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Mathematical Methods and Models of Systems Interactions and Network Dynamics Special Session B17

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Networks of dynamical systems exhibit complex global behaviors characterized by non-equilibrium phase transitions, self-organization and criticality, noise-induced patterns and nonlinearity, partial synchronization and desynchronization, and a variety of adaptive responses to internal and external perturbations. A fundamental question is how such collective behaviors emerge from the interplay between the dynamics of individual systems and the network topology in which the systems are embedded.

In recent years numerous investigations have focused on the mathematical properties of adaptive networks of dynamical systems to quantify the laws of time-varying and nonlinear network interactions, to uncover multiple forms of coupling and feedback loops, and to derive principles of coordination and network integration among dynamical systems in association with network states and functions.

Novel analytic and computational methods derived from applied mathematics, nonlinear dynamics, information theory, control and adaptive networks theory as well as high-dimensional network modeling approaches were recently developed to build an adequate theoretical framework and explore how hierarchical organization in network structure (sub-networks and modules) relates to multi-stability and meta-stability of dynamic states with emergent global functions.

While these advances have a broad spectrum of applications to physical, biological, ecological and social systems, investigations of the temporal complexity and emergent global behaviors in adaptive networks of dynamical systems lay the foundation of a new theoretical framework to study living systems. Of particular relevance to physiology and medicine is how physiological systems, processes and functions can be mathematically described and studied within the context of complex networks dynamics.

A new field, Network Physiology, has emerged to address the fundamental question of how physiological systems and sub-systems continuously coordinate, synchronize, and integrate their dynamics as a network to optimize functions and to maintain health. In addition to the traditional approach in biology and physiology that defines health and disease through structural, dynamic, and regulatory changes in individual systems, the new conceptual framework of Network Physiology focuses on the coordination and network interactions among systems as a hallmark of physiological state and function. This poses new challenges in developing generalized methodology adequate to quantify complex dynamics of networks where nodes represent diverse dynamical systems with distinct forms of coupling that continuously change in time.

Novel methods are needed to provide insights into physiological structure and function in health and disease, and across levels of integration from genomic interactions to inter-cellular signaling and metabolic networks, to communications among integrated organ systems.

This special session will provide a venue for leading experts to present and discuss recent advances in the theory of adaptive networks of dynamical systems and their applications to physiology and medicine, as well as to outline key questions and future directions of research aimed to uncover the relations of network topology and dynamics with emerging physiological states and functions in health and disease. This session is scheduled on July $25^{th} - 26^{th}$.

Schedule and Abstracts

July 25, 2024

11:30–11:55 The New Field of Network Physiology: Building the Human Physiolome
Plamen Ch. Ivanov (Boston University, USA)

Abstract. The human organism is an integrated network where complex physiological systems continuously interact to optimize and coordinate their function. Organ-to-organ interactions occur at multiple levels and spatiotemporal scales to produce distinct physiologic states. Disrupting organ communications can lead to dysfunction of individual systems or to collapse of the entire organism. Yet, we do not know the nature of interactions among systems and sub-systems, and their collective role as a network in maintaining health.

We initiated a new interdisciplinary field, Network Physiology, which aims to address these fundamental questions. Through the prism of concepts and approaches from statistical and computational physics, nonlinear dynamics, and applied mathematics, we will present a new framework to identify and quantify dynamic networks of organ interactions. We focus on inferring coupling and dynamical interactions among organ systems from continuous streams of synchronized recordings of key physiologic variables. In contrast to traditional complex networks theory, where edges/links are constant and represent static graphs of association, novel approaches in Network Physiology aim to establish dynamical aspects of organ communications in real time, to track the evolution of organ network interactions and quantify emerging collective network behaviors in response to changes in physiological state and condition.

We will report first findings utilizing this new framework to (i) investigate brain-brain network interactions across distinct brain rhythms and locations, and their relation to new aspects of neural plasticity in response to changes in physiologic state; (ii) characterize dynamical features of brain-organ communications as a new signature of neuroautonomic control; and (iii) establish basic principles underlying coordinated organ-organ communications. We will demonstrate how physiologic network topology and systems connectivity lead to integrated global behaviors representative of distinct states and functions. The presented investigations are initial steps in building a first Atlas of dynamic interactions among organ systems and the Human Physiolome, a new kind of Big Data of blue-print reference maps that uniquely represent physiologic states and functions under health and disease.

12:00–12:25 Analysis of Medical Data with Synolitic Networks
Alexey Zaikin (University College London, UK)

Abstract. There's a merging trend in Nonlinear Dynamics, Graph Theory, and Artificial Intelligence. Our focus here is on representing multidimensional data as graphs, even when the network structure is unknown. This method shows promise in handling the complexity of biological systems by linking features through biological and thermodynamic laws. However, it requires prior knowledge of feature connections. Alternatively, correlation graphs for time-series or correlation-prediction graphs can aid in early detection or survival analysis. Another method, pioneered by Zanin and Bocaletti, constructs a "parenclitic" network without prior knowledge of interactions. This approach has been effective in identifying key genes and metabolites in diseases, including cancer using DNA methylation data. Additionally, 2-dimensional kernel density estimation (2DKDE) is proposed for modeling control distribution when linear models are insufficient. We've also introduced "synolitic" networks, an ensemble of classifiers in graph form [1,2], useful for analyzing age-related trajectories in Down's syndrome [3] and predicting survival in severe Covid-19 cases [4,5]. These networks can be considered an ensemble of classifiers in a graph form and thus are a kind of correlation network where the correlation is in the changes between two classes (e.g. disease and non-disease). Notably, Synolitic graphs facilitate the use of Graph Neural Networks for data analysis, hence, enabling Graph Neural Networks to analyse the data which were initially not represented in the form of a network..

12:30–12:55 Cytokine-induced coherent structures in a reaction–diffusion-chemotaxis model of Multiple Sclerosis

Rossella Rizzo (University of Palermo, ITALY)

Abstract. In this work, we develop a model for the evolution of the Multiple Sclerosis pathology that considers the modulatory influence of cytokines on the activation rate of macrophages. Our starting point is the reaction diffusion-chemotaxis model proposed in [1], which generalizes the system proposed by Khonsari and Calvez [2,3] to describe Baló's sclerosis. The model represents the interaction dynamics among three species of cells: macrophages $\tilde{m}(T,X)$, cytokines $\tilde{c}(T,X)$ and oligodendrocytes $\tilde{d}(T,X)$.

$$(1)\quad \begin{cases} \dfrac{\partial \tilde{m}}{\partial T} = D\Delta_{\mathbf{X}}\tilde{m} - \nabla_{\mathbf{X}}\cdot(\Psi(\tilde{m})\nabla_{\mathbf{X}}\tilde{c}) + \lambda\dfrac{\tilde{c}}{k_{\tilde{c}}+\tilde{c}}\tilde{m}(\overline{m}-\tilde{m}), \quad \Psi(\tilde{m}) = \psi\dfrac{\tilde{m}}{\overline{m}+\tilde{m}} \\ \dfrac{\partial \tilde{c}}{\partial T} = \dfrac{1}{\nu}(\varepsilon\Delta_{\mathbf{X}}\tilde{c} + \mu\tilde{d} + b\tilde{m} - \alpha\tilde{c}), \\ \dfrac{\partial d}{\partial T} = \kappa\, F(\tilde{m})\,\tilde{m}\,(\overline{d}-\tilde{d}), \qquad F(\tilde{m}) = \dfrac{\tilde{m}}{\overline{m}+\tilde{m}} \end{cases}$$

Through a weakly nonlinear analysis, we focus on the study of the Turing-type instabilities of the nontrivial homogeneous steady state, leading to the settlement of stationary patterns of inflammation and demyelination. Using biologically relevant parameter values, we show that the asymptotic solutions of our model system reproduce the concentric demyelinating rings, confluent plaques, and preactive lesions observed in Balò sclerosis and type III Multiple Sclerosis. Comparing the present model with the model proposed in [1], where the macrophages activation mechanism is due to innate immunity, the cytokine-induced macrophages activation rate encourages pattern formation, and leads to a pattern with smaller wavenumber. Moreover, our findings reveal that the alternative scenario proposed here results in a less aggressive pathology characterized by reduced inflammation levels and significantly slower disease progression.

Under the appropriate regularity conditions on the initial data, we prove the existence of a unique global solution to our proposed system,following the strategy used in [4].

This study provides insights into the role of cytokines in the pathogenesis of Multiple Sclerosis, shedding light on the disease's dynamics and offering potential avenues for therapeutic interventions.

14:30–14:55 Nonlinear Schrödinger Equation on Networks and Hybrids

Riccardo Adami (Politecnico di Torino, ITALY)

Abstract. We review the results obtained in the last decade on the problem of modeling systems described by the Nonlinear Schrödinger Equation, constrained to exotic domains like networks, or metric graphs, and hybrids, i.e. structures made of pieces of different dimensionality. Our results apply to the dynamic of Bose-Einstein condensates in ramified traps, for the case of network, and in magneto and optical traps in the case of hybrids. We will mainly focus on the problem of the existence of Ground States. This is a joint project with Filippo Boni, Raffaele Carlone, Simone Dovetta, Alice Ruighi, Enrico Serra, Lorenzo Tentarelli, and Paolo Tilli.

15:00–15:25 Effects of anomalous diffusion on pattern formation in the FitzHugh-Nagumo model

Gaetana Gambino (University of Palermo, ITALY)

Abstract. Anomalous diffusion provides a more realistic description of various physical phenomena in different contexts, such as in autocatalytic chemical reactions on porous media, ion channels in the plasma membrane, and also population dynamics. In this talk we shall discuss the influence of anomalous diffusion on the onset of stationary nonhomogeneous structures in the Fitzhugh-Nagumo model, as it is considered a prototype system to describe excitable dynamics both in chemical reactions and population dynamics.

The anomalous diffusion relaxes the classical requirement of a rapidly diffusing inhibitor, allowing spatial segregation of the species in both cases of short-range activation/long-range inhibition or long-range activation/short-range inhibition. Specifically, the anomalous diffusion exponent of the activator small enough, compared to the inhibitor diffusion exponent, enlarges the range

of parameters for Turing instability. We will also prove that the presence of anomalous diffusion in the model leads to different phasing of the species maintaining the same kinetics. In particular, the spatial structures induced by long-range activation/short-range inhibition mechanism are always out of phase and subcritical in most of the instability region.

Finally, we shall provide detailed descriptions of the possible emerging patterns in the 1D and 2D rectangular domains via bifurcation analysis.

15:30–15:55 Assessment of complexity and dynamical coupling between complex systems using Entropy Rate and Mutual Information Rate Measures: simulations and application to physiological data

Riccardo Pernice (University of Palermo, ITALY)

Abstract. The human organism has been recently described, according to the "Network Physiology" approach, as a complex integrated network composed of multi-component organ systems continuously interacting through various feedback mechanisms to provide homeostatic balance and to react to external stimuli or intrinsic physiological alterations [1]. The dynamical behaviour of a complex system and its pairwise interactions with another system can be respectively evaluated using information-theoretic measures of Entropy Rate (ER) and Mutual Information Rate (MIR). In particular, ER has been widely employed to assess the complexity of a random process, related to nonregularity and unpredictability of its dynamics [2]. On the other hand, MIR is a dynamic measure of the non-directed symmetric interrelationships between coupled systems, and can be expressed as the sum of the individual ERs of the two processes minus their joint entropy rate [3]. Thanks to their suitability for describing short-length data with strong stochastic and noisy components, such measures are of great interest for the practical analysis of physiological time series [2,3].

In this work, after defining the theoretical formulation of ER and MIR dynamical measures, different approaches for their estimation are compared: a linear model-based estimator relying on Gaussian data, two model-free estimators based on discretization of the variables carried out either via uniform quantization through binning or rank ordering through permutations, and a model-free estimator based on direct computation of the differential entropy via k-nearest neighbor searches. The various estimators are first validated and compared on simulated univariate and coupled dynamic systems, including linear autoregressive or mixed non-linear deterministic and linear stochastic dynamics processes. Then, the framework is applied to different datasets of real-world time series describing the dynamics of coupled biomedical physiological systems, including physiological variability series descriptive of cardiovascular and cardiorespiratory interactions assessed at rest and during physiological stress or during controlled breathing conditions.

Our results evidence that statistically significant and physiologically meaningful patterns of the ER and MIR measures can be achieved in the analyzed datasets with a proper selection of the estimation parameters. Simple and fast approaches based on linear parametric or permutation-based model-free estimators allow efficient discrimination of changes in the short-term evolution of complex dynamic systems, while computationally expensive nearest-neighbour method achieves more reliable results in presence of non-linear dynamics [2,3].

16:00–16:25 Modeling tumor disease and sepsis in physiological networks

Eckehard Schöll (Technische Universität Berlin, GERMANY)

Abstract. In this study, we provide a network physiology perspective to the modelling of pathological states induced by tumors or infection. A unified disease model is established using the innate immune system as the reference point. We propose a two-layer network model for carcinogenesis and sepsis based upon the interaction of parenchymal cells (organ tissue) and immune cells via cytokines, and the co-evolutionary dynamics of parenchymal, immune cells, and cytokines [1,2]. Our aim is to show that the complex cellular cooperation between parenchyma and stroma (immune layer) in the physiological and pathological case can be functionally described by a simple paradigmatic model of phase oscillators. By this, we explain carcinogenesis, tumor progression, and sepsis by destabilization of the healthy state (frequency synchronized), and emergence of

a pathological state (multifrequency cluster). The coupled dynamics of parenchymal cells (metabolism) and nonspecific immune cells (reaction of innate immune system) are represented by nodes of a duplex layer. The cytokine interaction is modeled by adaptive coupling weights.

17:00–17:25 Assessment of cortico-muscular synchronization via complex-system measures

Franca Tecchio (CNR - Consiglio Nazionale delle Ricerche, ITALY)

Abstract. A deeper understanding of the cortico-muscular synchronisations in the central and muscular component and their interaction will provide insight into how specific coupling characteristics are modulated by age (Graziadio et al., 2010), autonomic regulation in different physiological states and chronic fatigue. Particular attention will be paid to the identification of state and synchronisation measures specific to complex systems, in order to capture the richness of information transfer expressed in the patterns exchanged between the various nodes of the network, and their sensitivity to different behavioural and structural conditions, in particular dominance of manual control networks. We will focus on developing novel analytic and computational methods within the network physiology framework, as well as with attention to fatigue.

The fine-tuning of central networks, which depends on the continuous integration of sensory influxes with programming and executive activities within feedback circuits, supports all behavioural expressions, and in particular the control of hand movement (Tecchio et al., 2020), which are modified by the levels of fatigue (Tecchio et al., 2006; Tomasevic et al., 2013; Padalino et al., 2021). Given the crucial role of hemicorporeal dominance, we studied the synchronisation between brain and muscle electrical activities, depending on the side of the body performing the movement (Tecchio et al., 2006). Furthermore, we manipulated visual feedback during an elementary isometric handgrip to investigate the effects on brain-muscle synchronisations (L'Abbate et al., 2022). We approached this study through the most widely used measure, cortico-muscular spectral coherence (CMC), and a new measure that takes into account the complex nature of the signals involved (the normalised compression distance, CMncd, Pascarella et al., 2024). Modulation of visual information modified the cortico-muscular synchronisations assessed by the two measures and cortical involvement, reflecting the crucial role of gaze in human behaviour. Dominance-dependent features were captured by CMncd more than by CMC, suggesting that signal representation by sinusoids misses an important aspect of neural network communication.

17:30–17:55 Normal Form of Turing-Hopf Codimension Two of a Chemotaxis Model of Three-Species Lotka-Volterra with IGP

Faezeh Farivar (University of Palermo, ITALY)

Abstract. In this talk, we explore the codimension two analysis of Turing and Hopf bifurcations within the framework of a three-species Lotka-Volterra model. The model comprises an IG-predator species, an IG-prey species, and a common resource species shared by both. Incorporating Lotka-Volterra type interaction dynamics, coupled with nonlinear diffusion to capture the movement of IG-prey towards lower density areas of IG-predator, our investigation delves into the potential for species extinction within this system [1].

Utilizing linear stability analysis around the coexistence point, we establish the conditions for the occurrence of Hopf instability. Furthermore, we investigate the role of cross-diffusion, which can induce Turing instability in the system [2,3]. Notably, the introduction of cross-diffusion leads to Turing instability, a phenomenon not observed with only classical diffusion terms. Our analysis also examines the influence of individual parameters on both Turing and Turing-Hopf instabilities.

In addition, we employ a perturbation technique based on the method of multiple scales [4] to compute the normal form of the reaction-diffusion system in the proximity of the Turing-Hopf codimension-2 bifurcation point. This allows for a deeper understanding of the system's behavior near critical bifurcation points.

By elucidating the intricate dynamics of Turing and Hopf bifurcations in this Lotka-Volterra model, our study contributes to a deeper understanding of the complex interplay between species interactions, spatial diffusion, and perturbation effects. These findings hold significance for

ecological systems where such bifurcations can have profound implications for biodiversity and ecosystem stability.

July 26, 2024

11:30–11:55 Saddle-Node Separatrix-Loops and Neuronal Network Dynamics
Krasimira Tsaneva-Atanasova (University of Exeter, UK)

Abstract. Normal reproductive function and fertility rely on the rhythmic release of gonadotropin-releasing hormone (GnRH), orchestrated by the hypothalamic GnRH pulse generator. The posterodorsal subnucleus of the medial amygdala (MePD), a brain region implicated in processing external environmental cues including stress effects, acts as a key regulator of the GnRH pulse generator. However, the specific neuronal pathways governing the dynamic, stress-induced modulation of GnRH secretion remain largely elusive. Here we employ computational modelling and analysis to investigate the effects of dynamic inputs on GnRH pulse generator activity.

To this end, we develop and analyse a mathematical model representing MePD neuronal circuits comprised of GABAergic and glutamatergic neuronal populations [1], integrating it with our GnRH pulse generator model [2]. Numerical bifurcation analysis enables us to identify critical model parameters and distinct neuronal network dynamic regimes. Moreover, our analysis highlights the significance of saddle-node separatrix-loops in influencing these dynamics. To investigate further the saddle-node separatrix-loops identified in our model, we propose unfolding a generic heteroclinic loop featuring one nonhyperbolic and one hyperbolic saddle using discrete (Poincaré) maps.

12:00–12:25 Breaking consensus in kinetic opinion formation models on graphons
Mattia Zanella (University of Pavia, ITALY)

Abstract. In this work we propose and investigate a strategy to prevent consensus in kinetic models for opinion formation. We consider a large interacting agent system, and assume that agent interactions are driven by compromise as well as self-thinking dynamics and also modulated by an underlying static social network. This network structure is included using so-called graphons, which modulate the interaction frequency in the corresponding kinetic formulation. We then derive the corresponding limiting Fokker Planck equation, and analyze its large time behavior. This microscopic setting serves as a starting point for the proposed control strategy, which steers agents away from mean opinion and is characterised by a suitable penalization depending on the properties of the graphon. We show that this minimalist approach is very effective by analyzing the quasi-stationary solutions mean-field model in a plurality of graphon structures. Several numerical experiments are also provided the show the effectiveness of the approach in preventing the formation of consensus steering the system towards a declustered state.

12:30–12:55 How complex is to be a hub? Complexity as a proxy for the network degree distribution
Inmaculada Leyva (Universidad Rey Juan Carlos, SPAIN)

Abstract. The relationship between topology and dynamics along the path to synchrony in complex networks has been thoughtfully explored, and the knowledge gathered so far has driven crucial applications. However, there are relevant cases in which the system operates in a partial or weakly synchronization regime to preserve the balance between functional integration and parallel processing, whereas full synchronization is pathological. However, even in this incoherent state, each unit is encoding the signature of its structural role in its own dynamics. We explore how this feature can be used to extract information about the network without having to make any reference to pairwise correlations, particularly useful when the structure is unknown [1,2]

To evaluate our hypothesis about the relationship between topological role and node dynamics, we study the evolution of the k-class statistical complexity $\langle C \rangle_k = \sum_{[i|k_i=k]} C_i/N_k$ in large complex networks of dynamical units. We observe that, immediately after coupling, the relative complexity splits as a strongly hierarchical function of k, that persists for all the range of coupling d up to the system synchronization, where all nodes recover the complexity of the uncoupled state. This behaviour suggests a way to rank the nodes according to the complexity of their time series and, therefore, to potentially use this anti-correlation as a proxy for the degree sequence.

The fact that this correlation between $\langle C \rangle_k$ and k persists along a large range of coupling d means that the method could be useful in natural systems, where in general, the coupling is not an accessible parameter. We have obtained equivalent results in a large variety of other systems as non phase-coherent chaos as Lorenz model, pulse-coupled neurons [1], delayed systems as Mackey-Glass, higher dymensional systems as Saito [3], and has been experimentally observed in networks of nonlinear electronic circuits [1]. The result is robust against node heterogeneity, noise and dynamical and topological changes.

To conclude, we have shown that a distinctive negative correlation between complexity and degree in a large variety of systems can be observed in the weakly coupled regime. These results suggest that the role played by the topology of a network could be unveiled by just computing the dynamical complexity associated with the time series sampled at each node. The fact that structural information of a network can be inferred without computing pairwise correlations like those commonly performed in functional networks could be exploited in diverse fields as neuroscience, econophysics or power grids.

14:30–14:55 Data-Driven Modeling, Inference and Control of Complex Time-Varying Networks: A Neuroscience Application Perspective

Paul Bogdan (University of Southern California, USA)

Abstract. From brain activity dynamics to microbiome, and even chromatin interactions within the genome architecture, the biological processes exhibit a pronounced non-stationary, non-Markovian and non-Gaussian behavior. While the modeling of interactions among neurons and various brain regions builds on the assumption of complete knowledge of the associated complex network (CN) and Markovian (memoryless) assumptions, due to sensing limitations only a part of the complete CN is available at most of the times. In this talk, we will discuss a general and comprehensive mathematical framework for inferring, modeling and controlling adaptive networks of dynamical systems. A special emphasis will be put on capturing the multi-fractal / non-Markovian, non-stationary and non-Gaussian behavior of biological networks and identifying the laws of time-varying and nonlinear network interactions that drive these adaptive networks of dynamical systems. In order to infer the "unknown unknowns" influencing this TVCN, we will discuss techniques for jointly estimating the fractional latent node activities, and unknown drivers, as well as iteratively infer the complete model (latent + observed). In order to infer, characterize, model and efficiently control adaptive networks of dynamical systems, we will discuss the weighted multifractal graph (WMG) generator that allows us not only to deal with scarce and noisy observations but also derive the higher order network statistics of biological systems. This new mathematical framework can uncover the multiple forms of coupling and feedback loops among biological processes that will enable us to derive principles of coordination and network integration among dynamical systems in association with network states and functions. We will review the benefits and remaining challenges related to this framework for adaptive networks of dynamical systems across several neuroscience case studies and highlight several problems to be addressed by our community in order to define the theory of adaptive networks of dynamical systems and their applications to physiology and medicine.

15:00–15:25 Brain dynamics in a simple class of adaptive neural networks: from oscillations to avalanches and scaling in collective behaviors

Fabrizio Lombardi (University of Padova, ITALY)

Abstract. Brain networks exhibit collective dynamics as diverse as scale-specific oscillations and scale-free neuronal avalanches. Although existing models account for oscillations and avalanches separately, they typically do not explain both phenomena, are too complex to analyze analytically or intractable to infer from data rigorously. Here we propose a feedback-driven Ising-like class of neural networks that captures avalanches and oscillations simultaneously and quantitatively. In the simplest yet fully microscopic model version, we can analytically compute the phase diagram and make direct contact with human brain resting-state activity recordings via tractable inference of the model's two essential parameters. The inferred model quantitatively captures the dynamics over a broad range of scales, from single sensor oscillations to collective behaviors of extreme events and neuronal avalanches. Furthermore, the model reproduces distributions

of coarse-grained resting-state activity, which we find to approach a fixed non-Gaussian form with evidence of scaling. Importantly, the inferred parameters indicate that the co-existence of scale-specific (oscillations) and scale-free (avalanches) dynamics, as well as the scaling behaviors observed in coarse-grained activity, occurs close to a non-equilibrium critical point at the onset of self-sustained oscillations.

15:30–15:55 On a multiscale mean field spin glass
Emanuele Mingione (University of Bologna, ITALY)

Abstract. We will consider a mean-field disordered system with Sherrington-Kirkpatrick Hamiltonian in the presence of multiple thermal equilibria, namely assuming that the random coupling can be divided into a finite number of families having their own effective equilibrium temperature. The generating functional (thermodynamic pressure) of the model is constructed trough a hierarchical sequence of annealed averages, reminiscent of the Replica Symmetry Breaking interpolation. The above construction can be also seen as a multiscale decomposition of the Hamiltonian viewed as a Gaussian process. We show that the thermodynamic limit of the pressure per particle can be represented as a solution of an infinite dimensional variational principle of the Parisi type. In particular we will show that the multiscale structure acts as constraint in the space of functional order parameters.

16:00–16:25 Mapping the dynamics of physiological systems and their interactions: An Information Theoretic Perspective with applications in Network Physiology
Yuri Antonacci (University of Palermo, ITALY)

Abstract. The emerging field of Network Physiology (NP) combines empirical and theoretical knowledge from various disciplines to gain insight into the dynamic interaction of physiological systems as a network [1]. Data-driven network inference methods play a key role in NP and are designed to build a network model from a set of observed time series. Such a model is typically encoded by a graph where nodes constituting dynamical systems are connected by edges representing functional dependencies, describing self-effects and pairwise interactions. Moreover, many physiological systems exhibit high-order interactions, i.e. interactions involving more than two nodes [2]. In this study, we employ various information-theoretic measures across three analytical approaches to characterize: i) node dynamics using the entropy rate (ER) which measures the complexity of a system and quantifies the interactions of order one, i.e. the interaction occurring internally to the analyzed node [3]; ii) pairwise interactions via the Mutual Information Rate (MIR) to measure dynamical coupling and hence quantify the interactions of order two; iii) higher order interactions through the O-Information Rate (OIR) to identify synergistic and redundant behaviors [2]. We show how network interactions can be studied by shifting from the time domain to the frequency domain in the presence of activity rich in oscillatory content, and how these domains can be combined to allow time-resolved and time-frequency analysis when transitions between different physiological states are present [3]. In particular, different approaches and information theoretic measures are used to characterize physiological interactions at different orders to study: i) brain dynamics [4] and brain-heart interactions [3] in humans; ii) respiratory dynamics during sleep apnoea; and iii) transitions in higher order interactions occurring in different sleep stages. Our results highlight the effectiveness of information-theoretic measures in describing the behavior of dynamical interactions at different orders of interaction thus underpinning their application in NP. Using the NP framework, we elucidate the interplay between different organ systems, taking advantage of recent insights from information theory to unravel the redundancy/synergy balance within brain and physiological networks, facilitating a deeper understanding of diverse physiological mechanisms.

17:00–17:25 Entropy measures for long-range correlated sequence
Anna Carbone (Politecnico of Torino, ITALY)

Abstract. The talk will address how the *cluster entropy* $\mathcal{S}_{\mathcal{C}}[P]$, based on the Shannon functional of the empirical cluster distribution P and the *relative cluster entropy* $\mathcal{D}_{\mathcal{C}}[P\|Q]$ based on the Kullback-Leibler functional of the empirical cluster distribution P and Q, a model probability

distribution of the clusters, can be used to quantify long-range correlated sequences, i.e characterized by probability distributions in the form of power laws.

Case studies in biology (for the characterization of chromosomes, pangenome graphs) and finance (to estimate the Hurst exponent of prices and volatility series build a multiperiod portfolio) will be presented.

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