# DY 5: Statistical physics in biological systems (joint session DY/BP)

Time: Monday 14:30–16:45 Location: MA 001

DY 5.1 Mon 14:30 MA 001

**Point Mutations Effects on Charge Transport Properties of the Tumor-Suppressor Gene p53** — •Rudolf A. Roemer[1], Chi-Tin Shih[2], and Stpehan Roche[3] — [1]Department of Physics and Centre for Scientific Computing, University of Warwick, Gibbet Hill Road, Coventry, CV4 7AL, UK — [2]Department of Physics, Tunghai University, 40704 Taichung, Taiwan — [3]CEA/DSM/DRFMC/SPSMS, 17 avenue des Martyrs, 38054 Grenoble, France

We report on a theoretical study of point mutations effects on charge transfer properties in the DNA sequence of the tumor-suppressor p53 gene. On the basis of effective tight-binding models which simulate hole propagation along the DNA, a statistical analysis of mutation-induced charge transfer modifications is performed. In contrast to non-cancerous mutations, mutation hotspots tend to result in significantly weaker changes of transmission properties. This suggests that charge transport could play a significant role for DNA-repairing deficiency yielding carcinogenesis.

DY 5.2 Mon 14:45 MA 001

**A simple model for spike timing dependent plasticity** — Heinz Georg Schuster and •Jörg Mayer — Institute of Theoretical Physics and Astrophysics University of Kiel, D-24098 Kiel, Germany

Several experimental results indicate that the strength of cortical synaptic connections varies in dependence of the relative timing of pre- and postsynaptic spikes. We introduce a simple time discrete model of spike timing dependent plasticity. In our model the strength of synapses is increased if the timing of pre and postsynaptic spikes occur causal and decreased if they occur anti-causal. For reasons of convergence we further introduce a leakage term in the dynamics of the synapses. We find that stochastic independent input leads to a phase transition and further observe coherence resonance in dependence of the amplitude of the input noise. Further we derive the equations of motion for the mean field activity analytically.

DY 5.3 Mon 15:00 MA 001

**Self-organized critical control in human behavior** — •Felix Patzelt, Markus Riegel, Udo Ernst, and Klaus Pawelzik — Institute of Theoretical Neurophysics, University of Bremen, Bremen, Germany

When humans perform closed loop control tasks like in upright standing or while balancing a stick, their behavior exhibits non-Gaussian fluctuations with long-tailed distributions. We investigated if they might be caused by self-organized critical noise amplification which emerges in control systems when an unstable dynamics becomes stabilized by an adaptive controller that has finite memory. We generalized the basic model of self-organized critical control and compared it with experimental data from human control behavior. Our results suggest, that the nervous system involved in closed loop motor control nearly optimally estimates system parameters on-line from very short epochs of past observations. We discuss possible microscopic implementations of this principle in neuronal networks and multi-agent models which reveal its potential for explaining power law behavior in other physical systems.

DY 5.4 Mon 15:15 MA 001

**Panic reactions and global disease dynamics** — •Rafael Brune[1], Christian Thiemann[1], Bernd Blasius[2], Theo Geisel[1,4], and Dirk Brockmann[1,3] — [1]Max-Planck-Institute for Dynamics and Self-Organization, Göttingen — [2]Georg-August-University, Göttingen — [3]ICBM, Oldenburg — [4]Northwestern University, Evanston IL, USA

We analyze spatially extended disease dynamics in a system in which individuals change their dispersal characteristics in response to the local infection level. The key question is to what extent infectious wave front dynamics and the time course of the global infection change in response to host awareness and individuals trying to avoid infection by increased dispersal. We investigate two qualitatively different responses to the local degree of infection. In one system (panic reaction) the local diffusion coefficient increases with the concentration of infecteds, in the other system (directed reaction) indivuals drift proportional to infection level gradients. For both systems we develop a mean field model. Although one expects that the invididual rationale of avoiding an epidemic wave mitigates disease dynamics we find extended parameter regimes in which this rationale actually facilitates epidemic spread. Finally we investigate the dynamics of a fully stochatic system in which the effects prevail but which also show an increased extinction probability of the epidemic as a function of increasing dispersal response.

DY 5.5 Mon 15:30 MA 001

**Drift reversal in asymmetric coevolutionary conflicts: Influence of the microscopic processes and the population size** — •Jens Christian Claussen — Theoret. Phys. & Astrophys., Univ. Kiel

Coevolutionary dynamics in finite populations is investigated in chemical catalysis, biological evolution, social and economic systems; often formulated within the unifying framework of evolutionary game theory. From a payoff matrix characterizing the elementary interactions, traditionally evolutionary game theory proeceeds with the (deterministic) replicator equation ansatz tacitly assuming an infinite population size, In contrary, in finite populations the dynamics is inherently stochastic which can lead to new effects. In asymmetric conflicts between two populations with a cyclic dominance, a finite-size dependent drift reversal recently was demonstrated, depending on underlying microscopic process of the evolutionary update. Cyclic dynamics appears widely in biological coevolution, be it within a homogeneous population, or be it between disjunct populations (asymmetric conflicts) as female and male. Here the average drift is calculated analytically for the frequency-dependent Moran process and for different pairwise comparison processes. It is explicitely shown that the drift reversal cannot occur if the process relies on payoff differences between pairs of individuals. Further, also a linear comparison with the average payoff does not lead to a drift towards the internal fixed point. Hence the nonlinear comparison function of the frequency-dependent Moran process, together with its usage of nonlocal information via the average payoff, is essential for the (meta)stability of the internal fixed point.

DY 5.6 Mon 15:45 MA 001

**Bilateral interactions in disease dynamics - Decreasing epidemic thresholds with facilitated contact rates** — •Alejandro Morales Gallardo[1], Dirk Brockmann[1,3], and Theo Geisel[1,2] — [1]Max-Planck-Institute for Dynamics and Self-Organization, Göttingen — [2]Georg-August-University , Göttingen — [3]Northwestern University, Evanston IL, USA

Compartmental epidemiological models are very successful modeling paradigms in epidemiology. Typically, they are used for quantitative assessments of key parameters such as the basic reproduction number $R_0$. These models rest on two key assumptions: 1.) a population is well mixed 2.) transmission is triggered by a population averaged contact rate. However, experimental evidence shows that contact rates vary substantially, and it has been hypothesized that this variability can change the dynamics of population relevant disease dynamics. However, for inhomogeneous populations the translation of distributed contact rates into effective disease transmission events is non-trivial. Transmission may either depend only on the contact rate of the transmitting individual alone (unilateral transmission), or on the contact rates of transmitting and receiving individual (bilateral transmission). In the SIS model we show that in either systems the endemic state of a disease can be stable for values of $R_0 < 1$ unlike homogeneous systems with a critical value $R_0 = 1$. Furthermore, bilateral contact dynamics entail parameter regimes in which a stable endemic state can cease to exist if the mean contact rate is increased, an unexpected effect absent in homogeneous populations.

DY 5.7 Mon 16:00 MA 001

**Continuous description of a contact diffusion spread: complete separation of variables and the approximate analytical solution** — •Eugene Postnikov[1], Uta Naether[2], and Igor Sokolov[2] — [1]Lehrstuhl für Theoretische Physik, Staatliche Universität Kursk, Russland — [2]Institut für Physik, Humboldt - Universität zu Berlin, Deutschland

Despite of a century of thorough work, the problem of mathematical description of spread of an epidemic is still an actual question. In the present contribution we present the analytical considerations on the PDE system describing an SIR epidemic spread reproducing the re-

alistic asymmetric Kendall waves of infection as well as to verify the analytical solution by stochastic simulations. The model system as comprises three kinds of individuals (or cells with the perfect mixing of individuals inside) which are the susceptible (S), the infected (I), and the removed (R) ones. The probability for a cell to be infected can only change in case the cell is susceptible and depends on the number of its infected nearest-neighbors.

We show that the corresponding system of PDEs allows for a complete separation of variables. Moreover the solutions for I and R are given in a closed form if the solution for S is known. The autonomous equation for S admits the approximate analytical solutions for a wide range of parameters including the regions of a strong non-linearity.

The results of our analytical treatment are compared with direct Monte-Carlo simulations as well as with real epidemiological data on the epidemic among the harbor seals in Wadden Sea and Baltic Sea.

DY 5.8 Mon 16:15 MA 001

**The effects of bidirectional host movements on infectious disease dynamics** — •Vitaly Belik[1], Benjamin Schwenker[1], Theo Geisel[1,2], and Dirk Brockmann[1,3] — [1]MPI for Dynamics and Self-Organization, Göttingen — [2]Georg-August-University, Göttingen — [3]Northwestern University, Evanston IL, USA

Reaction-Diffusion equations such of the Fisher-Kolmogorov-Petrovsky-Piskunov (FKPP) type are widely applied in the context of spatial dynamics of directly transmitted diseases. This ansatz assumes that host individuals perform random walks or diffuse in space. Although this approach may find applications for animal host systems, its validity must be questioned for human infectious diseases. Although humans visit various places, they subsequently return to their abode thus performing bidirection movements on starlike topologies. Ordinary reaction and diffusion models are therefore not adequate for description of human spatial disease dynamics. We propose a stochastic model in which individuals can travel between their home and distant locations. We establish a link between explicit travel behavior of individuals and effective coupling among populations. We derive and analyze corresponding mean-field equations for the epidemic spread, which are structurally different from FKPP equation, e.g. diffusion and reaction are no longer uncoupled. The dependence of the front speed of the epidemic wave on the travelling rate is bounded from above, contrary to the common reaction-diffusion case, where it can attain any value. Our analysis is supported by agent based simulation of the full stochastic model.

DY 5.9 Mon 16:30 MA 001

**From Cannibalism to Active Motion** — •Pawel Romanczuk and Lutz Schimansky-Geier — Humboldt Universität zu Berlin, Newtonstr. 15, 12489 Berlin

The detailed mechanisms leading to collective dynamics in animal and insects groups are still poorly understood. A recent study by Simpson et. al. suggests cannibalism as a driving mechanism for coordinated migration of mormon crickets [1].

Based on this result we propose a simple generic model of brownian particles interacting by asymmetric, non-conservative collisions accounting for the cannibalistic behaviour and the corresponding avoidance strategy. We discuss our model in one and two dimensions and show that a certain type of collisions drives the system out of equilibrium and leads to coordinated active motion of groups.

[1] Stephen J. Simpson, Gregory A. Sword, Patrick D. Lorch and Iain D. Couzin: *Cannibal crickets on a forced march for protein and salt*, PNAS, 103:4152-4156, 2006