the extent to which a given process depends on the structure of the network on which it unfolds is a matter of relative time scales of the topology and of the relevant dynamics. Given the complex spatial and temporal structure of brain activity, the analysis of the interplay between topology and dynamics in neural activity represents a vast and still insufficiently explored field of investigation. Of interest would of course also be to understand how function may retroact, possibly at much longer time-scales, to optimize network topology and brain dynamics.

Anatomy and dynamics

While spatial structure $\mathcal{S}$ can in principle refer to both anatomy and dynamics, considerable effort has been devoted to understanding the role the anatomical network structure plays in shaping dynamics. In a sense, anatomical networks can be seen as homeomorphic to resting dynamical ones in the limit of an infinitely slow time scale. The resting state and task-activated dynamical networks are related by some fluctuation-dissipation relationship,[49] but the relationship between the latter and anatomical networks appears to be more complex. How anatomical connectivity relates to brain dynamics has motivated several experimental and modelling studies (see Ref. 50 for a review) but remains an open question. At slow time scales, the correlation structure of spontaneous resting fluctuations has been shown to be related to the underlying anatomical circuitry both in humans[51–53] and in monkeys.[54] This has led to the suggestion that resting activity arises from neuronal noise correlations between brain areas that are coupled by the underlying anatomical connectivity. At fast time scales, on the other hand, anatomy is best regarded as a boundary condition for the dynamics, but exactly what role it plays and at what (spatial and temporal) scales this constraint cannot be neglected remain unclear,[51,55]

and *collectivity*[56] may in fact play a role no smaller than that of *connectivity*. At these scales, there is little reason to see any overlap between dynamical and structural networks, in much the same way as there is no reason for the properties of information packets in a given communication system to be isomorphic to the physical network within which they travel. Indeed, dynamical networks can be described in spaces not necessarily isomorphic to the anatomical one reflecting the fact that the generic complexity of the phase space in (disordered) spatially extended systems can only very partially be accounted for by looking at the physical structure in which the dynamics takes place. Furthermore, anatomical networks are presumably optimized (with respect to a number of plausible properties) at evolutionary time scales, whereas brain dynamics need not be optimized, particularly when no hard-wired system was developed during the course of evolution to subserve a given function (e.g., in thinking or reasoning).

OVERVIEW OF THIS ISSUE

This *Focus Issue* proposes contributions addressing three main issues related to brain dynamics: (1) the emergence of avalanche dynamics, (2) the relationship between anatomy and dynamics, and (3) the quantification of dynamical brain connectivity.

Damicelli and colleagues[57] present a paper that studies how the emergence of modularity in structural connectivity networks may be explained by synaptic plasticity mechanisms. To do so, the authors assess a minimalistic network model with excitable nodes and discrete deterministic dynamics. In particular, the authors use a variant of the three-state cellular automaton Susceptible-Excited-Refractory (SER) model of excitable dynamics which is deterministic and time-discrete and relies on three synchronous update rules. This model allowed to estimate *in silico* pairwise functional connectivity as the normalized co-activation matrix. For the structurally connected pairs, such a matrix was used to estimate a link probability retention (pruned links are immediately reintroduced in the network following a randomly uniform fashion). Simulations combined the SER model, and the plasticity rule was run on top of structural connectivity networks with 100 nodes (synthetic topologies tested included directed random graphs, scale-free networks, and modular networks). Different network densities ranging between 20% and 60% were considered for the case of random graphs, 20% otherwise. Under these conditions, the results indicate that a local Hebbian plasticity rule acting on a brain network model of excitable units can generate a global reorganization of the initial networks, evolving them towards modular architectures, leaving their degree-distribution unchanged. Moreover, such structural reorganization has consequences for the network dynamics and is associated with an increase in the correlation between the structural and functional connectivities. Overall, the presented approach suggests that the emergence of some topological features of complex networks may be a consequence of specific dynamical properties that govern the system.

Lombardi and colleagues[58] investigate the emergence of neuronal avalanches, i.e., cascades of activity bursts involving a variable number of neurons and whose size distribution can be approximated by a power law. The authors use a minimal neural network model inspired in self-organized criticality to explain the 1/f decay of the power spectrum associated with the neuron activity. Their minimal model shows that this particular decay, reported in a number of experimental studies, is only obtained when a certain percentage of inhibitory neurons is included in the network (around 30%). The results hold for different network structures. This kind of power law scaling in the distribution of avalanche sizes has been related to the existence of long-range spatial correlations in a diversity of dynamical systems. Furthermore, when the percentage of inhibitory neurons is modified (or even disregarded), bimodal distributions arise, which suggests that the balance between excitation and inhibition plays a crucial role in normal, non-pathological functioning of neuronal networks.

Kanders and colleagues[59] go one step beyond and investigate criticality arising in large networks of neurons using a recurrent neural network model based on Rulkov neurons with dynamical synapses. They obtain the Lyapunov spectrum for the subcritical, critical, and supercritical cases, by using the Jacobian matrix evaluated at points along the trajectory of the network's state vector. Interestingly, they show that avalanche criticality does not necessarily co-occur with edge-of-chaos criticality. Rather, there are different network and dynamical conditions under which avalanche criticality is possible, which points to a link between avalanche and edge-of-chaos criticality. These findings suggest that an analysis of the dynamical state of the network should be provided together with a full analysis of the avalanche behaviour. In addition, their study highlights the presence of a paradox that may be of importance for understanding the biological network behaviour: Upon an increase in the synaptic coupling, chaos may intensify in the sense of a larger entropy production rate, while losing coherence, indicated by a decrease in the largest Lyapunov exponent.

The approach of Battiston and colleagues[60] to the understanding of the interplay between structural and anatomical networks relies on the existence of anatomo-functional motifs in the human brain. Motifs are certain subgraphs containing a reduced number of nodes whose structure is over-represented in the network when compared with equivalent random networks. The novelty of this work is the construction of multi-layer motifs containing both the anatomical and functional connections of the human brain, recorded using diffusion tensor imaging (DTI) and functional magnetic resonance (fMRI), respectively. The authors describe how to classify the motifs of a multiplex network and extend motif analysis to networks with an arbitrary number of layers. The results reveal that subgraphs in which the presence of a physical connection between brain areas coexists with a non-trivial positive correlation in their activities are statistically overabundant. On the contrary, motifs containing direct anatomical connections and negative correlations between brain regions are not common. These results suggest the existence of a reinforcement mechanism between the two layers that is also reported by looking at how the probability of finding a

link in one layer depends on the intensity of the connection in the other one.

López-Madrona and colleagues[61] examine the interplay between anatomical and effective networks in the hippocampus. Specifically, they use a computational model of the hippocampal formation to explore the capacity of linear and nonlinear measurements of directed statistical interdependencies between simulated neurophysiological time series, aiming to retrieve effective connections between the simulated populations and draw complete functional diagrams. Importantly, they analyse the effects of modifying the anatomical connections between the different areas of the hippocampus, the dentate gyrus, and the entorhinal cortex. Their results show that the causality measurements, viz., Granger Causality (GC) and Partial Transfer Entropy (PTE), strongly depend on the internal connectivity of the entorhinal cortex. Surprisingly, causal links can be robustly inferred regardless of the excitatory or inhibitory nature of the connection, which highlights the critical importance of comprehensive neuroanatomical information since single inhibitory connections or cortical layer interactions may drastically change the effective information flow in the system. Finally, GC and PTE methods are also shown to potentially lead to distinct effective connectivity patterns, adding a higher level of complexity to their interpretation.

Stramaglia and colleagues[62] run a variant of the Ising model with conserved magnetization on top of structural connectivity networks obtained from awake and anesthetized individuals. Spin correlations from this Ising model are compared to empirical functional connectivity at both the single edge level and the modular level. Overall, it is shown that conservation of magnetization is associated with better correspondence between the structure and function. The structure-function relationship is strengthened under anaesthesia, both at the link and modular levels, when compared to awake conditions. Moreover, at the peak of specific heat (corresponding to the critical state), spin correlations are minimally shaped by the underlying structural connectivity, explaining how the highest match between the structure and function is obtained at the onset of criticality. Altogether, these findings suggest that brain dynamics under anaesthesia shows a "departure" from criticality and could lead to new perspectives for understanding emergent functional patterns by interpreting the concept of conserved magnetization as a homeostatic constraint on neural activity.

Sethi and colleagues[63] compare mouse structural connectivity to regional BOLD signal dynamics. To do so, the authors combine the group-level Allen Mouse Brain Connectivity Atlas as the underlying directed weighted structural connectivity with BOLD fMRI time-series data measured in 184 brain regions in eighteen anesthetized mice. Instead of establishing pairwise relationships between the structure and function of pairs of brain regions, this study focuses on region-level features from structural connectivity (namely, in-degree, out-degree, betweenness, and clustering coefficient) and their association with BOLD signal dynamics. After correcting for volumetric variations among regions, significant correlations between regional properties derived from structural connectivity and resting state fMRI dynamics were found only when edge weights were accounted for. Furthermore, those were associations with variations in the autocorrelation properties of the resting state fMRI signal. In particular, the strongest relationships involved weighted in-degree, which was positively correlated with the autocorrelation of fMRI time series at a time lag of 34 secs, as well as a range of related measures such as relatively high frequency power ($f > 0.4$ Hz). Altogether, these results indicate that the topology of inter-regional axonal connections of the mouse brain is closely related to intrinsic, spontaneous dynamics such that regions with a greater aggregate strength of incoming projections show longer time-scales of fluctuations in activity. Indeed, these findings might lead to new ways of constraining models to simulate brain dynamics.

Bettinardi and colleagues[64] examine the relationship between structural and resting-state functional connectivities in the human brain by introducing a novel graph measurement denominated topological similarity. The authors start from the assumption that pairwise functional correlations do not result from unique or single structural paths but from signal propagation through all possible paths, with a progressive attenuation of the influence as the number of steps (i.e., path-length) increases. Accordingly, they have developed a graph measure, the topological similarity, which estimates the expected cross-correlation between pairs of nodes based on the similarity of the estimated "influences" two nodes receive from the whole network. If two nodes receive the same sets of inputs, then they will tend to be strongly correlated. In other words, topological similarity between two brain regions quantifies their tendency to be dynamically or functionally coupled based on the resemblance of their inputs with respect to the whole network. The results show that structural connectivity shapes, to a large extent, the time-average functional connectivity observed at rest. These results supports that the global path structure of the underlying structural network determines the contribution of the network over the functional collective dynamics while implicitly incorporates information about all other network features such as degree distribution or modularity.

A fundamental issue of functional brain networks is the quantification of functional links between brain regions since they strongly determine the overall properties of the network. Zanin and Papo[65] propose the use of Granger Causality (GC) to evaluate coordination between brain sites. For inherent statistical reasons, causal relationships (viz., GC, transfer entropy, and dynamic causal modelling) are usually quantified over time-windows much larger than functionally meaningful time scales. However, this approximation fails to account for the time-limited dynamical nature of brain interactions, as information between brain regions is typically transmitted in transient or intermittent bursts. To overcome this limitation, Zanin and Papo propose an algorithm to evaluate GC that is capable of accounting for intermittent causal couplings and of changing the direction in neural activity. The algorithm calculates the causality between two brain regions considering every possible time window and next non-overlapping windows in which causality is stronger are selected. Intermittent causality was shown to differentiate

between two experimental groups (controls and alcoholic subjects) better than causality averaged over long time-windows. Furthermore, differences showed up more at a local level rather than in the form of global network properties. This new methodology can potentially be applied to any kind of brain imaging data sets, as long as the temporal resolution is good enough to evaluate causality.

The way brain connectivity is quantified may not be the only important issue. The way connectivity itself is understood may be problematic. Graph metrics are defined upon dyadic representations of brain activity. However, the extent to which dyadic relationships can capture the brain's complex functional architecture and information encoding is unclear. Moreover, because network representations of global brain activity are derived from continuous response measures, it is methodologically complex to characterize the architecture of functional networks using traditional graph-based approaches.[66] To address these limitations, Stolz and colleagues[67] use *Persistent Homology* (PH), a method from computational topology that quantifies global topological structures and their persistence through scales. In this paper, the authors focus on "loops" in functional networks. A loop in a graph is a set of at least four edges that are connected in a way that forms a topological circle. Specifically, PH is applied to functional networks constructed from time-series obtained with the Kuramoto model and fMRI data from human subjects performing a simple motor-learning task in which subjects were monitored during three days. Interestingly, non-persistent 1-loops were reported in all cases (Kuramoto model and fMRI), which can be interpreted as an indicator of the emergence of functional community structures. Moreover, changes in the 1-dimensional loops during the motor-learning task take place after the second day of the learning task. In particular, brain regions that yield 1-loops in the functional networks on days 2 and 3 seem to exhibit stronger synchronization on average than those that yield 1-loops on day 1, indicating that the PH is a suitable tool to track the evolution of functional communities. The interplay between PH and graph Laplacians adds possible directions for future applications of this new approach.

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