BP 23: Evolution

Time: Thursday 10:00-10:45

Location: H13

BP 23.1 Thu 10:00 H13

New phenotypes appear in an evolving population in non-Poissonian bursts — •NORA S. MARTIN¹, STEFFEN SCHAPER¹, CHICO Q. CAMARGO^{1,2}, and ARD A. LOUIS¹ — ¹Department of Physics, University of Oxford, Oxford, UK — ²Department of Computer Science, University of Exeter, Exeter, UK

For adaptive evolution, a central question is when and how frequently random mutations produce the specific rare adaptive phenotypes that have a selective advantage. A widely studied scenario is the following: a population starts with a given initial phenotype and accumulates neutral mutations, and new phenotypes are also introduced at a certain rate. Many theories implicitly assume that new phenotypes appear through simple stochastic processes which lead to Poissonian statistics. In this contribution, we use simulations on the biophysically motivated computational genotype-phenotype map from RNA sequences to secondary structures and show that new structures appear in highly non-Poissonian "bursts". In other words, if a new structure appears once, it is highly likely to appear multiple times in a relatively small number of generations. We show that there are several sources for this non-Poissonian behaviour, for example correlations in the mappings from genotypes to phenotypes, which may be a generic property of realistic genotype to phenotype maps. We find that these bursts can affect probabilities of fixation, especially when there are multiple competing adaptive phenotypes.

BP 23.2 Thu 10:15 H13 Proliferative advantage of specific an euploid cells drives evolution of tumor karyotypes — • LUCIJA TOMAŠIĆ¹, IVANA BAN¹, MARIANNA TRAKALA², IVA TOLIĆ³, and NENAD PAVIN¹ — ¹Department of Physics, Faculty of Science, University of Zagreb, Croatia — ²David H. Koch Institute for Integrative Cancer Research, Howard Hughes Medical Institute, Massachusetts Institute of Technology, Cambridge, Massachusetts 02142, USA — ³Division of Molecular Biology, Ruder Bošković Institute, Croatia

Most tumors have abnormal karyotypes, which arise from mistakes during mitotic division of healthy euploid cells and evolve through numerous complex mechanisms. In a recent mouse model with high levels of chromosome missegregation, chromosome gains dominate over losses both in pretumor and tumor tissues, whereas tumors are characterized by gains of chromosomes 14 and 15. However, the mechanisms driving clonal selection leading to tumor karyotype evolution remain unclear. Here we show, by introducing a mathematical model based on a concept of a macro-karyotype, that tumor karyotypes can be explained by proliferation-driven evolution of aneuploid cells. In pretumor cells, increased apoptosis and slower proliferation of cells with monosomies lead to predominant chromosome gains over losses. Tumor karyotypes with gain of one chromosome can be explained by karyotype-dependent proliferation, while for those with two chromosomes an interplay with karyotype-dependent apoptosis is an additional possible pathway. Thus, evolution of tumor-specific karyotypes requires proliferative advantage of specific aneuploid karyotypes.

BP 23.3 Thu 10:30 H13

Data-driven modeling of social interactions in bats across time scales — •FRANK SCHWEITZER¹, PAVLIN MAVRODIEV¹, and GERALD KERTH² — ¹Chair of Systems Design, ETH Zürich, Switzerland — ²Applied Zoology and Nature Conservation, University of Greifswald, Germany

The study of bat's social and foraging behavior is of great relevance to forecast the outbreak and distribution of virus induced deseases. To analyze this behavior we use a large-scale data set from two colonies of Bechstein's bats over five years. From this data, we reconstruct the social interactions of bats at three different time scales: (a) At the scale of minutes: social influence and information transfer. This leads to the formation of leader-follower pairs, where an informed individual leads an uninformed one to a roost box. (b) At the time scale of days: fission-fusion dynamics. This leads to the formation and dissolution of roosting groups of different size, composed of different individuals. (c) At the time scale of months: Emergence of social structures. This leads to the formation of communities within a colony. While the analysis of (a) requires statistical data analysis and hypothesis testing, for (b) we employ agent-based models, and for (c) social network analysis. The combination of these approaches allows us to bridge time scales in social behavior, which cannot be observed together. With our models we are able to develop the bigger picture of how social interactions feed back to long-term social structures.