DY 14: Networks: From Topology to Dynamics I (joint session of BP, DY, SOE)

Time: Wednesday 10:15–12:45 Location: H44

DY 14.1 Wed 10:15 H44

Stability of continuous vs. Boolean dynamics — •Fakhteh Ghanbarnejad and Konstantin Klemm — Department of Bioinformatics, University of Leipzig, Germany

Boolean networks are time- and state-discrete models of dynamical systems with many variables and quenched disorder in the couplings. The use of such discrete models makes large systems amenable to detailed analysis. The discretization, however, may bring about "artificial" behavior not found in the continuous description with differential equations. The usual definition of Boolean attractor stability is based on flipping the state of single nodes and checking if the system returns to the attractor, similar to a damage spreading scenario. This stability concept, however, does not reflect the stability of limit cycles in the corresponding continuous system of delay differential equations. Here we have a fresh look at the correspondence of stability definitions in continuous and discrete dynamics. We run extensive numerical simulations to test stability on various system architectures (networks). We establish a criterion for assessing stability of the continuous dynamics by probing the discrete counterpart.

DY 14.2 Wed 10:30 H44

Reliable Boolean networks with threshold functions — •Manuel Ross, Tiago Peixoto, and Barbara Drossel — Institut für Festkörperphysik, TU Darmstadt

Boolean networks are used to model biological networks, such as gene regulatory networks. The nodes of the networks are in this case interpreted as genes, and the state is taken as activity, discretised to Boolean values. An attractor trajectory of the Boolean network is equivalent to a periodic time evolution of the respective network. The usual approach to analyzing these models consists in studying the dynamics of a given network or ensemble. The reverse approach, which we take here, is to deduce the structure of a network from dynamical properties, done for instance by Lau et al. [1]. The dynamical property considered here is a robust sequence of states, i.e., the dynamical trajectory shall not change under perturbations in the update times. We consider the extreme case where the dynamics is reliable under any update sequence, so only one node can possibly change its state at any given moment in time. Such reliable networks were introduced recently in our group [2]. We now extend this work by permitting only threshold functions as update functions. This imposes severe restrictions on the possible reliable trajectories, in contrast to the original study, where all Boolean functions were permitted. We explore the consequences of this restriction for the statistical properties of the possible dynamical trajectories. These statistical properties are finally compared to microarray data. References: [1] K. Y. Lau et al. Phys. Rev. E, 75(5):051907, 2007. [2] T. Peixoto and B. Drossel. arXiv:0905.0925v1, 2009.

DY 14.3 Wed 10:45 H44

Contact networks and the spread of MRSA in hospitals — Lisa Brouwers[1], •Andrzej Jarynowski[1,2,3], Fredrik Liljeros[1], and Xin Lu[1] — [1]Stockholm University, S106 91 Stockholm, Sweden — [2]Department of Physics, Cologne University, Zülpicher Str. 77. 50937 Köln, Germany — [3]The UNESCO Chair of interdisciplinary studies, Wroclaw University, pl. M. Borna 9 50-204 Wrocław, Poland

The bacterium meticillin resistant Staphylococcus aureus(MRSA)is known to be the largest care related the infection problem. We investigated the Common Care Registry containing information about all patient visits within Stockholm County during the outbreak period with registry over diagnosed MRSA cases. Methods to analyze the contact network of persons visiting the same care unit is developed within the project as well as methods to analyze in what way network structure affects the transmission of MRSA. We study matrixes of disease transition in hospitals population(infected versus people, who could sent infection). In stationary case:(a)We have matrixes of estimators of that probabilities and other statisical properties of contact networks. In time evolution case:(b)We divided outbreak in smaller, periodical intervals and looked at how MRSA was spreading in time. Quasi-MCMC(Markov chain Monte Carlo) method and artificial networks(main parameter is number of contacts during specific time interval)help us to understand real- and simulated-paths of disease transition. Matrixes of probabilities(b)were used to find mechanism of change states(vectors of all population 0-health or 1-ill)and we can run quasi-MCMC to get most likely paths.

DY 14.4 Wed 11:00 H44

A novel threshold mechanism for epidemics on complex networks — •Vitaly Belik[1] and Theo Geisel[1,2] — [1]Max-Planck-Institut für Dynamik und Selbstorganisation — [2]Georg-August-Universität Göttingen

Recently much effort was devoted to modeling of spatial spread of infectious diseases, triggered by latest pandemics, such as SARS and H1N1 influenza. Theoretical understanding of different modeling frameworks and taken assumptions are substantial factors determining reliability of predictions based on the models. We investigate on an epidemiological model explicitly taking into account such an important factor of human mobility as tendency to move frequently among several most preferred locations rarely undertaking long trips. We considered complex network topologies as an underlying mobility network and discovered new threshold behavior of the global epidemic outbreak in terms of time spent on distant location. Our results are supported by extensive stochastic numerical simulations. We believe our findings contribute to understanding of epidemiological dynamics and development of effective control and preventive measures.

DY 14.5 Wed 11:15 H44

Stochastic load-redistribution model for cascading failures in interconnected systems — •Jörg Lehmann and Jakob Bernasconi — ABB Switzerland Ltd., Corporate Research, Segelhofstrasse 1K, CH-5405 Baden-Dättwil, Switzerland

We present a new class of stochastic models for cascading failure propagation in interconnected systems [1]. These models take into account, in a statistical sense, important physical characteristics of realistic load-redistribution mechanisms: (i) the load increments after a failure depend on the load of the failing element; (ii) the failed load is redistributed non-uniformly among the remaining elements. Within a Markov approximation, we are able to describe the cascading failure dynamics of these models in terms of a generalized branching process. This yields an analytical solution for the breakdown probability in the limit of large system sizes. The application to blackouts in power grids is discussed.
[1] J. Lehmann and J. Bernasconi, arXiv:0909.4185.

15 min. break

DY 14.6 Wed 11:45 H44

Synchronization in laser networks: From motifs to complex topologies with multiple delays. — •Thomas Dahms and Eckehard Schöll — Institut f. Theo. Physik, Sekr. EW 7-1, Technische Universität Berlin, Hardenbergstr. 36, 10623 Berlin, Germany

We investigate networks of delay-coupled lasers. These include small network motifs, i.e. uni- and bidirectional rings and linear chains, as well as complex topologies including random and small-world networks. The nodes of the networks are described by the widely used Lang-Kobayashi model. By extending the well-known master stability function to networks with time-delay and non-vanishing coupling terms, we are able to separate the local dynamics from the topology. This way we can predict stability of synchronization for any network topology simply by calculating the eigenvalues of the corresponding adjacency matrix. Besides in-phase synchronization, we also observe alternating anti-phase synchronization, where only the next-nearest neighbors are synchronized. Our approach provides deep insight and understanding of the connection between topology and stability of synchronization. While our results are obtained for laser networks, we stress that the results are applicable to a wider range of systems, since only the local dynamics in terms of the master stability function will differ for other models.

DY 14.7 Wed 12:00 H44

Dynamics of neural networks with delay — •Judith Lehnert, Thomas Dahms, Philipp Hövel, and Eckehard Schöll — Institut f. Theo. Physik, Sekr. EW 7-1, Technische Universität Berlin, Hardenbergstr. 36, 10623 Berlin, Germany

We investigate synchronization in networks of delay-coupled FitzHugh-

Nagumo systems. The paramater values are chosen such that an uncoupled element operates in the excitable regime. However, the coupling acts as a noninvasive control force (Pyragas control) stabilizing the unstable periodic orbit of the synchronized oscillation of all elements. We calculate the master stability function, which denotes the maximum transverse Lyapunov exponent of the synchronization manifold as a function of the eigenvalues of the coupling matrix. Hereby we are able to demonstrate that all network topologies realized by excitatory coupling terms show stable synchronization in a wide range of coupling strengths and delay times.
Furthermore, we investigate small-world-like networks: In a regular network of neurons with excitatory coupling we randomly interpose additional inhibitory links. We show that this introduces a phase transition from the synchronized state to a desynchronized one as the number of these additional inhibitory links approaches a critical value.

DY 14.8 Wed 12:15 H44

Criticality in models of evolving neural networks — •Matthias Rybarsch and Stefan Bornholdt — Institut für Theoretische Physik, Universität Bremen, Otto-Hahn-Allee, 28359 Bremen

We investigate self-organization mechanisms in models of evolving neural networks. Already simple spin models can exhibit self-regulated evolution towards a critical state and are used as toy models for self-tuning in biological neural networks [1]. Recent models as, for example, ref. [2] are defined closer to the biological details, resulting in more complex node dynamics and link evolution. Here, we study a correlation-dependent mechanism for self-organized connectivity evolution as introduced in ref. [1]. In particular we focus on a model that is biologically motivated, yet keeping the dynamics as simple as possible. We find that independently from initial connectivity, the network evolves to an average connectivity close to criticality in terms of damage spreading.

[1] S. Bornholdt and T. Roehl: Self-organized critical neural networks, Phys. Rev. E 67, 066118 (2003)

[2] A. Levina, J.M. Hermann, and T. Geisel: Dynamical Synapses Causing Self-Organized Criticality in Neural Networks, Nature Physics 3, 857-860 (2007)

DY 14.9 Wed 12:30 H44

Spreading Synchrony in Neural Networks with Non-Additive Interactions. — •Sven Jahnke[1,2,3], Raoul-Martin Memmesheimer[4], and Marc Timme[1,2,3] — [1]Network Dynamics Group, Max-Planck-Institute for Dynamics & Self-Organization, Germany — [2]Bernstein Center for Computational Neuroscience, Germany — [3]Georg-August-University, Göttingen, Germany — [4]Center for Brain Science, Faculty of Arts and Sciences, Harvard University, USA

Recent neuro-physiological experiments [1] revealed that the response of cortical neurons to simultaneous pre-synaptic stimulation may be supra-additively enhanced. This enhancement is due to active nonlinear waves on the dendrite of a neuron (dendritic spikes) and offers a mechanism to synchronize neural spiking activity. Here we study the impact of nonlinear coupling on the dynamics of large neural circuits provide evidence that nonlinear dendritic enhancement is capable of inducing propagation of synchrony [2]. This yields the possibilty to generate patterns of precisely timed spiking activity, as observed in several neuro-physiological experiments. Our results indicate that and explains why densely connected feed-forward anatomy, as so far assumed in model studies [3], is not required for synchrony propagation but much more sparser connectivity is sufficient.

[1] Polsky, A., Mel, BW. and Schiller, J., Nature Neurosci. 7 (2004).

[2] Memmesheimer, RM. and Timme, M., Frontiers Comput. Neurosci., doi: 10.3389/conf.neuro.10.2008.01.009 (2008).

[3] Diesmann, M., Gewaltig, MO and Aertsen, A., Nature 402 (1999); Kumar, A., Rotter, S., and Aertsen, A., J.Neurosci. 28 (2007).